



**NEW JERSEY CHAPTER
AMERICAN COLLEGE OF PHYSICIANS
REGIONAL SCIENTIFIC MEETING**

**RESIDENT/FELLOWS AND
STUDENT
ABSTRACT COMPETITION**

MARCH 22, 2019

PARTICIPATING INSTITUTIONS

Thank you to all the programs who submitted abstracts for this year's abstract competition.

Abstracts were received from the following programs:

Atlanticare Regional Medical Center (Dominik Zampino, MD, FACP)

Atlantic Health (Overlook) (Jeff Brensilver, MD, FACP)

Capital Health Regional Medical Center (Saba Hasan, MD, FACP)

Cooper University Hospital (Brian Gable, MD, FACP)

Englewood Hospital and Medical Center (Karlene Williams, MD)

HackensackUMC Mountainside (Bijal, Mehta, MD)

HackensackUMC Palisades (Sameh Elias, MD)

Jersey Shore University Medical Center (Perth Amboy/Raritan Bay) (Mayer R Ezer, MD)

Jersey Shore University Medical Center (Arif Asif, MD)

Monmouth Medical Center (Margaret Eng, MD, FACP)

Newark Beth Israel Medical Center (Jersey City) Program (Chris Engell, MD)

RowanSOM/Jefferson Health/Our Lady of Lourdes Health System Program (Judith Lightfoot, DO)

Rutgers Robert Wood Johnson Medical School Program (Ranita Sharma, MD)

Rutgers New Jersey Medical School Program (Mirela Feurdean, MD)

Saint Barnabas Medical Center (Sunil Sapru, MD)

Saint Francis Medical Center (Sara Wallach, MD, FACP)

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DISCLAIMER

It is assumed that all participants adhered to the rules as stated in the original abstract submission form. It is also assumed that the abstracts submitted were original works, represented by the true authors. The abstracts appear in no particular order. Judging was performed in an attempt to minimize bias. Judges were unaware of the authors unless they were directly involved with the associate. Although there were many excellent abstracts those selected to be presented as poster or oral presentation were chosen on the basis of content. This content was felt to be intriguing from a clinical education standpoint, thought provoking, or could stimulate debate regarding our current practice of medicine.

Thank You to Our Oral Abstract Presentation Judges

Jon Shammash, MD, FACP
Mirela Feurdean, MD, FACP
Tamie Proscia-Lieto, MD, MBA, FACP
Balaji Yegneswaran, MD, FACP

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ORAL ABSTRACT PRESENTATIONS

**Reinforcing Teamwork in a High Call Volume Step Down Unit: A Collaboration Among the Night Float
Residents, Hospitalists, Nurses**
Quality Improvement/Patient Safety

Krizelle Garde-Jara, MD; Amulya Dakka, MD; Urmil Patel, MD; Vishal Patel, MD;
Richa Dhawan, MD; Dominick Zampino, DO; Aliien Hocbo, MD; Jennifer Ketchled
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Background

The Progressive Care Unit (PCU) is an intermediary unit between the ICU and the general medical ward. Coverage is provided by a dedicated Hospitalist during the day. During the hours of 7pm to 7am, coverage is provided by a Nocturnist. Issues requiring immediate attention are channeled through a phone call/paging system, to the Nocturnist. In addition to the PCU, the Nocturnist is designated to respond to calls from the general medical ward, resulting in a high call volume to responder ratio. If the nocturnist is unable to respond to the calls, the Medical Evaluation Team (MET) is called to bedside.

Introduction

The PCU Call Volume Project puts in place a systematic approach to preventing disruption of workflow, and interference with patient care stemming from large call volumes, which result in overall team dissatisfaction. An evening PCU rounding system, will be in place for the duration of the project, with the aim to reduce the number of outgoing PCU calls/pages and MET calls during the night shift, and increase overall team satisfaction.

Method

Evening PCU rounds were conducted by residents, to address anticipated areas of concern. Day and Night Shift PCU nurses filled out a 9-question survey that evaluated the efficiency of the exchange of information with physicians, response times, and issue resolution over a 9-month pre-implementation period, which were compared with responses during the implementation period. Responses pertaining to communication with Medical Residents and Hospitalists, teamwork between Medical Residents and Hospitalists, and support from Medical Residents and Hospitalists fall under the umbrella of overall team satisfaction reported by the nursing staff. Data collected, including the number of phone/MET calls to providers, and the effectiveness of team dynamics based on communication and response times, were plotted and analyzed to assess the utility of the PCU rounds.

Results

Comparison of pre and post survey data demonstrated differences in overall team satisfaction. 60% of PCU Nursing reported satisfaction with response and issue resolution times post-survey, in contrast with 25% satisfaction pre-survey. A remarkable 381.9% improvement in communication between Hospitalists and Nursing was reported. A comparative analysis of the Pre (Oct-Apr, 2016) and Post (Oct-Apr, 2017) survey periods demonstrated a 21.7% decrease in the number of MET calls since the PCU evening rounds were implemented. Prior to the project taking effect, the number of MET calls between the months of Jul-Sep 2017 and the months of Jul-Sep 2016 did not show significant change.

Conclusion

Data indicated that the evening rounding system resulted in a reduced number of MET calls, translating to a decrease in the number of patients that decompensated and required immediate evaluation during the night, resulting in improvement in workflow, manpower and time utilization, and overall increase in team satisfaction.

Carrots or Sticks - A Not so Novel Way to Dramatically increase the Immunization Rate

Quality Improvement/Patient Safety

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Daniel Goldsmith, MD
Capital Health Regional Medical Center Program (Saba A. Hasan, MD)

Background

Immunization rates in adults in the United States are low, especially amongst racial and ethnic minorities and the uninsured.

Resident-staffed free ambulatory clinics provide valuable healthcare access to the uninsured population, whom otherwise utilize the emergency department to seek medical care.

In participation with the ACP QI champion program and i-Raise the Rates, we set out to increase immunization rates in our free ambulatory clinic community. Utilizing the PDSA and SMART model of problem solving along with Rapid Experiments and Tests, we were able to improve our vaccination rates above our expectations.

Methods

To achieve our goals we initiated a series of steps to include nursing staff standing orders, daily team huddles and resident education through morning reports and noon conference series. These steps yielded a very minor increase in our immunization rates. The first PDSA cycle uncovered an issue with resident compliance as it is easy to overlook vaccination deficiencies during sick visits. We further expanded our efforts to include a Pre-Visit Chart Check List (PVCC) to ensure residents are reminded of vaccination deficiencies. We also incorporated bi-weekly random chart checks to monitor progress of the initiatives and to identify any issues. Our observation demonstrated that even with the PVCC many residents failed to comply with the initiative as it was not a required part of the daily routine. With collaboration of faculty and nursing staff, we made the PCVV mandatory part of the patient encounter, regardless of the reason behind the visit. Residents had to complete the PCVV prior to the visits and share the information with the nursing staff prior to presenting the case to the attending. If the PCVV was not completed, residents could not present the case and had to return after completing the list.

Results

Tdap vaccination volume in the cycles before the PCVV was 8.5 per month. Post-PCVV the rate was 17.5 per month, a 105% increase.

Pneumococcal vaccination volume in the cycles before the PCVV was 11.7 per month. Post-PCVV the rate was 14.3 per month, a 22% increase.

Discussion

We conclude that mandatory steps are required to ensure resident/physician compliance as a means to improve vaccination rates. Incentives play an important role to ensure minimum PCVV follow up; however, mandatory requirements along with hard-stop checkpoints were found to be more effective.

Another important aspect of PDSA cycles are their short duration. This allows mistakes and obstacles to be identified quickly, resulting in more rapid solutions and adaptations. Thus, carrot and stick approaches should be applied such that both have positive motivational effects on staff.

Capsule Endoscopy Transit Time to Duodenum: Data to Support Safety of Shorter Interval Between Colonoscopy Prep and Procedure

Research

Diana Curras-Martin, MD; Shreya Gor; Alsadiq AlHillan; Adaeze Ezeume; Varsha Gupta;
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Jersey Shore University Medical Center Program (Arif Asif, MD)

Background

To minimize risk of aspiration, anesthesia guidelines recommend fasting time before endoscopic procedures anesthesia practice guidelines state that procedures may be safely performed 2 hours after ingesting fluids. The American Society for Gastrointestinal Endoscopy (ASGE) states that the final oral preparation liquid should be administered 3-8 hours prior to the procedure. However, at our institution some anesthesiologists will delay endoscopy cases if liquids are consumed within 4,6, or 8 hours before the procedure. Some will delay if the liquid was milk. Objectively documenting gastric transit time via a review of pill endoscopy data could address clinicians concerns, prevent delays in patient care, and improve the rate at which our clinicians practice within national guidelines. The objective was to utilize capsule endoscopy data from our center to determine the gastric transit time (GTT) from stomach to duodenum and small bowel transit time (SBTT) in relation to patient factors such as BMI, albumin, diabetes mellitus (DM), chronic kidney disease (CKD).

Methods

This retrospective chart review obtained data on adult pill endoscopy (PillCam) studies on in- and out-patients. Past medical history and laboratory data was only available via the EMR only on inpatients. Mean GTT and SBTT reported in minutes+S.D. Statistics: Descriptive and t-test to compare means.

Results

163 records reviewed. 4 patients were excluded as the pill did not pass out of the stomach. Mean age 66 years, 57% female, 26% evaluated for GI bleeding, mean hemoglobin 9.1 mg/dL+ 3, mean ferritin 195+395. Mean GTT Time in Minutes: All patients (n=159) 35+49, median 19, range 1-383. There were no statistically significant differences in GTT between the following subgroups: CKD0 (n=100) 40+58 vs. CKD5 (n=11) 35+39, Albumin >3.0 (n=123) 37+53 vs. Albumin<3.0 (n=36) 27+30, Diabetes (DM, n=40) 51+71 vs. NonDM (n=119) 42+79, BMI>30, or aspirin use. SBTT Results: All patients (n=124) 238 minutes +88. Similarly, there was no relation between SBTT and albumin, any CKD, CKD0 vs CKD5, DM status, or BMI. The ages of patients with the capsule stuck in the stomach were 61, 66, 72, and 82; and they did not have other clinical history to explain the complication.

Conclusion

This analysis of objective data regarding pill endoscopy found that the average gastric transit time was 44 minutes, for 50% it was <20, and for 85% <60. We did not find that GTT was longer in those with DM, any CKD, albumin <3.0 mg%, BMI, aspirin, or CKD 5 vs. CKD0. This data supports the recommendations that endoscopic procedures, in accordance with anesthesia and ASGE guidelines, can be safely conducted in the majority of patients within 60 minutes of ingesting liquids.

Trimetazidine in the Prevention of Contrast-Induced Nephropathy in Patients with Renal Insufficiency: A Systematic Review Research

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Introduction

Contrast-induced acute kidney injury (CI-AKI), is an acute impairment in renal function and it is one of the leading causes of acute kidney injury. The incidence of CIN is increasing every year, which is associated with increased patients' morbidity and mortality. Trimetazidine (TMZ) is a type of anti-ischemic drug which inhibits the excessive release of oxygen-free radicals, increases glucose metabolism, and reduces membrane lipid peroxidation after ischemia–re-perfusion. Trimetazidine has been treated as a drug with a high safety and tolerability profile that rapidly absorbed from the intestinal tract. Data on the renal effects showed TMZ can significantly reduce the incidence of CI-AKI. We conducted this systematic review and meta-analysis of clinical trials that investigated the clinical effect of TMZ on prevention of CIN in patients with renal insufficiency undergoing coronary angiography.

Method

This meta-analysis was conducted according to the Preferred Reporting items for Systematic Reviews and Meta-Analyses guidelines (PRISMA). A search of the Medline, Cochrane and Google Scholar databases were conducted by two researchers independently. The primary search for clinical trials was for clinical trials comparing TMZ versus conventional hydration for prevention of CIN through September, 2018. The outcome of interest was the incidence of CI-AKI. The relative risk (RR) was estimated with 95% confidence interval (CI). Heterogeneity was reported with the I² statistic, using a fixed-effects model, and >50% of I² was considered to be statistically significant. P<0.05 was considered to indicate a statistically significant difference. All statistical analyses were performed using STATA software, V-12.0.

Result

1021 potentially relevant articles were compiled using the different databases. Of these, 751 articles were excluded because they did not meet the selection criteria on the title or abstracts. The remaining 270 results were considered to be of relevance and full papers were carefully screened. Finally 11 studies met our inclusion/exclusion criteria for this analysis which included 1611 patients. Among them, 797 patients were included in the TMZ plus hydration group and 814 patients in the control (hydration) group.

Results of the meta-analysis showed that the heterogeneity of studies is low: I²=0%, P=0.747,. The incidence of CIN in the TMZ plus hydration group was 6.0% (48/377) and the incidence of CIN in the hydration group was 20.6% (75/814).

A pooled analysis for all studies showed that TMZ can significantly reduce the incidence of CIN-AKI compared to saline alone (relative risk 0.35, [CI 95%] 0.25, 0.48, P=0.000).

Conclusion

This systematic review and meta-analysis was presented by combining data from 11 studies which showed Trimetazidine can effectively protect renal function and reduce the incidence of CIN in patients undergoing coronary angiography. We recommend large-scale design RCTs with long-term follow-up incorporating the evaluation of clinically patient-centered outcomes to confirm the robustness of our results.

**RESIDENT ABSTRACT
POSTER PRESENTATIONS**

RESEARCH

A Resident-Led Initiative to Bring Teaching Back to the Bedside at a Community Hospital

Research

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AtlantiCare Regional Medical Center Program (Dominick Zampino, DO)

Background

Historically, bedside teaching is one of the most important teaching modalities in medical education. Over the years, the prevalence of bedside teaching in residency programs has been on the decline while the prevalence of physician burnout has been on the increase. Many have attributed the decline in time spent with patients and increase in time spent with computers as a potential contributor to the rising physician burnout. We set out to find ways to allow residents to engage in meaningful encounters with patients at the bedside with the potential of helping the residents find meaning in their work and training.

Objective

To improve the prevalence of bedside rounds and to examine the effect of bedside rounds on residents' and faculty's satisfaction, burnout, and meaning-in-work.

Methods

This prospective study used a pre/post intervention design to obtain data from internal medicine residents and faculty. The intervention was a one-time training on how to provide and engage in bedside rounds. These trainings were conducted separately for residents and faculty in the first week of July 2018. Pre-intervention data were collected between January 8 and June 28, 2018 while the post-intervention data were collected between July 23 and October 19, 2018. The data collected included daily information on teaching rounds and residents and faculty surveys on satisfaction, burnout, and meaning-in-work. The sample included 20 residents and 12 faculty in the pre-intervention period and 29 residents and 10 faculty in the post-intervention period.

Results

Two teaching teams were studied, one in the Mainland Campus and another one in the City Campus. Each teaching team consisted of a faculty member, one resident, and two interns. On average, each team had sixteen to seventeen patients with three to four new patients per day. In the pre-intervention period, there were no bedside rounds in the City campus while one patient per day was rounded on at the bedside by the Mainland team. In the post-intervention period, the number of patients with bedside rounds increased to an average of three patients a day in both campuses. The duration of bedside rounds per patient in both campuses increased from an average of twelve to fourteen minutes in the pre-intervention period to fourteen to sixteen minutes in the post-intervention period. Residents' satisfaction and meaning-in-work significantly improved while residents' burnout rate significantly decreased during the study period. In contrast, the faculty had decreased satisfaction and increased burnout rate during the study period. There was no significant difference in the faculty's meaning-in-work.

Implications

Bringing residents back to the patients' bedsides not only improves their clinical skills but also has the potential to improve their wellbeing. Thus, emphasis should be placed on meaningful engagement with patients during residency training.

Screening Hospitalized Patients for Obstructive Sleep Apnea Research

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Background

Obstructive sleep apnea (OSA) is a disorder that is characterized by obstructive apneas and hypopneas due to repetitive collapse of the upper airway during sleep. OSA is the most common sleep-related breathing disorder. Prevalence estimates vary according to the way in which OSA is defined and the distribution of risk factors in the population being studied. The estimated prevalence in North America is 20 to 30 percent in males and 10 to 15 percent in females when OSA is defined broadly. Patients with OSA, particularly when it is moderate or severe and untreated, are at increased risk for a broad range of cardiovascular morbidities, including systemic hypertension, pulmonary arterial hypertension, coronary artery disease, cardiac arrhythmias, heart failure, and stroke. STOP BANG questionnaire is the most validated tool for screening.

Purpose

The study was interested in screening hospitalized patients at risk for underlying undiagnosed sleep apnea. Patients on select floors were screened using the STOP BANG survey. The study was interested in seeing how many patients fall into intermediate and high risk. A secondary goal was to educate intermediate and high risk patients regarding OSA and to have them follow up with outpatient physician for further evaluation.

Methods

A member of the hospital quality team, screened patients on select floors in the hospital. The patients were given the option to be screened or not. Screening was completed using STOP BANG. Patients who scored intermediate or high risk, were informed of their score. These patients were given a brochure on obstructive sleep apnea, education by a respiratory therapy, and instruction to follow up with their physician outpatient for further evaluation.

Data Analysis

Each patient was scored using the STOP BANG survey. Patients who scored 0 to 2 were low risk. Patients who scored 3 to 4 were intermediate risk and patients who scored greater than 5 were high risk. The total number of patients who scored low risk, intermediate risk and high risk was then totaled. Percentages were calculated for each of the three groups. The study found (92/276) 33% scored low risk; (102/276) 37% scored intermediate risk; (82/276) 67% scored high risk. Therefore, (184/276) 67% of the patients scored intermediate to high risk for underlying sleep apnea.

Conclusion

A majority of hospitalized patients, 67%, screened within intermediate/high risk using STOP BANG. In particular, these patients are at increased risk for cardiovascular events including, cardiac arrhythmias, congestive heart failure, hypertension, and cardiomyopathy. Other consequences of untreated sleep apnea include diabetes, depression, chronic fatigue, hypersomnia, memory loss, sexual dysfunction and stroke. These results will lead to a hospital wide intervention to screen all hospitalized patients in an effort to begin treating these individuals.

Prevalence of Obesity in Hospitalized Adult Patients: A Retrospective Cohort Study

Research

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Jersey Shore University Medical Center Program (Arif Asif, MD)

Background

While obesity is a serious chronic condition, its prevalence and documentation as a diagnosis has not been extensively studied in hospitalized patients. We conducted a retrospective chart review to investigate the prevalence and documentation of obesity as a diagnosis among patients admitted to our medical center.

Method: All consecutive patients admitted to inpatient medical floor during a 60-day period from September through October 2017 were selected for the study. The following information was abstracted from the charts: Patient demographics including Body mass index (BMI), admission/discharge note with obesity as one of the diagnoses, common medical comorbidities (hypertension, diabetes, hyperlipidemia, coronary artery disease, congestive heart disease, chronic kidney disease, chronic obstructive pulmonary disease), and length of stay. Obesity was categorized per the CDC guidelines as: normal weight (BMI 18–24.9 kg/m²), overweight (BMI 25–29.9 kg/m²) and obese (BMI > 30 kg/m²). The obese group was further divided into 3 classes. Class I (BMI 30–34 kg/m²), Class II (BMI 35–39.9 kg/m²) and Class III (BMI > 40 kg/m²). We also reviewed charts regarding documentation of counselling to the obese patients for weight loss.

Results

A total of 540 consecutive charts were reviewed. The mean age was 66.76 years. Out of 540 patients only 182(34%) had normal weight, 188(35%) were overweight and 170(31%) were obese. In the obese group, 55% were female. 100(59%) had class I obesity, 43(25%) had class II obesity and 27(16%) class III obesity. Obesity was documented on the admission problem list in 40/170(23.5%) patients and only 21(12%) had obesity documented as a discharge diagnosis. 3 (2%) patients were given appropriate obesity and weight reduction related counseling and referral during the hospitalization. Comorbidities and their prevalence included, hypertension (68%), diabetes (35%), hyperlipidemia (36%), coronary artery disease (18%), chronic kidney disease (17%), congestive heart failure (18%) and COPD (24%). The average length of stay regardless of BMI group was 4.5±0.5 days.

Conclusion

A significant number of hospitalized patients were overweight and obese. An overwhelming percentage never had weight status documented. Hospitalization offers health care providers a window of opportunity to identify obesity, communicate risks and initiate weight management interventions.

The Perspective of a Breast Cancer Patient: A Survey Study Assessing Needs and Expectations Research

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Background

Patient satisfaction is one of the key indicators of health care quality. We aim to identify patients' needs and expectations to achieve a patient centered care and better overall satisfaction.

Method

A 17-item survey was administered to 49 patients at a breast cancer clinic. The survey was designed after a thorough literature review and approved by an oncologist and a palliative care physician.

Results

Self-reported knowledge about disease was reported adequate by 95.9% of our patients yet only 46.9% of our patients could identify the stage of their cancer. More education was desired by 39% of patients including various treatment options (35%), common complications (29%), prognosis (22%) and risk factors (14%). Cancer related emotional stress, physical symptoms and effect on social/professional life was reported by 53%, 53% and 49% of our patients respectively. Majority of our patients (57%) wanted their oncologist to address social/emotional issues and 28% felt the need of more focus on physical symptoms in their subsequent visits. End of life care (EoLC) discussions were considered an integral component of overall care by 22% of our patients. Components of EoLC discussions that patients stated they could benefit from included prognosis (24.5%), life expectancy (24.5%), treatment effect on QoL (20.4%), palliative care (10.2%), hospice (10.2%), advance directives (14.3%) and family involvement in medical decision making (12.2%). More patients wanted to initiate EoLC discussions when they requested it (30.6%) or their oncologist thought it was appropriate (30.6%) as compared to when it may change treatment course (12.2%) or at diagnosis (8.2%). More patients wanted having EoLC discussions only once (40.8%) as compared to at regular intervals (32.7%)

Conclusion

A discrepancy between self-reported and actual knowledge in breast cancer patients emphasizes the need of patient education. Most patients rely on their oncologist for their diagnosis related emotional and social issues. Surprisingly, a quarter of our patients consider EoLC discussions important even though majority of our patients were healthy and having stage I disease.

Autoimmune Hepatitis and Extra-Hepatic Autoimmune Disease: A Nationwide Study Research

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Background

Autoimmune hepatitis (AIH) is a chronic hepatitis that is caused by a reaction towards the body's endogenous liver enzymes. Extra-hepatic manifestations are said to present in 20% of cases and may occur either before onset of disease or during the clinical course. However, the relationship that exists between AIH and extra-hepatic autoimmune disease (EHAID) has not been fully defined and it is suggested that different autoimmune conditions affect AIH differently. Indeed, this association as well as the increased incidence of depression seen in this cohort are enough impetus to warrant further examination. The aim of this study is to observe the national trends in AIH hospitalizations while examining the relationship with concurrent autoimmune disorders.

Methods

The National Inpatient Sample (NIS) is the largest all-payer inpatient database consisting of approximately 20% of all inpatient admissions to nonfederal hospitals in the United States. We collected data from years 2012 – 2014. Cases of AIH and other autoimmune conditions as secondary diagnoses were identified using the International Classification of Diseases, 9th Edition, Clinical Modification (ICD-9 CM). Percentages of AIH patients affected by at least one other EHAID, as well as length of stay (LOS), cost of admission, and mortality were evaluated and adjusted for comorbidities.

Results

Between 2012 - 2014, there were 11,270 cases of AIH identified. Approximately 22% of patients had at least one EHAID, with most affected by hypothyroidism, lupus, and rheumatoid arthritis. However, those with additional autoimmune conditions had a decreased LOS as compared to those with just an AIH diagnosis (4.81 days vs. 5.61 days, $p < 0.001$). Mortality rate was also decreased, with 0.97% in those with EHAID and 2.8% mortality in those with just AIH ($p < 0.001$). 33.1% of the total population had a severe Charlson score which comprised 29.6% of the AIH population and 44.5% of the cohort with additional autoimmune diseases ($p < 0.001$).

Conclusions

When AIH patients were found to have additional autoimmune comorbidities, there was a decreased LOS, cost, and mortality rate. It is possible that clinicians act more expeditiously in suspecting autoimmune hepatitis and initiating treatment once they are aware of additional autoimmune diagnoses. Increasing efficiency of patient care while decreasing the LOS and mortality rates in this fashion is meaningful and could greatly impact the medical community.

Systematic Review and Meta-analysis: Alcohol as a Risk Factor for Inflammatory Bowel Disease- Science or Implicit Bias?

Research

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Background

Alcohol consumption has generally been thought to be associated with increased risk of inflammatory bowel disease (IBD) development. However, recent studies have not shown consistent association between alcohol use and increased risk of ulcerative colitis (UC) and Crohn's disease (CD) development. The debate is ongoing and expert opinions have been written in editorials highlighting this discordance. Can a more robust scientific evidence through a meta-analysis finally lay this controversy to rest?

Methods: We identified published manuscripts and abstracts through October 2018 by systematic search of Medline, Web of science, Cochrane and other trial registries. Quality assessment was done using Newcastle-Ottawa scale and random-effect meta-analysis using pooled relative risks (RRs) and 95% CIs were calculated.

Results

Fifteen publications between years 1994 and 2018 were identified with a total of 4177 cases and 318,425 controls across studies pooled for the meta-analysis. Pooled RRs of UC risk with alcohol use was (0.83, 95% CI, 0.53-1.31), $I^2 = 85\%$ and (0.90, 95% CI, 0.43-1.89), $I^2 = 89\%$ for CD. Sensitivity analyses excluding studies with alcohol intoxication as exposure definition showed a significantly decreased risk of CD development (0.65, 95% CI, 0.44-0.97) but not UC (0.73, 95% CI, 0.46- 1.13) with alcohol use. There is no evidence of publication bias, p-values of Egger's test = 0.491 for UC and 0.109 for CD.

Conclusion

Contrary to earlier belief, this study did not find an association between alcohol use and increased risk of CD and UC development. Rather, a decreased risk of CD development was found when the analysis excluded studies on alcohol intoxication. Our results are in keeping with findings of more recent and better conducted studies. We believe the improved statistical power of our study has helped clarify the notion of alcohol being a risk factor for IBD development is more of an implicit bias rather than science.

Do Not Resuscitate: What Does it Mean?

Research

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Background

Do-Not-Resuscitate (DNR) patients in the acute setting experience significantly poorer outcomes compared to non-DNR patients. So, is it ethically right to provide aggressive medical care for the DNR population despite high mortality? This study was conducted to investigate the outcomes of patients with DNR status on admission.

Methods

Single center, retrospective cohort analysis of 538 patients with DNR orders on hospital admission from January 1, 2017 to June 30, 2017 at Saint Barnabas Medical Center. Demographics, comorbidities as well as outcomes such as palliative care use, critical care interventions, length of stay, cost and mortality were extracted from the electronic medical record. Chi-square and independent sample t-tests were used to calculate significance, with a 95% confidence interval.

Results

A total of 12572 patients were admitted, of which 538 were DNR (4.28%). All-cause mortality was 2.05% (258/12572) versus 24.5% (93/538) in the DNR cohort. Palliative care, hospice and chaplaincy services were all severely underutilized (45.2%, 29.2%, 32.5% respectively). Overall, hospital length of stay (LOS) was 7.5d, with alive patients having significantly longer LOS than those that died (6d vs 5d respectively, $p=0.003$); total cost of hospitalization was higher in patients who died but results were not statistically significant (\$13,606 in dead vs \$12,839 in alive, $p=0.20$). Mortality was significantly higher in those requiring ICU admission, (54/89, 60.1%), [OR 7.34, 95% CI (4.49 – 11.98), P -value<0.001], renal replacement therapy (8/15, 53.3%), [OR 3.68 95% CI (1.31-10.34), P -value=0.009], mechanical ventilation (37/45, 82.2%), [OR 19.38, 95% CI (8.74 – 42.96), P -value<0.001], non-invasive mechanical ventilation (40/99, 40.4%), [OR 2.56, 95% CI (1.61 – 4.06), P -value<0.001], and vasopressors (27/35, 77.1%), [OR 12.79, 95% CI (5.65 – 29.98), P -value<0.001].

Conclusion

Our analysis reveals that only a minor fraction of patients have DNR orders on admission, and these patients were 12 times more likely to die during hospitalization, especially if they required aggressive interventions. Early goals-of-care discussions are prudent necessitating a significant cultural change so that appropriate resources can be allocated to alleviate pain and suffering while providing comfort care at the end of life.

All-Cause Mortality Rate between Cardiac Contractility Modulation Group and Standard Therapy in Patients with Dilated Cardiomyopathy

Research

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Background

Dilated Cardiomyopathy is associated with very high mortality despite maximal medical therapy. Recently, a few randomized clinical trials involving usage of Cardiac Contractility Modulation for the management of dilated cardiomyopathy (not eligible for cardiac resynchronization) demonstrate significant improvement in cardiac function. These studies are not powered to calculate a mortality benefit. A previously published meta-analysis did not demonstrate a mortality benefit. A newly randomized trial has been published since the last meta-analysis, allowing for an updated meta-analysis.

Objective

Comparison of all-cause mortality rate between cardiac contractility modulation group and standard therapy Patients with Dilated Cardiomyopathy followed for 12 weeks or more.

Methods

We conducted a systematic search of Medline (PubMed) and Cochrane Central Register of Controlled Trials for abstracts and fully published studies (from inception to October 2018) comparing Cardiac Contractility Modulation device therapy with standard therapy for patients with dilated cardiomyopathy (ischemic and non-ischemic). Search conducted between September 1, 2018, and October 30 2018.

Study Selection

Fully published randomized clinical trials comparing all-cause mortality outcomes of device therapy and standard therapy for patients with dilated cardiomyopathy (ischemic and non-ischemic) were included in our meta-analysis. Total 1132 studies were identified. Studies which were duplicate, non-randomized, included pediatric population, systematic reviews or meta-analysis, study designs or protocols, trials including gene therapy or follow up of patients for less than 12 weeks were excluded.

Data Extraction and Synthesis

Data were abstracted by two independent reviewers. Using Mantel-Haenszel method, a random effect model was used to calculate weighted Risk ratio (RR). RevMan 5.3 was used for statistical analyses.

Main Outcome

The primary outcome was a comparison of all-cause mortality between the two groups (Cardiac contractility Modulation group and Standard therapy group) for patients followed for a period of 12 or more weeks.

Results

Four published randomized clinical trials met the inclusion criteria of our analysis. Using the Mantel-Haenszel method, a random effect model was used to calculate the weighted risk ratios. Analysis included a total of 930 patients. Cardiac Contractility Modulation therapy group showed no significant reduction in mortality compared to standard therapy group (risk ratio [RR], 0.63; 95% confidence interval, 0.29-1.35, $p=0.23$), however, the effect favored the device therapy. Tests for statistical heterogeneity did not show any significant heterogeneity $p\text{-value} = 0.82$ ($I^2 = 0\%$).

Conclusion

Cardiac Contractility Modulation device therapy is not associated with significant mortality reduction in patients with dilated cardiomyopathy. Our Meta-analysis underscores the need for a large randomized controlled trial on the efficacy of Cardiac contractility modulation in patients with dilated cardiomyopathy who are ineligible for Cardiac Resynchronization therapy.

**RESIDENT ABSTRACT
POSTER PRESENTATIONS
QUALITY IMPROVEMENT/PATIENT SAFETY**

Improving Hypertension Management in a General Internal Medicine Practice

Quality Improvement/Patient Safety

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Introduction

Hypertension (HTN) control prevents up to 13,000 deaths annually in the U.S. Screening for HTN and documenting follow-up is a quality metric that our general internal medicine (GIM) practice needed to improve. A review of clinic visits prior to our initiatives showed that only 15% of our patients who screened positive for HTN had documented follow-up plans. However, it was unclear if BP measurements were obtained appropriately or repeated for confirmation. Our quality improvement initiative aimed to improve the accuracy of HTN screening and increase the rates of <4-week follow-up for patients found to have HTN on repeat confirmation.

Methods

We conducted a root cause analysis (RCA) to identify barriers to optimal HTN screening and follow-up. We then implemented several plan-do-study-act (PDSA) cycles from April 2017 through January 2018. Our first intervention was the creation of a centrally located EMR BP tab that serves as virtual communication between the CMAs and providers and also facilitates documentation for follow-up plans. As a process measure, we found that this EMR enhancement needed further PDSA cycles for optimal use. The subsequent interventions included: 1) multidisciplinary education to the healthcare team on proper BP measurement technique and the importance of confirmatory BP measurement, 2) installing exam room flags to alert CMAs of patients needing repeat BP, 3) creating a process flow for BP confirmation, and 4) increasing the number of ambulatory sphygmomanometers. Our primary aim was to increase the frequency of repeat BP measurements by 30%, as compared to the baseline of 18%. Our secondary aims were to increase documentation of a follow-up plan to 50% and to increase repeat visits within 4 weeks for those truly hypertensive patients to 20%.

Results

Over a 10-month time frame, we show that our initiatives increased the frequency of repeat BP measurements from 18% to 51%, thus allowing for correct HTN diagnoses. Since the creation of the EMR tab (August 2017), we show that appropriate BP documentation increased from 0% to 16.2% and the rates of return visits within 4 weeks for newly diagnosed HTN patients increased from 3% to 21.6%.

Discussion

Through the use of quality improvement principles including RCA, PDSAs, buy-in, and measuring tests of change, we show that we successfully improved our clinic's quality metric for screening for HTN and documenting follow-up. However, the rates of documented follow-up, while improved from our baseline, remained surprisingly low. This highlights the underlying need for re-education efforts to allow for sustainability. As such, our next steps will not only include another series of RCAs and PDSAs to identify the barriers to establishing follow-up but also initiatives to promote re-education on the already established interventions to promote overall improvement and sustainability.

Effect of Clinical Documentation Education on Reimbursement – A Quality Improvement Project

Quality Improvement/Patient Safety

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Saint Peter's University Hospital Program (Nayan K Kothari, MD)

Background

The numbers of clinical documentation specialist (CDS) are increasing over the past decade. Many residency programs are including clinical documentation education as a part of resident education. We conducted a quality improvement project to understand the impact of such education on our hospital's reimbursement.

Study Design

A clinical documentation education program was instituted to all physicians and residents of our internal medicine department. The program consisted of didactic lectures, 1-on-1 provider training, case reviews, and clinical documentation queries from clinical documentation improvement staff and hospital coders. All queries for the month of August were reviewed and the charts relative weight (RW) of diagnosis, hospital reimbursement, and length of stay (LOS) was compared before and after the query was answered. Financial gain from responses to clinical documentation improvement queries was determined retrospectively. All data was saved in Microsoft Excel.

Results

A total of 111 charts were reviewed. Physicians had a 100% response rate to the query. Of the responded query 71.2% resulted in hospital significant impact for the hospitalization diagnoses-related group, risk of mortality (ROM) and severity of illness (SOI). Significant increases in department 's severity of illness, risk of mortality, case mix index (CMI), and Medicare Severity Diagnosis related group (MS-DRG) were found after clinical documentation education implementation. Provider responses to clinical documentation improvement queries resulted in an estimated \$814,121 increase in charges and allowed cumulative 125.9 extra days stay for the study population during the study period.

Conclusions

Clinical documentation improvement program in internal medicine department is an effective method to improve documentation rates, increase the hospital estimated reimbursement based on more accurate clinical documentation, and provide better compliance with quality measures. With increasing emphasis on quality metrics outcomes under current United States health care system, there is a need for institutions to accurately capture the complexity and acuity of the patients they care for. The 1-step query process provides a reproducible and effective way for clinical documentation specialists and physicians to collaborate on improving departmental clinical severity metrics.

The ACCESS Examination: A Quality Improvement Project

Quality Improvement/Patient Safety

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ACCESS - Improvement Of Residency Education Through The Introduction Of A High Stakes Clinical Examination To The Internal Medicine Residency Training Curriculum - A Quality Improvement Project

Introduction

PACES (Practical Assessment of Clinical Examination Skills) is a tool developed by the Royal College of Physicians (U.K.) to systematically evaluate history-taking and clinical examination skills. This examination is a prerequisite for the completion of graduate medical education. However, in the U.S., the USMLE Step 2 CS remains the first, last and only clinical examination for internal medicine residents prior to commencement of graduate medical education. No examination evaluating competency of history taking and clinical examination skills necessary for graduation exists as a part of ACGME residency training.

Objective

In collaboration with the Royal College of Physicians of Edinburgh, we conducted a high-stakes clinical examination named ACCESS (Assessment of Clinical, Communication and Examination Skills including Simulation) modelled after PACES, that assess bedside clinical competence in seven domains.

Method

The ACCESS examination adopted the structure of PACES. There were 5 interactive stations with 8 patient encounters. Six out of eight patients had real physical findings. Each station had two examiners who calibrated amongst themselves thus maintaining objectivity. The candidates were evaluated on 7 domains. Our faculty was trained by RCPE faculty. The impact of this examination in furthering history taking and clinical skills amongst residents was tested using a post-test questionnaire.

Results

42 residents have taken this exam across 8 sessions held over 3 years. 25 faculty members have undergone extensive training to qualify as standardized examiners. 80.95% of residents who took the exam felt that preparation and undertaking the examination lead to an improvement in their history taking proficiency and clinical examination skills. In addition, the initiative has brought a paradigm shift in the culture of our Program. From didactic sessions to rounds to training in the Simulation lab, the emphasis is on enhancing bedside clinical skills.

Conclusion

Internal medicine graduate medical education in the U.S. does not have a tool for critical appraisal of residents' bedside clinical skills necessary for the delivery of high quality care. Our Program aims to bridge this gap with the introduction of the high-stakes ACCESS examination. It comprises of real patients with senior faculty trained as examiners who inter-calibrate to remove subjective bias. Given that proficiency in these skills have been linked to comprehensive high-quality patient care, decreased wastage of medical resources and improved doctor-patient relationships, in the near future we hope to offer this examination as a summative tool prior to graduation.

Use Of High Fidelity Simulation-Based Medical Education In A Community Hospital Internal Medicine Residency Program To Enhance Resident Competency Of Three High-Acuity Clinical Scenarios Quality Improvement/Patient Safety

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Introduction

ACGME promotes Simulation Based Medical Education (SBME). This has emerged as an important but underutilized tool that provides risk-free learning of high acuity scenarios. Furthermore, at a time when healthcare is moving to a team-based interdisciplinary model, SBME allows the learner to hone their communication and collaborative skills.

Objective

To critically appraise competence and perception of simulation-based training amongst internal medicine residents before and after undertaking high fidelity simulation based education in three high-acuity patient scenarios (i.e. hyperkalemia, sepsis and airway management) as a means of quality improvement.

Methods

The prospective cohort study was conducted on internal medicine residents in all three years of training and analyzed through the administration of pre-and post-test questionnaires. The three high-acuity clinical scenarios were diagnosis and management of hyperkalemia, sepsis and threatened airway. Subjects in small groups of 6-9 were given a pre-test questionnaire evaluating their existing knowledge on these subjects followed by instructions in conduct of the clinical scenario. This was followed by role-play, where subjects were part of an inter-disciplinary team (critical care registered nurse, respiratory therapist, pharmacist and phlebotomist) in the conduct of the simulation based clinical scenario. The exercise was moderated remotely by the attending physician. Each simulation was followed by a debriefing session. The pre- and post-tests questionnaires were identical and aimed to assess competence and also user-rated efficacy using a Likert scale. Results of the questionnaires were analyzed using paired t-test.

Results

The number of resident encounters for sepsis, hyperkalemia and threatened airway were 46, 31 and 15 respectively. Statistically significant improvement in knowledge following SBME was noted in hyperkalemia ($p=0.02$) and airway management ($p=0.01$). Residents had a positive perception of SBME prior to the session. Following simulation training, residents felt more confident in managing patients with sepsis, hyperkalemia and threatened airway ($p=0.03$).

Conclusion

Our study showed statistically significant improvement in resident knowledge and confidence in tackling clinical scenarios pertaining to hyperkalemia, sepsis and threatened airway. Going forward, research needs to be targeted towards evaluating whether such simulation-based medical education exercises translate into improved patient outcome.

**RESIDENT ABSTRACT
POSTER PRESENTATIONS
CLINICAL VIGNETTE**

A Rare Case of Macrophage Activation Syndrome Associated With Systemic Lupus Erythematosus And Disseminated Histoplasmosis

Clinical Vignette

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Hemophagocytic lymphohistiocytosis (HLH) is an aggressive and life-threatening syndrome of excessive immune activation. HLH can occur as sporadic or familial disorder or a variety of conditions that disrupt the immune homeostasis. The most common conditions include malignancies, rheumatologic diseases, immunodeficiencies, and infections. HLH in the setting of rheumatologic disorders is termed as macrophage activation syndrome (MAS). It can occur with different rheumatologic conditions including idiopathic juvenile rheumatoid arthritis, systemic lupus erythematosus (SLE), systemic sclerosis, mixed connective disease, and others. We report a case of MAS in a patient of SLE treated with IV cyclophosphamide which was worsened by disseminated Histoplasmosis.

Case Report

A 28-year-old female with a history of SLE diagnosed 3 years ago complicated with stage V lupus nephritis, treated with intermittent azathioprine, Plaquenil, and prednisone presented with high -grade fevers to 104 F, right upper quadrant abdominal pain and nausea for two weeks. Based on the initial presentation, there was a concern for infectious etiology of the symptoms. There was no apparent source based on history and physical examination. Also, she underwent workup including computed tomography (CT) chest, abdomen, and pelvis, venous dopplers, echocardiogram, tagged white blood cell scan and magnetic resonance cholangiopancreatogram (MRCP) which were normal. The hospital course was complicated by persistent fevers, pancytopenia, worsening transaminitis (AST 141 and ALT 99) and elevated alkaline phosphatase. Further blood work showed elevated ferritin of 22,682 ug/L, triglycerides (TGs) 308 mg/dl , soluble IL-2R 12,820pg/ml, lactate dehydrogenase (LDH) 1,106 u/l and hypocomplementemia. The diagnosis of MAS was considered. Bone marrow biopsy showed normocellular bone marrow with hemophagocytosis which confirmed the diagnosis of MAS. She was started on cyclophosphamide and pulsed dose steroids with the improvement of her symptoms and blood work. So, the patient was discharged with close outpatient follow up. However, four weeks after the discharge, she was readmitted with unremitting fevers for two weeks. She also had erythematous nodules on her back, thigh , and legs. CT chest showed " tree in bud nodularity" pattern consistent with bronchiolitis/bronchopneumonia. Further blood work showed worsening serum ferritin (>40,000), LDH (1232u/l), TG (291mg/dl), pancytopenia and IL-2R (18,910pg/ml). Blood fungal cultures came positive for *Histoplasma capsulatum*. She was treated with intravenous (IV) amphotericin for disseminated histoplasmosis. This was consistent with the flare of MAS in the setting of disseminated histoplasmosis in a patient with SLE on immunosuppressants. She was treated with IV etoposide and dexamethasone for MAS. The patient showed consistent recovery of her symptoms and improvement in above -mentioned blood work.

Conclusion

HLH is a fatal disease that requires early recognition and assessment of secondary causes. *Histoplasma* has been associated as a rare trigger for MAS. Urgent treatment is required and therapeutic modalities may differ accordingly to the underlying cause.

Can You Eat Too Much Protein – A Fatal Case of Adult Onset Presentation of Urea Cycle Disorder Clinical Vignette

Loba Alam, MD; Loba Alam, MD; Asim Khan, MD; Gina LaCapra, MD; Andrew W Tarulli, MD
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Urea cycle disorders (UCD) represent a group of inborn errors of metabolism that classically affect neonates and children. In rare reports, these manifest in adulthood and are often fatal. We report a puzzling case of a 30 year old male found to have rapidly progressive encephalopathy. With no known medical history, he presented with recent tick bite one week prior to admission. He was found at home by his wife confused, lethargic, and disoriented, thus prompting urgent physician evaluation. He was found to have non-focal encephalopathy on exam, with prominent fevers, leukocytosis, and persistent hyperammonemia with levels ranging 245 - 310. Due to obtundation, he required mechanical ventilation and ICU support. His infectious workup remained negative, including lumbar puncture testing. MRI was unrevealing, and despite empiric treatment for meningitis, his comatose state did not improve.

His family revealed he had recently started non-FDA approved muscle building high protein supplements purchased on the internet. During initial trial of hemodialysis for ammonia excretion, he developed acute cerebral edema and was transferred to a tertiary care center for neurosurgical management. His intracranial pressures remained elevated, and despite normalization of ammonia with hemodialysis, he remained comatose. Based on genetic testing, he was eventually diagnosed with a novel mutation of OTC deficiency, the most common urea cycle disorder. He was eventually declared brain dead and care was withdrawn. Although more often diagnosed in the neonatal period as encephalopathy and seizures, UCD can present at any time into adulthood. The diagnosis is sometimes overlooked due to its complex and variable presentation including mild headache, vomiting, to more severe encephalopathy, seizures, and coma. Similarly, less striking features may predominate such as learning disorders, stroke-like episodes, and psychiatric manifestations. Elevated ammonia levels is a critical clue towards diagnosis in these patients. When diagnosis is suspected, a prompt consult to a metabolic specialist should be arranged. In the outpatient setting, treatment is largely prophylactic including strict dietary regulations. In the acute and urgent setting, hemodialysis provides the fastest method of ammonia excretion. Case series have also demonstrated benefit with urgent liver transplantation at a specialized center. This case demonstrates the importance of simple testing for ammonia levels in encephalopathic patients, and the need for further investigation in critically elevated levels in the absence of liver disease. A diagnosis of UCD, if treated early enough, could be life saving.

The Role of Ashwagandha in the Setting of Multiple Sclerosis Clinical Vignette

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When confronted by patients with the question of whether they can take a natural or complimentary medicinal supplement, doctors are obliged to give an answer that is supported by evidence with regards to the safety, efficacy, and quality of the supplement, as well as the potential interaction the supplement may have with other medications that the patient might be taking. Ashwagandha is an increasingly popular herbal supplement traditionally used for anti-anxiety, improved sexual function, healthier skin, and strengthened mental function. However, it has also been shown in various studies to demonstrate increased inflammatory processes and cytotoxicity, with potentially deleterious effects in patients with autoimmune diseases. We present a patient on Ashwagandha who is diagnosed with multiple sclerosis, and we were left with a dilemma when the patient insists on continuing the supplement.

A 28-year-old female with a past medical history of Celiac's disease and scleroderma presented with a chief complaint of pain and loss of vision in her left eye. She went to an ophthalmologist, who diagnosed her with optic neuritis. She was urged by the ophthalmologist to get treated and be further evaluated at the hospital. Upon initial evaluation in the emergency department, her neurological exam revealed decreased vision in her left eye, as well as diffuse hyperreflexia. MRI head showed scattered foci of high FLAIR signal in the paraventricular region, suggestive of demyelination. Lumbar puncture revealed oligoclonal bands in the CSF. She was diagnosed with multiple sclerosis and was started on steroid treatment.

Prior to admission, she was not taking any medication other than an herbal supplement called Ashwagandha, which she had started taking 6 years prior to admission and was taking 800 mg daily. Ashwagandha (binomial name is *Withania somnifera*) is an herb that is commonly found in various regions of India. It is commonly used in Ayurvedic medicine, but its uses are very non-specific. However, there has been particular concern over the use of *Withania* in patients with autoimmune diseases. The Multiple Sclerosis Society recommends patients with MS avoid Ashwagandha, as studies show that it enhances the immune system, which may lead to flare up. While there are no clinical trials done on patients with multiple sclerosis taking Ashwagandha, studies have shown that Ashwagandha stimulates the same inflammatory cell lines implicated in MS.

Upon discharge from the hospital, she was instructed to discontinue Ashwagandha. This was met with resistance from the patient, who said she uses Ashwagandha to help her relax and help relieve stress from work. After several discussions with her which involved us having to research and present several different studies, she ultimately agreed to stop taking Ashwagandha. This case highlights the importance of investigative research to determine the safety of a supplement.

Fatal Fish: A Rare Case of Botulism After Ingestion of Fermented Fish

Clinical Vignette

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AtlantiCare Regional Medical Center Program (Dominick Zampino, DO)

Botulism is a rare, life-threatening neuromuscular syndrome caused by the ingestion of *Clostridium botulinum* neurotoxin. *C. botulinum* is a spore-forming gram-positive rod that is known to inhabit surfaces of vegetables, fruits, soil, and seafood. Although uncommon, outbreaks of foodborne botulism have occurred after the consumption of fermented fish. The last outbreak to be reported in New Jersey was in 1992. Clinical manifestations of botulism vary however, descending flaccid paralysis affecting cranial nerves remains the classic presentation. Common presenting symptoms include blurry vision, diplopia, ptosis, and difficulty speaking and swallowing.

We discuss the cases of a mother and daughter who presented to the emergency department with variable presentations of botulism two days after ingesting Fesikh; a traditional Egyptian dish consisting of raw unviscerated mullet fish fermented in salt water.

Case Presentation

A 30-year-old female and her mother both presented to the emergency department with vomiting, diarrhea, and abdominal pain two days after eating Fesikh. The daughter's symptoms appeared more severe on presentation as she also complained of blurry vision and difficulty speaking. On examination, the daughter was lethargic in appearance and demonstrated difficulty with horizontal eye movements past midline. Ptosis; though noted bilaterally, was more pronounced in the left eye with slight deviation of the left pupil from midline. Although the patient's muscle strength in her tongue was intact, her speech was muffled and she complained of difficulty swallowing.

Both patients presented hypotensive on admission, however the mother's blood pressure improved after intravenous fluid resuscitation. The daughter remained hypotensive requiring vasopressor support and was subsequently admitted to the intensive care unit. Although the mother did not demonstrate any symptoms suggestive of cranial nerve involvement on admission, during her hospital stay she began to experience dysphagia and evidence of autonomic dysfunction with dizziness and orthostatic hypotension.

Poison Control was contacted on admission and initially suspected Ciguatera toxin poisoning. As our suspicion for botulism remained high given the patient's cranial nerve abnormalities and recent ingestion of Fesikh, the Center of Disease Control (CDC) was contacted and the decision was made to treat both patients for presumed botulism with heptavalent botulinum antitoxin. After administration of antitoxin, both patients demonstrated significant clinical improvement in their symptoms. Botulism was later confirmed by the CDC after stool, blood and specimens of the fish ingested were analyzed and tested for *C. botulinum* and its neurotoxin.

Discussion

When not recognized and treated promptly, botulism can be potentially fatal leading to respiratory collapse. The importance of a thorough history and physical exam are of utmost importance in early recognition of botulism. When there is a high clinical suspicion, the threshold for treating botulism should be low as delay in treatment can result in increased mortality and worsened overall outcomes.

Refractory Hyperthermia In ICU

Clinical Vignette

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Learning Objectives

Fever in critically ill patients may have various etiologies. It usually triggers a multimodal diagnostic workup. The infectious process, thrombosis or underlying malignancy should be ruled out. On many occasions, medications can be a cause of hyperthermia. One of the medications used as a sedative - dexmedetomidine. Dexmedetomidine is an Alpha2-Adrenergic Agonist that is approved to be used in ICU for sedation. Timely recognition of drug-induced causes may avoid more invasive, costly or extensive interventions

Clinical Case

37-year-old female with multiple comorbidities including DM type 1, ESRD, kidney transplant, pancreas transplant, and CAD was admitted to critical care unit due to septic shock secondary to HCAP, bacteremia, and fungemia. After several days of antibiotic therapy and vasopressors, the patient's condition improved and it was decided to wean the patient off mechanical ventilation. To achieve conscious sedation Dexmedetomidine was started. Within a few hours, the patient temperature reached 108F and remained elevated, despite discontinuation of the medication. Other possible causes of fever (such as secondary infection or venous thrombosis) were eliminated. The hyperthermic state has been refractory to antipyretics and mechanical cooling and the temperature remained elevated for 8 hours. Finally, after achieving the mark of the 98.8F, the patient became unresponsive to verbal and painful stimuli. Eventually, the patient became bradycardic, developed cardiogenic shock, requiring dopamine drip. Unfortunately, despite the effort of the medical team, the patient's condition deteriorated, she developed PEA cardiac arrest and passed away.

Discussion

There are increased incidence of reports about Dexmedetomidine as a cause of refractory severe hyperthermia. The possibility of this adverse reaction listed as relatively low (5-7%), but as seen in this case, may lead to increased morbidity and mortality. In ICU the causation of fever is usually initially contributed to the possible infectious process. It is important for providers to recognize this side effect of the medication. It is not known by what mechanisms Dexmedetomidine can cause hyperpyrexia. Some animal studies showed that Dexmedetomidine can be used to actually treat hyperpyrexia.

Pylephlebitis Presenting as a Case of Fever of Unknown Origin

Clinical Vignette

Philip Kanemo, MD; Mariam Saand; Sabena Ramsetty
Englewood Hospital and Medical Center Program (Karlene Williams, MD)

Introduction

Pylephlebitis, infective suppurative thrombosis of the portal vein, is typically associated with intraabdominal infections. It is often characterized by fever, abdominal pain, and polymicrobial bacteremia. We present a case of pylephlebitis presenting as indolent fever of unknown origin (FUO).

Case Presentation

An 82-year-old man with diabetes presented with a four-month history of fevers, drenching night sweats, thirty-pound weight loss, and vague right-sided abdominal pain. His last colonoscopy at age 52 was normal. He denied travel, sick contacts or tick exposures. Outpatient CBC and chemistries were unremarkable, blood cultures were negative, and CT scan of chest and abdomen without contrast revealed possible lung infiltrates. Patient received one week of Levofloxacin for possible pneumonia with no clinical improvement. He was admitted for further workup.

On admission, he appeared weak and diaphoretic. He was febrile to 100.5. Physical exam was only remarkable for mild right upper quadrant tenderness. Labs revealed leukocytosis of 14.74 k/ul, hemoglobin 10.6 g/dl and platelets 250 k/ul. Electrolytes and liver function tests were normal. Gallium scan was ordered and was non-diagnostic. CT abdomen/pelvis was repeated with contrast revealing extensive portal vein thrombosis. Hypercoagulable workup was negative. *Fusobacterium* was isolated from one of five sets of blood cultures drawn during hospitalization.

Metronidazole was started and patient defervesced within 48 hours. He was discharged home with 6 weeks of oral Metronidazole as well as anticoagulation.

Discussion

Pylephlebitis is a rare but serious condition which may complicate intra-abdominal conditions including diverticulitis, appendicitis, peritonitis, and rarely Crohn's disease^{2,3}. It can be life threatening if it goes undiagnosed. *Fusobacterium* is a slow growing anaerobe, and there are several reported cases documenting its association with thrombophlebitis of the portal vein as well as internal jugular vein thrombophlebitis (Lemierre's syndrome). The organism itself is thought to have thrombogenic properties³. Management of pylephlebitis typically involves a prolonged course of pathogen-targeted antimicrobials in addition to several months of anticoagulation.

Conclusion

Pylephlebitis should be considered in cases of FUO presenting with vague abdominal pain. Our case illustrates the importance of contrast imaging of the abdomen/pelvis when evaluating a patient for FUO. This should be done before nuclear imaging or invasive diagnostic tests. We also emphasize the importance of obtaining multiple blood cultures in efforts to isolate fastidious organisms such as *Fusobacterium*.

Bile Cast Nephropathy (BCN): A Journey from Severe AKI to Near Resolution of Renal Function Post-ERCP

Clinical Vignette

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Hackensack UMC Palisades Program (Sameh Elias, MD)

Introduction

Bile Cast Nephropathy (BCN)-previously known as Bile Nephrosis, is renal dysfunction in the setting of hyperbilirubinemia. BCN was reported as early as 1899 when patients with jaundice and renal failure were found to have bile cast deposition on kidney biopsies. Not many cases have been reported on this entity in the last decade due to a lack of established treatment guidelines. Our case is one of the few documenting severe renal insufficiency followed by near resolution with timely correction of the hyperbilirubinemia.

Case Report

A 67-year old Male with no past medical history presented with fatigue, decreased appetite, and dark urine for two weeks. He was found to have an AST/ALT of 170/134 IU/L, a total bilirubin/direct bilirubin of 32.2/22 mg/dl and an obstructive pattern suggested by elevated Alkaline phosphatase > 763 IUL/L. He had an acute kidney injury (AKI) with BUN/Cr of 115/7.08 mg/dl and a urine analysis demonstrating no proteinuria, dysmorphic RBC's or Eosinophils. Urobilinogen however was elevated at 4 EU/dL. A renal ultrasound did not reveal any signs of obstructive uropathy. Ultrasound of the abdomen revealed gallbladder wall thickening and biliary ductal dilatation. MRCP confirmed the presence of a 1.1 cm distal CBD stone with proximal intrahepatic and extrahepatic biliary ductal dilatation. Patient underwent an ERCP with sphincterotomy and stent placement. Following the ERCP, renal function improved over the next 2-3 days to near normalization upon discharge.

Discussion

Only a handful of cases were reported until van Slambrouck et al published a study of 44 patients with BCN in 2013. While the exact etiology of BCN remains unknown, it is presumed to be secondary to multiple concurrent insults from direct toxic effects of bile salts on the tubular epithelium, leading to proximal tubulopathy. A direct mechanical intratubular cast obstruction and systemic hypoperfusion from bilirubin-induced-vasodilation has also been proposed as a causative mechanism. There should be a high index of clinical suspicion for BCN in patients with hyperbilirubinemia and concomitant AKI. BCN is characterized using light microscopy to identify the presence of bile casts within renal biopsy. Although Renal biopsy is the solitary means of definitive diagnosis, it is often too invasive. UA with characteristic bile-stained granular casts which are pathognomonic for BCN and acute tubular necrosis (ATN), is a simple and inexpensive method and can be highly suggestive of the disease, if renal biopsy is contraindicated. Of note, muddy brown granular casts may be absent in 20–30% of patients with ATN. Treatment should be targeted at improving hepatic dysfunction and relieving the biliary obstruction without delay. This may improve renal function, but the patient may still need temporary dialysis or plasmapheresis if renal function fails to improve following treatment directed at the underlying cause of hyperbilirubinemia.

How Low Can You Go? Severe Hyponatremia With a Sodium of 94 mg/dL Complicated By Retroperitoneal Hemorrhage

Clinical Vignette

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Introduction

Hyponatremia is commonly defined as a serum sodium concentration below 135 meq/L. It is the most common electrolyte disorder in medicine that can range from a myriad of different etiologies ranging from drug induced to SIADH. Management of hyponatremia is determined by level of sodium, symptomatology, and etiology. Severe hyponatremia is classified as sodium levels of less than 120 with symptoms including confusion, seizures, and coma. Management is difficult as correcting too rapidly can cause severe neurological impairment, most notably causing osmotic demyelination syndrome.

Case Report

An 83-year-old male with a past medical history of Diabetes Mellitus Type 2, benign prostatic hyperplasia, and hypertension presented with nausea and vomiting for three days. Oral intake was poor, but he reportedly drank 18 glasses of water the day prior to admission and was recently started on hydrochlorothiazide for hypertension. Physical exam was benign, but serum sodium was 94 mg/dL. Patient was monitored in the intensive care unit for sodium correction with fluid restriction, and concomitant 3% hypertonic saline and Desmopressin. Hypertonic saline was administered as 50 cc/hr boluses with 2 mg of desmopressin q6h for the first 3 hospital days until Na was 105 mg/dL.

Hospital course was complicated by retroperitoneal hemorrhage and hypovolemic shock secondary to enoxaparin use. A resultant acute tubular necrosis further complicated the initial management of fluid restriction. Patient was transfused 3U of packed red blood cells, and bleeding managed conservatively. Patient was discharged without long-term neurologic or renal complications and a sodium of 136 mg/dL.

Discussion

Literature review presents only one other case of a more severe hyponatremia with a sodium of 87 mg/dL.¹ In contrast, our patient had no long term neurological complications nor renal replacement requirements. To our knowledge, we present the first case of severe hyponatremia complicated by the need for multiple blood transfusions and fluid boluses with a successful outcome of treatment with the infrequently used strategy of concomitant hypertonic saline and desmopressin.² The extreme hyponatremia in this patient was multifactorial secondary to severe ongoing sodium and water loss from vomiting, large amount of water intake, recent initiation of thiazide diuretic, low dietary solute intake, urinary retention, and SIADH. Management was further complicated as patient developed retroperitoneal hemorrhage secondary to DVT prophylaxis and needed blood transfusion. Blood transfusion can often times lead to further electrolyte imbalance affecting such entities such as sodium. This case demonstrates the medical management of sodium level that often times can lead to death and further complicated with bleeding and necessity of blood transfusion, which was effectively treated and had the patient suffer no long term neurologic or renal injury.

SIADH or CSWS? A Case of Severe Tolvaptan-Resistant Hyponatremia Clinical Vignette

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Introduction

Syndrome of inappropriate antidiuretic hormone (SIADH) is characterized by inappropriate production of antidiuretic hormone (ADH) leading to free water retention. Cerebral salt wasting syndrome (CSWS), on the other hand is extracellular volume depletion due to a tubular defect in sodium transport caused by excess secretion of natriuretic peptides. Loss of sympathetic stimulation to the kidneys is a partial component. The term CSWS was introduced in 1957 prior to the description of SIADH. Subsequently, CSWS virtually vanished, only to reappear a quarter century later in the neurosurgical literature. SIADH is a common electrolyte disorder seen in hospitalized patients, whereas CSWS is often overlooked as it is an 'uncommon' diagnosis.

Case Report

A 69-year-old male presenting with dyspnea was incidentally found to have severe hyponatremia and decreased serum osmolality (Na of 115 mmol/L and Osmolality 254 mOsm/L) on admission. Over the course of his hospitalization, he was treated for SIADH with fluid restriction, Prostat, KCl, and NaCl supplementation without improvement in serum sodium or osmolality (serum sodium ranged from 115-122). Urine sodium remained elevated (> 180) along with urine osmolality (highest > 990) suggesting severe SIADH. Patient was trialed on Tolvaptan which failed to normalize his serum sodium, followed by Furosemide without any success. A diagnosis of CSWS was entertained as the patient had been hypotensive since admission. CT head did not reveal any acute abnormalities. Oral Fludrocortisone therapy at 0.1 mg/day was eventually started along with a total of 16 L normal saline. Serum sodium and osmolality then normalized over the following week, confirming the clinical suspicion of CSWS rather than Tolvaptan resistant severe SIADH.

Discussion

Our review of the current literature yielded several cases of Tolvaptan-resistant severe SIADH, which can make recognition of CSWS difficult. The distinction between the two entities is, however, critical because of opposing therapeutic goals - fluid restriction vs. fluid resuscitation. As with our patient, treatment of CSWS can be achieved with a combination of isotonic saline/hypertonic saline with the addition of a mineralocorticoid. Another crucial factor in distinguishing CSWS is the accurate clinical ability to identify volume depletion and an appropriate ADH response. This can, however, be inconsistent from patient to patient. Inappropriate urinary salt losses and reduced effective arterial blood volume may aid in delineating between the considerable overlap in the laboratory findings of SIADH and CSWS. Keep in mind that both syndromes are associated with intracranial pathology, have normal renal, thyroid, and adrenal function. These conditions need to be worked up prior to the consideration of SIADH or CSWS.

A Challenging Case of Neurosarcoidosis Masquerading as Recurrent Transient Ischemic Attacks

Clinical Vignette

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Introduction

Neurosarcoidosis is an extremely rare presentation of systemic sarcoidosis, with only 4-6% of cases having neurological involvement. The neurological manifestations are diverse based upon distribution of lesions in CNS and is a significant cause of mortality and morbidity in patients with sarcoidosis.

Case Description

A 56-year-old man with history of recurrent transient ischemic attacks on aspirin and statin, presented in emergency department for tingling and numbness of left arm and leg for the past 3 days. Patient also complained of diplopia for the past 2 weeks. Neurological exam revealed right abducens nerve palsy, decreased sensations in left arm, and left leg. CT scan of head was negative for ischemia or bleed. Patient was admitted and started on non-TPA stroke protocol. Brain MRI done later, showed nonspecific white matter hyperintense lesions. MRA head and neck and echocardiogram were unremarkable. Since the patient had recurrent and incongruent neurological symptoms, we searched for an alternative diagnosis. Cervical, thoracic and lumbar spine MRI, to rule out multiple sclerosis, revealed small intradural enhancing lesions along nerve roots suggestive of carcinomatous meningitis, lymphomatous meningitis or granulomatous disease. CSF analysis showed increased opening pressure, increased proteins, leukocytosis and was negative for ACE, acid-fast bacillus and cytopathology. CT scan of chest/abdomen, to rule out underlying malignancy, showed hilar lymphadenopathy with multiple pulmonary nodules. Lymph node biopsy showed non-caseating granulomas suggestive of sarcoidosis. A diagnosis of neurosarcoidosis was made. Patient was started on prednisone with marked improvement in neurological symptoms and was subsequently discharged home.

Discussion

Systemic sarcoidosis rarely presents with neurological symptoms as the first manifestation of disease. Sarcoid lesions in CNS usually cause cranial neuropathies, vasculitis, meningitis and rarely meningeal mass lesions as in this case. Patients who develop neurological disease consistent with neurosarcoidosis, but with no prior diagnosis of sarcoidosis, pose a diagnostic challenge. Imaging and CSF analysis are primarily done to rule out other differentials, while diagnosis relies mainly on clinical suspicion. Antiplatelet therapy used for prevention of stroke does not help in preventing recurrent neurological symptoms of sarcoidosis. Immunosuppression mainly with steroids is the first line of treatment, which can alleviate the disabling symptoms and prevent future complications.

Conclusion

This case raises the awareness about this rare but serious presentation of a clinically known entity, Sarcoidosis. It is important to keep a diagnosis of neurosarcoidosis high on the list of differential in patients with recurrent strokes refractory to medical therapy, especially if they have no vascular risk factors. This will help in proceeding with the appropriate investigations, leading towards the diagnosis. Timely administration of steroids is the key to treatment and prevention of recurrence, making neurosarcoidosis a treatable cause of stroke.

“Liquid Presenting as Solid:” A Rare Presentation of Acute Myeloid Leukemia as a Solid Epidural Mass

Clinical Vignette

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Learning Objectives

Recognize that acute myeloid leukemia (AML) can present as a solid extramedullary mass termed myeloid sarcoma/chloroma.

Case Summary

59-year-old male with history of diabetes, atrial fibrillation, and lower back pain worsening for 2-3 months presented with sudden onset paraplegia, lower extremity numbness and urinary incontinence. EXAM: bilateral lower extremity muscle strength 0/5, absent sensation to pain and touch, and absent deep tendon reflexes. Upper extremity and cranial nerve exams were normal. LABS: Hemoglobin 14.1 gm/dl, WBC 14.8 k/ul, PLT 162 k/ul, HCT 42.6%, sodium 135 mmol/l, creatinine 0.91 mg/dl. Magnetic resonance imaging of the spine revealed epidural masses extending from the level of T7 to T10 with spinal stenosis and cord compression, and another epidural mass at the L2 vertebra. He underwent laminectomies and resection of the masses. Biopsy of L2 mass showed high grade malignant neoplasm invading the skeletal muscle consistent with chloroma. Immunophenotyping was positive for CD45, CD117, CD43 and MPO stains, and negative for CD34, consistent with AML. Bone marrow biopsy was consistent with AML as flow cytometry showed blasts accounting for 77% and positive for CD11c, CD4, CD33, HLA-DR, CD117, CD13, CD38, and MPO. Fluorescence in situ hybridization (FISH) testing was negative. Molecular profile was consistent with NPM1 mutation, indicating good prognosis. The day after resection of the epidural mass his pain resolved, but the numbness and weakness in his legs persisted. He was transferred to an advanced cancer facility for induction chemotherapy.

Conclusion

Myeloid sarcoma or chloroma is a rare (<1%) extra-medullary manifestation of AML which can present anywhere in the body including muscle, skeleton, and CNS. The clinical manifestations and complications are related to the location and size. Typically they present concurrently with active leukemia and evidence of concurrent hematological disease. Secondary chloroma can occur following remission. Since chloromas are frequently misdiagnosed as lymphoma, a thorough evaluation including immunohistochemistry, flow cytometry, FISH, and molecular analysis is needed. Early diagnosis and treatment is associated with a significantly lower probability of developing AML and with longer survival. Markers like MPO, CD117 and CD68PG-M1 support the diagnosis. Current guidelines recommend initial treatment with induction chemotherapy. This case highlights the need to consider chloromas in the differential diagnosis of solid tumors, even in the absence of hematological abnormalities.

Relapsing Invasive Salmonella Typhi Infection with Bone Marrow Suppression in an Otherwise Healthy Young Female

Clinical Vignette

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Typhoid fever caused by *Salmonella typhi* is one of the reportable diseases in the United States. Only less than 400 new cases were recorded in 2014 due to advances in sanitation through the years. *Salmonella* serovars *typhi* and *Paratyphi* account for less than 1% of an estimated 1.2 million cases of *Salmonella* infection in the United States annually. We present a unique case of relapsing invasive *Salmonella* serovar *typhi* infection after initial clinical response to ceftriaxone.

A 26-year-old female with no past medical history relocated from India to the United States five days prior to admission. On her second day in US, she had fever with shaking chills, nausea, bifrontal headaches, abdominal cramps, and few episodes of watery diarrhoea associated with vomiting. She recalled eating egg and beef at a restaurant in India prior to her travel. Physical examination revealed fever of 103 F with dry oral mucosa. Labs showed pancytopenia, increased LDH and reticulocyte count. Her Malaria smear was negative and the tests for Dengue NS-1, Chikungunya, Lyme, and Brucella antibody were done which all turned out negative. Within 72 hours, initial blood cultures stained for gram negative rods, soon identified as *Salmonella typhi*. Magnetic resonance angiography ruled out aortitis and transthoracic echocardiography ruled out endocarditis. She was started on empiric IV ceftriaxone and after 5 days of therapy and was subsequently discharged on amoxicillin-clavulanic acid for 10 days. However, a month later, she developed a relapse, presenting with fatigue, headache, nausea, watery stools, high grade fever, chills, and anorexia. Blood cultures confirmed the presence of *Salmonella* species and therefore readmitted for relapse of invasive *Salmonella typhi* enteritis. She underwent midline insertion upon documented bloodstream clearance of infection. She had significant clinical improvement and hence discharged on IV ceftriaxone to complete a total of 21 days of therapy. She was asymptomatic when she followed up after 6 weeks.

An estimated 5,700 cases of *Salmonella typhi* infection occur in the United States each year. Three of four cases occur in international travelers (CDC, 2018). It is potentially fatal with 50% recurrence rate and about 2-5% become chronic carriers and therefore at risk for relapse (WHO, 2018). Due to advances in sanitation, typhoid fever is now rare in the US and the latest available results showed only 329 new cases in the US in 2017. Cultures are considered the gold standard for detecting pathogens in patients with invasive infections but however, the diagnostic yield is only 50% and stool cultures may not be positive until the second week of illness. Furthermore, emerging resistance to ceftriaxone, fluoroquinolone, and cotrimoxazole has led to heightened national vigilance. Despite adequate therapy, patients can develop a relapse, therefore close follow up is imperative.

What are the ODS? A Case of Osmotic Demyelination Syndrome Associated with Hemodialysis in End-Stage Renal Disease
Clinical Vignette

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Osmotic demyelination syndrome (ODS) is the irreversible demyelination of the central pons, among other areas of the brain, that is most often associated with rapid correction of hyponatremia. Per contra, reversible ODS is a rare state that can be seen in patients with end-stage renal disease (ESRD) on hemodialysis (HD). This clinical case offers an alternative problem representation for acute altered mental status in a patient with ESRD on HD.

A 76-year-old African American female with a past medical history of ESRD on HD, diabetes mellitus, hypertension and hyperlipidemia presented to the ED with new-onset aphasia and confusion. Patient is compliant and completed dialysis that morning. Home medications include glipizide, insulin, amlodipine, metoprolol, and simvastatin. Vitals at presentation were within normal limits except a blood pressure of 195/86. At admission, the patient exhibited global aphasia and apraxia, but moved all four extremities spontaneously. GCS was 9 and NIHSS was 7, later increasing to 14. Hemoglobin was 12.7, and sodium was 138. Recent outpatient labs were normal. CT head, CT angiogram, and CT perfusion did not show acute intracranial ischemia or hemorrhage. However, MRI brain showed diffuse increased T2 signal intensity within the pons, suggestive of osmotic demyelination. Aspirin 325mg was administered, and aspirin and high-intensity statin were continued for concern of cerebral vascular accident (CVA). EEG was negative for seizure. There were no active signs of infectious or autoimmune etiology. Urine drug screen was not performed due to anuria. Repeat MRI and MRA brain/head/neck was negative for CVA or reversible posterior leukoencephalopathy syndrome, but continued to show osmotic demyelination in the pons. Sodium remained between 135 and 141. Supportive treatment was initiated without antibiotics or steroids: blood pressure was kept normotensive, electrolytes were kept within normal limits, and fluid shifts were minimized during HD. The patient spontaneously recovered, and at discharge six days later, she was at her neurologic and cognitive baseline. Aspirin was discontinued, and home statin was continued.

Rapid correction of hyponatremia >10-12mEq in 24 hours is the most common cause of ODS, but case reports have also shown ODS in patients undergoing dialysis; these case reports correlate with our patient with global aphasia and apraxia. Treatment of this type of ODS consists of minimizing fluid shifts during HD and maintaining normotension. The patient had three HD sessions in her hospital stay, two of which were filtration without fluid exchange. Extensive workup was negative for other etiologies, and symptoms resolved without other intervention. Although rare, HD-associated ODS should be considered in the problem representation of altered mental status in patients with ESRD in the right clinical context. It is imperative that more research is conducted on its cause and prevention in the future.

Mysterious Hypothermia in Psychiatric Patient

Clinical Vignette

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Hyperthermia is a well known and well documented side effect of antipsychotic medications. However, hypothermia, although almost as deadly, is not nearly as well known of a side effect in antipsychotic medications.

A 68-year-old man presented to the emergency department from a nursing home due to increasing lethargy and hypothermia. The patient has a history of schizoaffective disorder for which he was receiving oral risperidone and paliperidone monthly injection. This patient had been hospitalized for lethargy and hypothermia twice earlier in the year, coincidentally after receiving paliperidone injection 2 and 3 days before the hospital admissions respectively. This patient's other medical issues included hypertension, hyperlipidemia, chronic kidney disease stage 3, prostate cancer post radiation therapy. In addition to risperidone and paliperidone, he was also concurrently taking aspirin, atorvastatin, benztropine, bupropion, divalproex, furosemide, tamsulosin, and leuprolide. In the ED, patient was arousable to voice but lethargic and fell asleep easily. His rectal temperature was 91.2 F (32.8°C); pulse 88 bpm; blood pressure 135/73 mm Hg; respirations 16/min; oxygen saturation 98% on room air. His physical exam was benign. Common causes of hypothermia were ruled out. There was no cold environment exposure, toxin exposure, thyroid dysfunction, hypoglycemia, hypopituitarism, hypoadrenalism, or malnourishment. CT head did not show evidence of CVA or hemorrhage. Chest x-ray was normal. WBC, procalcitonin and lactic acid level were normal. The patient was treated with warm blankets and warm intravenous normal saline. He was also started on broad spectrum antibiotics but they were stopped as soon as the blood and urine cultures resulted as negative. After discussion between medicine, neurology and psychiatry, it was concluded that paliperidone was the most likely cause of the patient's hypothermia with the Naranjo Adverse Drug Reaction (ADR) Probability Scale score 6. All antipsychotic medications were stopped. The patient's temperature was stable at 97-98 F (36.1°C - 36.7°C) after these interventions. His lethargy resolved more slowly and caused him to require intubation for 3 days but he subsequently returned to baseline mental status. His antipsychotic medication was switched to haloperidol. One month after the hypothermic episode, the patient was at baseline functioning without any lethargy or hypothermia.

Hypothermia induced by antipsychotic drugs is not novel, but paliperidone-induced hypothermia is rarely reported in literature. This case shows that hypothermia can present as a rare side effect of long-acting paliperidone even after a relatively prolonged administration (greater than 1 year). It is important to consider adverse drug effect in the differential list of hypothermia in patient with history of psychological disease and antipsychotic drug use, regardless of the drug intake duration. Recognition of such processes will allow clinicians to stop the offending drug earlier in the disease course.

**“A Big MAC or a Heart Attack?” Massive Mitral Annular Calcification in a Young Male with ESRD:
A Forgotten Complication
Clinical Vignette**

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Introduction

Mitral annular calcification (MAC), a representation of dystrophic calcification is associated with advanced age, female gender, coronary artery disease and is a common cardiac finding on autopsy. A premature form of MAC occurs in patients with ESRD and prevalence is estimated to be as high as 40%. To reiterate the importance of this often-missed clinical entity, we present a case of accelerated MAC in a young man with ESRD.

Case

A 37-year-old male with ESRD on hemodialysis, hypertension and subtotal parathyroidectomy was brought to the ED for chest pain. The pain had started 30 minutes prior, was pressure-like, localized to the precordium and non-radiating.

BP was 188/125 mmHg, bilaterally equal, HR was 86/min and regular, RR was 16/min and SpO₂ was 94% on room air. A IV/VI holosystolic murmur was appreciated at the mitral area with radiation to the axilla. Bibasilar pulmonary crackles were heard and an AV fistula on the left forearm was observed.

Investigations revealed an abnormal metabolic panel (sodium: 131 mmol/L, potassium 3.7 mmol/L, BUN 54 mg/dL, creatinine 12.2 mg/dL), corrected calcium of 8.9 mg/dL and phosphorous of 7.5 mg/dL. Calcium-phosphorous product was 66.75 mg²/dl² (Normal: <55 mg²/dl²). Troponin was 0.14 ng/mL.

Electrocardiogram met criteria for LV hypertrophy and LA enlargement with no ST-T wave changes. Chest x-ray revealed MAC. He was treated with aspirin, clopidogrel, atorvastatin and heparin drip. Repeat troponin 6 hours later was 0.15 ng/mL. Echocardiography showed a dilated LA (57 mm), severe posterior mitral annular calcification with restricted leaflet opening, severe mitral regurgitation, concentric LV hypertrophy, diastolic dysfunction and an EF of 65%. Coronary angiography showed no significant occlusion. The chest pain was attributed to coronary steal secondary to severe mitral regurgitation. Given the severity of his MR, the patient underwent open heart surgery for decalcification of the annulus with annular reconstruction and valve replacement.

Discussion

MAC is not a benign entity and carries the risk of complications including valvulopathy, embolization, atrioventricular block and infective endocarditis. Initially thought to be a result of high ventricular systolic pressures associated with systemic hypertension and the resultant degeneration, the etiology appears to be more intricate with a strong link between its prevalence and dysregulation of calcium-phosphorous metabolism typical of ESRD. High calcium-phosphorous product precipitates calcium in the vulnerable tissue of the mitral annulus. Calcification follows the curve of the annulus with relative sparing of the anterior leaflet and commissures, distinguishing it from rheumatic mitral disease. The annular diameter is increased (not decreased) leading to regurgitation. MAC remains under diagnosed and has a 5-year survival of 47%. Given the poor prognosis, early identification and intervention is imperative for a chance of better outcome.

A Left to Right Presentation of Guillain-Barre Syndrome

Clinical Vignette

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Introduction

Guillain-Barre Syndrome (GBS) is an acute inflammatory polyneuropathy characterized by weakness and areflexia. The severity and morbidity of the disease warrant a thorough understanding of presentation to aid in prompt diagnosis and initiation of treatment. Herewith we present a case of GBS with left to right presentation of paralysis.

Case Description

A 61-year-old male with a past medical history of hypertension and depression presented to the emergency department with progressive weakness in his left arm and left leg. One month prior to his presentation he was gardening when he injured his 3rd digit with a metallic foreign body. He removed the foreign body and developed redness and swelling of the digit. He was evaluated at an urgent care and started on doxycycline and given a tetanus booster for presumed infection in the affected arm. Two weeks after injury he started to have left arm weakness. The weakness progressed to the left lower limb followed by right lower limb and then right upper limb. Physical examination revealed areflexia in the left arm and left lower limb and reduced reflexes in the right arm and right leg. The patient had 2/5 strength in the left hand and foot. The patient was admitted to the ICU for management where his condition continued to deteriorate over the next several days. He progressed to complete flaccid paralysis with areflexia. GBS was suspected at this time and the patient was started on IVIG. A lumbar puncture showed elevated protein levels and a normal cell count consistent with the diagnosis of GBS. The patient underwent five rounds of IVIG with no improvement. During the hospital course the patient continued to deteriorate and required intubation. Treatment was switched to plasmapheresis and the patient underwent 5 rounds. The patient remained intubated for 17 days with no improvement in respiratory function or muscle strength. He underwent a tracheostomy and was transferred for further management at an outside facility.

Discussion

This patient's left to right presentation of paralysis progression is a unique variation for GBS. The patient's etiology is likely related to the foreign body or the tetanus booster that he received. Identifying unique presentation patterns such as this and making a timely diagnosis aid in initiating treatment regimens. Early management can help to reduce the disease burden, course and provide high value care.

Comedy to Tragedy: A Laughing Gas Induced Ischemic Stroke

Clinical Vignette

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Introduction

Hyperhomocysteinemia has been associated with increased risk of systemic atherosclerosis, cardiovascular disease and stroke. Hyperhomocysteinemia can result from impaired homocysteine metabolism resulting from dietary deficiency of folic acid, vitamin B6, vitamin B12 or due to genetic predispositions like MTHFR C677T gene polymorphism. Nitrous oxide use can also lead to hyperhomocysteinemia and here, we report a rare case of young male who presented with acute ischemic stroke after five years of nitrous oxide abuse.

Case

A 32-year-old male with known history of bipolar disorder presented to the emergency department with altered mental status, slurred speech and motor weakness on left side of the body complicated by seizures. He was immediately intubated for protection of airways and transferred to intensive care unit. Non contrast CT of the head was significant for an infarct in the right fronto-parietal region of the brain. CT angiogram of head and neck revealed non occlusive thrombus in right internal carotid and middle cerebral artery. The etiology of stroke was unclear at this time in this young patient. There was no personal history of known thrombophilia and family history was not available as he was adopted. Laboratory investigation was positive for macrocytic anemia and leukopenia. Vitamin B12 and Folate level was found to be low (198 pg/ml, and 2.5 ng/ml respectively). Methylmalonic acid level was in normal range (0.12 mcmol/L; reference range 0.0-0.4 mcmol/L) while homocysteine level was elevated (253 mcmol/L; reference range 0-10 mcmol/L). Laboratory investigations were significant for hyperhomocysteinemia, which led us to gather more history in order to understand its etiology, which ultimately revealed repeated use of inhaled nitrous oxide as recreational agent for the past five years with last use being 4 hours prior to presentation. Patient is doing well on follow up.

Discussion

An increased plasma total homocysteine level is an independent risk factor for vascular events. It leads to endothelial injury promoting atherogenesis. The underlying metabolic mechanism is based on the fact that N₂O induces oxidation of vitamin B12 and the oxidized form inhibits methionine synthetase resulting in accumulation of homocysteine. It can have serious clinical implications including myocardial ischemia, stroke, peripheral neuropathy and dementia. Heavy intake of nitrous oxide in the form of energy drinks could also be a risk factor for hyperhomocysteinemia resulting in thrombotic events. Our case supports the association of nitrous oxide and hyperhomocysteinemia with thromboembolic events and therefore, a thorough history taking can be pivotal to uncovering the underlying pathology responsible for the clinical picture.

The Heart Slows On Yellow: Hyperbilirubinemia Induced Bradycardia

Clinical Vignette

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Introduction

In 1877, Murchison commented on jaundice noting, "in a large proportion of cases, the pulse is unusually slow." Up until 1972, this phenomenon merited mention in medical textbooks. However recent references are limited and it has become an association commonly overlooked.

Case

A 64-year-old man presented to the emergency department with syncope. Earlier that day, he experienced cramping upper abdominal pain that lasted an hour. He visited an urgent-care clinic where he felt lightheaded before losing consciousness. He had no jerking or oral frothing and recovered spontaneously within a minute. He was thus sent to the emergency department. On examination, he was lying comfortably in bed. Pulse was 39 beats/minute, regular and normovolemic. Blood pressure was 116/52 mm of Hg, evaluation for orthostasis was negative and temperature was 98.3oF. His conjunctivae were icteric. S1 and S2 were normal with no murmurs or gallops. Abdominal examination revealed no tenderness.

Serum chemistry revealed aspartate aminotransferase 342 U/L, alanine aminotransferase 470 U/L, alkaline phosphatase 145 U/L and total bilirubin 4.8 mg/dl (direct bilirubin: 3.1mg/dl), a pattern consistent with obstructive jaundice. Electrocardiogram showed new sinus bradycardia (43 beats/min), 1st degree AV block (PR interval 216 ms) and RSR' in lead V1.

Ultrasonography showed cholelithiasis and a 4mm wide common bile duct without choledocholithiasis, confirmed on magnetic resonance cholangiopancreatography. Liver chemistries returned to normal during hospitalization suggesting obstructing choledocholithiasis which had since passed. As the hyperbilirubinemia resolved, the heart rate began to rise. Repeat electrocardiogram 22-hours after the initial showed heart rate of 52 beats/minute, reversal of the 1st degree A-V block (PR interval 190 ms) and persistence of RSR' in V1. He underwent a laparoscopic cholecystectomy the same admission. At discharge his resting heart rate was 82 beats/minute and total bilirubin was 0.9 mg/dl. His electrocardiogram at outpatient visit 2 months later showed a heart rate of 71 beats/minute, PR interval of 134 ms and unchanged RSR' in Lead V1.

Discussion

The etiology of syncope was likely sinus and A-V nodal dysfunction secondary to hyperbilirubinemia. Joubert et al ligated bile ducts of rats and found that post-ligation, the mean heart rate decreased from 374 (SD= 21) beats/min to 348 (SD=11) beats/min (P<0.0005). Electrocardiogram of these rats showed a spectrum from bradycardia to varying degrees of A-V block. Bogin demonstrated similar mechanisms by adding the serum of rats whose bile ducts had been ligated to cultured myocardium. The deoxycholate and cholate rich serum decreased heart rate and led to early cessation of contractions. A literature review reveals this association only holds true with obstructive jaundice. This case highlights the cardio-toxic properties of bilirubin and its potential to induce nodal dysfunction. Indeed, like a model citizen, the heart too slows on yellow.

Thrombotic Thrombocytopenic Purpura Masquerading as NSTEMI

Clinical Vignette

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Introduction

Though Thrombotic Thrombocytopenic Purpura is a rare disorder estimated to occur in approximately 3 cases per 1 million adults, it remains a medical emergency when encountered as it carries a mortality rate of about 90% if left untreated.

An 86-year-old female who presented with complaints of chest pressure of a day duration and heaviness behind her eyes of 3 days duration. She also reported a transient 15-minute numbness of the left arm which prompted her to present. Shortness of breath, palpitations, nausea, lightheadedness at home but had lightheadedness in the ED. Of note, she was on Cefuroxime for sinusitis and also had developed spontaneous bruising over her left arm and thigh few days prior to presentation.

Past medical history significant for hypertension and hyperlipidemia. Patient was a non-smoker, drank 1 glass of wine daily. Her initial vitals were within normal limits except for BP of 167/92 mmHg. Physical examination only revealing of multiple bruises on extremities and bilateral pitting pedal edema.

Initial bloodwork:

Hb-10.4g/dl, WBC-5.1x10³cumm, Platelet-6x10³cumm, BUN-23mg/dL, Creatinine-0.72

Na-125 mmol/L, K-4.0mmol/L, Cl-94mmol/L, CO₂-25 mmol/L, Alkaline Phosphatase-50U/L,

AST-89U/L, ALT-33 U/L, Total Bilirubin-2.3mg/dl, Creatine Kinase- 465U/L, CK-MB Fraction-78.2U/L, Troponin-I: 5.190ng/dl

Patient was admitted to the ICU for NSTEMI and possible ITP or Cefuroxime-induced thrombocytopenia and hemolysis. Hematology and cardiology were consulted. She was started on IV Methylprednisone, high intensity statin only as anticoagulation was contraindicated; also 2% NaCl for suspected SIADH. Further hemolytic work-up were suggestive of intravascular hemolysis but no schistocytes on peripheral smear. Troponin levels trended down but thrombocytopenia persisted.

On the third day, patient suddenly became confused and agitated, initially attributed to a combination of steroid use, hyponatremia and ICU delirium. CT head done was negative for a bleed or acute infarct. ABG showed: pH: 7.38, pCO₂: 25, pHCO₃:15, pO₂: 86. Mental status worsened with each hour with patient becoming obtunded within 6 hours and had to be intubated to protect the airway.

At this time, TTP was suspected given the persistent thrombocytopenia (platelet count-8000), hemolytic anemia (Hb: 9.6, indirect bilirubin:2.1) and severe neurological findings. A repeat peripheral smear revealed schistocytes. Patient was planned for a possible plasma exchange; however, patient had a cardiac arrest before the same could be done and died. ADAMTS13 assay sent on Day 3 returned a week later at <3.

Discussion

TTP is caused by severely deficient activity of the ADAMTS13 protease, clinically defined as an activity level <10 percent. The classical presentation is a pentad but more commonly presents in myriad sequence or form thus requiring a high index of suspicion and emergent plasmapheresis to prevent an almost certain fatality.

A Bone Scan Could Save a Lung Biopsy?

Clinical Vignette

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Introduction

Metastatic pulmonary calcification (MPC) is a known complication of end stage renal disease. It can also occur secondary to derangement of calcium and phosphate homeostasis due to other metabolic causes. Variable clinical and radiographic presentations often make the diagnosis challenging. Failure to consider MPC in the differential diagnosis might culminate into unnecessary testing, invasive procedures and eventual patient morbidity.

Case Presentation

A 63-year-old female with hypertensive end-stage renal disease (ESRD) status post three renal transplants presents to the ED with a non-productive cough and exertional dyspnea of three months duration. Despite several courses of outpatient antibiotics her symptoms had failed to resolve. Past history included thyroidectomy and partial parathyroidectomy. The patient denied fever, night sweats, and sick contacts. Medications included tacrolimus, mycophenolate, prednisone, vitamin D and calcium. VS BP138/80, HR 97, RR 20, T 98 F and O2 94% on 2L. Examination: Wheezing in bilateral upper lung zones. Lab findings: Calcium 12 mg/dl, Phosphorus 4.3 mg/dl, Creatinine 1.52 mg/dl. Chest Xray was normal. CT chest: bilateral upper lobe ground glass opacities. Pulmonary function test: mild restrictive pattern with reduced DLCO. Patient's symptoms remained unchanged with antibiotic therapy with worsening findings on repeat CT scan. Bronchoscopy was negative for infection. Patient underwent VATS and biopsy showed metastatic pulmonary calcification with interstitial inflammation. Calcium level normalized after discontinuing Vitamin D and calcium supplements. She was discharged on home oxygen with lung transplant team follow up.

Discussion

MPC is commonly seen in patients with ESRD, especially patients on dialysis. It has also been described in patients following orthotopic liver transplant, hypercalcemia of malignancy, primary hyperparathyroidism, parathyroid carcinoma and multiple myeloma. Interestingly, the deposition of calcium occurs most commonly in the upper lung due to alkaline environment. An elevated calcium-phosphate product $>70\text{mg}^2/\text{dL}^2$ is often seen.

Although a known entity, pre-mortem diagnosis of MPC can be difficult. Patients are often asymptomatic with abnormalities detected incidentally on chest radiograph. Cough is a common symptom. Rarely severe progressive calcification can result in restrictive ventilatory defect, hypoxemia and subsequent respiratory failure. Chest X-rays are usually normal. CT findings may include multiple diffuse calcified nodules, diffuse or patchy areas of ground-glass opacity or patchy parenchymal consolidation, predominantly involving upper lobes. Radiologic differential diagnosis includes hypersensitivity pneumonitis, atypical infections, sarcoidosis and workplace/environmental exposure. When in doubt, a technetium-99m isotope bone scan which demonstrates increased uptake at the affected areas could be diagnostic and obviate the need for lung biopsy. Treatment might require renal transplant for ESRD or identification and correction of the underlying metabolic disorder.

Conclusion

Consideration of MPC in the right clinical scenario is extremely important. Early and appropriate diagnosis of MPC could potentially prevent significant patient morbidity and save unnecessary medical expenditure.

It's Very Safe, Until It Isn't

Clinical Vignette

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Introduction

Acupuncture has emerged as an increasingly popular choice for complementary and alternate approaches to conventional medical treatment. More than 10 million acupuncture treatments are administered in the United States alone. The procedure is generally considered safe but a variety of complications have been reported from time to time. We report a case of serious bilateral pneumothorax following acupuncture.

Case Presentation

A 53-year-old Caucasian male presented to the ER with progressively worsening shortness of breath following an acupuncture treatment earlier in the day. Shortly after the procedure, he developed a dull tightness in his upper back and difficulty in breathing while speaking. He had received regular acupuncture treatments from the same practitioner over the past seven years for chronic back pain. Physical examination was remarkable for bilaterally reduced breath sounds. Chest radiography showed moderate right sided pneumothorax and a small left apical pneumothorax. CT scan showed bilateral moderate to large pneumothorax and a slight shift of the mediastinal contents from the right to left. Bilateral thoracostomy tubes were placed with Seldinger technique using the Thal-Quick Set and repeat radiography demonstrated almost complete re-expansion of both lungs. He improved over the next few days, chest tubes were removed after complete re-expansion was confirmed, and patient was discharged home. Two weeks later, chest radiograph showed no residual pneumothorax.

Discussion

Acupuncture, an ancient technique that originated in China, is based on the theory that disease is treated by allowing for normal flow of energy throughout the body using as many as 2000 acupuncture points. It is believed that these points are connected by different pathways that conduct energy between the body surface and internal organs. So far, there is evidence for possible efficacy in post-operative nausea and vomiting, postoperative dental pain, and chronic pain conditions. The possible mechanism of action has been attributed to a release of endogenous opioids augmented by the release of ACTH and cortisol as well as down-regulation of signaling through pain fibers. Typically, the depth of insertion of acupuncture needles varies from a few millimeters to a few centimeters with the tip in the muscle but it can also overlies structures like pleura and nerves and cause a wide variety of adverse events including bruises, needling site pain, infections due to inadequate sterilization techniques, broken needle fragments left behind, tension pneumothorax, hemothorax, cardiac tamponade, spinal cord trauma, subarachnoid bleeding, organ perforation and even death. Of note, this was the first time our patient received acupuncture at a site on the front of his chest. There is wide variability in the skill level among acupuncture practitioners. There is a need for more education and increased awareness of the complications that can arise from this seemingly safe procedure.

Puffs Of Smoke: A Common Presentation Of A Rare Disease

Clinical Vignette

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Introduction

Moyamoya disease (MMD) is a chronic occlusive cerebrovascular disease characterized by progressive stenosis affecting distal internal carotid arteries (ICAs) and their proximal branches with concomitant collateral circulation and an abnormal vascular network at the base of the brain. The diagnosis relies on characteristic angiographic findings resembling a 'puff of smoke'. There is striking ethnic difference in the prevalence of MMD: 3.2% vs. 0.0009%, in Asians vs. Europeans/African-Americans respectively. We describe a female with a long-standing history of headaches who was found to have diffuse steno-occlusive disease of the cerebral vasculature manifesting as intracranial hemorrhage.

Case Description

A 46-year-old white female with a history of hemiplegic migraines presented to the emergency room from home with complete expressive aphasia and right-sided hemiparesis preceded by a witnessed syncope and a tonic-clonic seizure. There were no associated constitutional or prodromal neurological symptoms. Her seizure was treated with lorazepam. The patient exhibited profound right facial droop, spastic unilateral weakness rated 1/5, intact sensorium and expressive aphasia. CT of the head demonstrated a left putamenal acute intraparenchymal hemorrhage with a 2 mm rightward midline shift. CT angiography of the head and neck revealed diffusely stenosed bilateral petrosal and intracranial ICA segments, poorly visualized proximal ACA and MCA vessels, and dilated lenticulostriate vessels without arteriovenous malformation or aneurysm. A hypercoagulability panel was negative. There was no evidence of vasculitis. Cerebral angiography revealed collateralized filling of distal bilateral MCA and ACA vasculature via lenticulostriate vessels consistent with MMD. Occlusive disease process was also identified in the posterior circulation. The patient was discharged to a rehabilitation facility on post-bleed day seven. There was mild residual expressive/receptive aphasia and dysarthria, but continued hemiparesis. Three months after her stroke, the patient underwent bilateral direct extracranial-intracranial cerebral bypass without complications.

Discussion

This case illustrates that MMD should be considered in young non-Asian individuals who present with ischemic or hemorrhagic stroke. Approximately 60% report migraine-like headaches and 30% describe tension headaches and therefore MMD should also be considered in patients with recurrent migraine-like headaches refractory to therapy. This rare condition is twice as frequent in females and follows bimodal age distribution with a second peak at 45–55 years of age. Studies suggest that patients with hypercoagulable conditions including autoimmune diseases and classical risk factors are more prone to developing strokes with MMD. Genetic studies indicate that MMD has a complex polygenic architecture. Based on limited world-wide experience headache frequency and intensity reportedly improves after revascularization. Timely neuroimaging to assess cerebral blood flow and perfusion reserve and provision of collateral pathways may have prevented this patient from recurrent strokes. Further studies are warranted to ascertain cost-effectiveness of primary stroke prevention with antiplatelet agents and elective surgery.

Thyroid Storm with Multiorgan Failure Treated with Plasmapheresis

Clinical Vignette

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Introduction

Thyroid storm is an endocrine emergency caused by a fulminant excess of thyroid hormones which has a mortality rate of 20-30%. Treatment consists of antithyroid drugs, radioiodine therapy and thyroidectomy. In severe, refractory cases or inadequate response to conventional treatment, therapeutic plasmapheresis could be considered. We present a case of severe thyroid storm with multiorgan failure and lactic acidosis.

Case Presentation

A 66-year-old female with history of uncontrolled Grave's disease secondary to medication non-compliance, atrial fibrillation, ebstein anomaly, hepatic failure presented with palpitations. In the emergency department, she was hypotensive, febrile, in rapid atrial fibrillation with severe leukocytosis, lactic acidosis, with TSH<0.005 UIU/mL, fT4 3.46 ng/dL, fT3 6.40 pg/mL, total T3 214.0 ng/dL, TSI >700. Physical exam revealed scleral icterus, jugular venous distention and diffuse anasarca. Patient was admitted to the ICU for severe thyroid storm and septic shock with renal failure, cardiomyopathy and shock liver. A month prior, she was admitted for acute hepatic failure. Further investigations revealed nodular hepatic contour, echocardiogram showed a normal ejection fraction, grade 3 diastolic dysfunction with severely dilated heart chambers. Labs showed elevated bilirubin, deranged liver function enzymes and elevated thyroid hormones. Methimazole and propylthiouracil were contraindicated due to liver dysfunction and patient refused radioactive iodine, lithium or surgical treatment at that time. This admission she was intubated and started on sepsis protocol, pressor support, broad spectrum antibiotics and an esmolol infusion. Continuous renal replacement therapy was started for acute renal failure. Once stable, plasmapheresis and intravenous immunoglobulins were administered. After the third session her thyroid function tests normalized.

Discussion

Thyroid storm presenting with multiorgan failure and lactic acidosis is rare. Our patient's course was complicated by right heart failure, atrial fibrillation, liver dysfunction, lactic acidosis and acute kidney failure. Lactic acidosis was due to increased metabolic demand, decreased cardiac output and hepatic clearance. Conventional treatments for thyroid storm include methimazole, propylthiouracil, lithium, radioactive iodine or thyroidectomy. Burch and Wartofsky score is used to make a diagnosis of thyroid storm (greater than 45 is indicative). Our patient scored 110 and methimazole and propylthiouracil were contraindicated due to liver dysfunction, hence plasmapheresis was considered. The American society of apheresis categorizes use of plasmapheresis in treatment of hyperthyroidism as category III (unestablished use category). This works by removing albumin bound T3 and T4, autoantibodies, 5'-monodesiodase (which converts T4 to T3) catecholamines, and cytokines. The replacement fluid binds and further lowers free hormones. In our patient plasmapheresis reduced T3 by 64% and T4 by 62%. It is important to recognize that although conventional treatments remain first line, plasmapheresis is safe particularly when traditional methods are unsuccessful or contraindicated.

Euglycemic Diabetic Ketoacidosis a Diagnostic Challenge

Clinical Vignette

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Introduction

Diabetic ketoacidosis is a common life threatening condition that has an excellent response to treatment when initiated in a timely manner. The diagnosis and treatment of diabetic ketoacidosis is therefore very important. Euglycemic diabetic ketoacidosis is a commonly misdiagnosed or missed condition. SGLT 2 inhibitors are an important 2nd or 3rd line treatment for uncontrolled diabetes but physician should be aware of their tendency to cause diabetic ketoacidosis especially euglycemic diabetic ketoacidosis.

Case Presentation

A 53-year-old male presented to the emergency department with nausea, vomiting and abdominal pain for four days. The patient had a past medical history of hypertension, hyperlipidemia and type II diabetes mellitus treated with empaglifozin and metformin. The patient had recently been changed from insulin to empaglifozin. Abdominal exam was nonfocal. Chemistry revealed bicarbonate: 22 mmol/L, anion gap: 2, lactate: 2.3 mmol/L and a blood glucose level of 250 mg/dl. The urinalysis showed glucose of > 1000 and ketones 15 mg/dl. The patient was vitally stable. The slight ketonuria and lactic acidosis was thought to be due dehydration/ starvation because of nausea and vomiting. The patient was given IV fluids antiemetics and discharged home. The patient presented two days later with more severe abdominal pain, nausea and vomiting. Abdominal exam was again non focal. Chemistry showed a pH of 7.27 bicarbonate level: 17.4 mmol/L, anion gap: 27 and a blood glucose level of 169 mg/dl. Urine studies showed a glucose level of >1000 and ketones 160 mg/ dl. Serum acetones were 1:8 positive. Serum osmolarity was normal and methanol and ethylene glycol levels were negative. A diagnosis of euglycemic diabetic ketoacidosis was made and patient was started on IV fluids and insulin therapy according to diabetic ketoacidosis protocol. The patient improved with this therapy and his anion gap closed and his acidosis resolved slowly in 48 hours.

Discussion

Diabetic ketoacidosis is a combination of hyperglycemia (>250 mg/dL), acidosis (arterial pH <7.3 and bicarbonate <15 mEq/L) and ketosis. Euglycemic diabetic ketoacidosis on the other hand is defined as metabolic acidosis (pH <7.3 and serum bicarbonate <18 mEq/L), ketosis and a blood glucose <200 mg/dL. SGLT 2 inhibitors are antidiabetic medications that are active through the kidney which is unique. In May 2015 FDA issued a warning for SGLT 2 inhibitors. These drugs were shown to cause diabetic ketoacidosis and most of the cases were in type II diabetics. The exact mechanism of action of diabetic ketoacidosis is unknown. One proposed mechanism is that these agents lower glucose level and impair insulin secretion thus contributing to insulin deficiency. This case illustrates the importance of awareness about this diagnosis.

Hydralazine Induced Acute Dystonia

Clinical Vignette

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Introduction

Neurological side effects from chronic use of hydralazine, resulting in pyridoxine deficiency driven peripheral neuropathy has been reported, however no other motor disturbances have been reported. Acute dystonia is a state of abnormal tone that results from involuntary contraction of muscles. This can present as overextension or flexion of limb, neck feet and fixed grimaces or can result in spasms of the whole musculature. There are also restricted forms of dystonia such as spasmodic torticollis which may occur in diseases like Wilson's disease and Parkinson's disease. The most common acute dystonic reaction is drug related usually due to antipsychotics like phenothiazine. In this case we report a case of acute dystonic reaction secondary to hydralazine.

Case Report

We report a case of a 60 year old female with recent diagnosis of hypertension on three blood pressure medications, who presented with hypertensive urgency, and complaints of arm numbness. Neurological exam failed to show focal signs and patient had good strength in all extremities. A CT of the head did not show any evidence of stroke. Patient was initially treated in the emergency department with an IV Nicardipine drip, and titrated off once blood pressure improved. The patient then was transferred to the medical floor, where she had recurrence in elevated blood pressure 190/95, which was managed with IV hydralazine 10 mg. A few minutes later the patient became flushed, diaphoretic, and developed severe stiffness with significant spasmodic voluntary contraction of right arm/forearm muscles. These were followed by torticollis and twisting of the neck toward the right side, and locking of the jaw which resulted in inability of the patient to open her mouth. The patient's blood pressure was measured at 220/110, Heart rate 114, and she was treated with IV Diphenhydramine 50mg, IV steroids and 0.1mg of Clonidine. Patient stiffness and dystonia improved quickly with multiple doses of IV Dyphenhydramine. A followup MRI/MRA failed to show any acute signs of cerebral ischemia. Laboratory exams did reveal hypokalemia of 2.6 g/dL and hypomagnesemia of 1.5 g/dL, however there was no seizure like activity, and hypokalemia was aggressively and appropriately repleted. The patient's symptoms developed minutes after Hydralazine was administered.

Discussion

Acute dystonic reactions and other extrapyramidal reactions have never been associated with hydralazine. These manifestations have been associated with anti-psychotics, but also have been reported to be associated with unusual drugs such as ranitidine. Treatment of these acute reactions involves giving diphenhydramine multiple times, but other options such as trihexyphenidyl and benztoprine can be administered. In some cases treatment can involve injection of botulinum toxin. In this case we report an unusual and never reported side effect of hydralazine and discuss the appropriate management.

ABSTRACT POSTER PRESENTATIONS STUDENTS

Detection of Non-cardiac Disorders During Routine Tc99 Sestamibi (Cardiolite) Stress Test

Clinical Vignette

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Cooper University Hospital Program (Brian P. Gable, MD)

Introduction

The use of Tc99 sestamibi (Cardiolite) stress test is an important cardiac diagnostic and management tool. However, the extra-cardiac uptake of radioactive tracer could serve in detection of non-cardiac etiologies.

Case

A 77-year-old female with diabetes mellitus type-2, hypertension, coronary artery disease, treated carcinoma of breast, hyperparathyroidism status post parathyroidectomy underwent a routine Cardiolite stress test. It showed minimal changes in her myocardium but an increased uptake of radiotracer focalized to her rib cage and sternum. Patient was asymptomatic. Her vitals were in the normal range and her physical examination was unremarkable. Abnormal bone uptake prompted further workup to identify potential underlying myeloproliferative disorder. The relevant abnormal diagnostic test results showed serum albumin of 3.5 g/dL, globulin 4.6 g/dL, albumin globulin ratio of 0.76, Serum Protein Electrophoresis (Protein Total: 10.7 g/dL, M spike: 4.3 g/dL); Free Kappa 2,030 mg/dL, Free Lambda 1.59 mg/dL, Kappa/Lambda Ratio 1,276.73; serum Calcium 8.8 mg/dL, EGFR greater than 60 mL/min/1.73 square meter, and hemoglobin 10.8 g/dL. Based on these abnormal findings of plasma cell dyscrasia skeletal X-rays and bone marrow biopsy were planned to establish a diagnosis of smoldering multiple myeloma vs. multiple myeloma requiring treatment, in order to decide her further management.

Discussion

Extra-cardiac skeletal uptake of Cardiolite is commonly seen in hyperparathyroidism. Although nuclear bone scan has not been established as a primary diagnostic tool to investigate skeletal involvement in multiple myeloma, increased bone uptake provides a potential direction to investigate for multiple myeloma and other myeloproliferative disorders. Many studies have reported a high accuracy of Cardiolite in detecting active multiple myeloma (Pace, et al, 2006; Balleari, et al 2002; Svaldi, et al, 2001, Alexandrakis, et al, 2001) with a positive predictive value of 100% and a negative predictive value of 83%. Catalano and associates (1999) found positive Cardiolite skeletal scintigraphy in 30% of patients who had no evidence of multiple myeloma on a radiologic full skeletal survey, 76% of whom were later diagnosed with multiple myeloma. Non-cardiac abnormal findings with Cardiolite, especially heightened skeletal uptake, can be an effective and probably superior diagnostic tool compared to the total body skeletal X-ray survey in defining the target organ involvement in multiple myeloma.

Conclusion

Abnormal skeletal uptake during Cardiolite stress test can provide valuable non-cardiac diagnoses. Cardiolite scan could serve in the detection of myeloproliferative disorders, and in screening for skeletal involvement in multiple myeloma.

Drug-Induced Aseptic Meningitis: A Rare Adverse Reaction to a Common Antibiotic

Clinical Vignette

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Introduction

Amoxicillin is a beta-lactam antibiotic commonly prescribed for the treatment of bacterial infections. Although generally well tolerated, amoxicillin can rarely be associated with complications requiring a thorough medical history and high index of suspicion.

Case

A 36-year-old healthy Asian female presented with persistent generalized headaches for two weeks. Two weeks prior to the onset of her headaches, she was diagnosed with acute streptococcal pharyngitis for which she received a 10-day course of amoxicillin. A day after initiation of amoxicillin therapy, she began to experience worsening headache, neck pain and nausea. Patient denied sick contacts, travel outside the country, intake of over-the-counter or other medications besides amoxicillin, and was afebrile with no alteration in mental status. Three years prior, patient had similar headache and neck pain following amoxicillin treatment for an upper respiratory tract infection for which she was diagnosed with suspected non-viral meningitis. Her symptoms resolved after she finished the course of amoxicillin. Patient's vital signs were within normal limits. Physical examination showed nuchal rigidity; the rest of the physical examination was unremarkable. Her complete metabolic panel and complete blood count were within normal limits. A head CAT scan was normal. Cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis, normal glucose levels, nonreactive VDRL, negative Gram stain and culture, negative AFB, and negative HSV. Her HIV test was negative. Patient was diagnosed with Drug-Induced Aseptic Meningitis (DIAM) secondary to amoxicillin. Patient was treated symptomatically and she recovered fully.

Discussion

Aseptic meningitis is most commonly caused by enteroviruses and refers to meningeal inflammation with negative bacterial cultures. Other causes of aseptic meningitis include infectious agents such as HSV or HIV, malignancies and drugs (DIAM) such as NSAIDs, trimethoprim-sulfamethoxazole, ciprofloxacin, cephalexin, metronidazole, amoxicillin, penicillin, isoniazid, allopurinol, etc. Amoxicillin-induced aseptic meningitis is a rare adverse drug reaction with only 13 reported cases in the medical literature. It presents like bacterial meningitis with fever, headache, nuchal rigidity and altered mental status, but it is self-limiting, only requiring symptomatic management. Meningeal immunologic hypersensitivity reaction to amoxicillin is believed to be the underlying pathophysiology. Diagnostic testing includes a thorough history and physical exam followed by CT neuroimaging in patients presenting with abnormal neurological exams. A CSF examination should be performed followed by CSF culture, AFB, HSV and viral PCR. CSF analysis usually demonstrates a lymphocytic pleocytosis with an elevated protein level and normal glucose. Diagnosis is made by establishing a temporal relationship between drug administration, onset of symptoms and resolution of symptoms following discontinuation of the drug.

Conclusion

Amoxicillin is a widely used antibiotic which can cause DIAM, a rare but serious adverse drug reaction that requires a thorough medication history and evaluation.

Implications of Hepatitis C Virus Screening Test in Baby Boomers

Clinical Vignette

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Introduction

The USPSTF recommends screening for hepatitis C virus (HCV) infection in adults born between 1945 and 1965. In some cases the results generate unnecessary worry.

Case

A 63-year-old Hispanic female with hypothyroidism, hyperlipidemia and emphysema underwent a well visit. Her medications included levothyroxine, atorvastatin and tiotropium. She had no history of jaundice, or known hepatitis virus infection. Her vital signs and physical examination were unremarkable. Her screening test for hepatitis C antibodies (anti-HCV) was positive. She became very concerned. Her test for HCV RNA was negative. She was reassured that she had a false-positive hepatitis C antibody test.

Discussion

According to NHANES, HCV is present in around 1% of all adults. HCV infects hepatocytes and lymphocytes. It replicates within the cells, spreads and slowly causes an acute or chronic hepatitis. In around 25% of patients the disease resolves spontaneously. Positive HCV antibody is found in around 2.1% of all adults. In immune assays, HCV antigen is immobilized onto solid phase, such as magnets, and are detected by a preformed conjugate antibody. HCV RNA test checks for viral replication in the bloodstream and is used in patients with a positive anti-HCV antibody. The anti-HCV antibody test has a low positive predictive value in populations with a low prevalence of HCV making it necessary to confirm results with the HCV RNA test. Studies have shown the possibility of a false-positive anti-HCV antibody from recent Left Ventricular Assist Device (LVAD) or a Fungal Peritonitis. There are 3 antibodies that are checked in the HCV antibody screen core, NS5, and NS3. The NS5 and NS3 antigens are recombinant antigens which react positively on the ELISA antibody test, however when reassessed with RIBA, it is shown that there is no correlation between NS5 positivity as the sole marker and active HCV infection. There are also instances in testing where someone who tests positive for anti-HCV antibody is cross reactive with another organism such as Dengue virus, HIV, or Plasmodium vivax as studied in Brazil. Activation of the immune system, such as a recently administered flu vaccine or other diseases can cause a false-positive anti-HCV antibody test. According to the archives of Clinical Microbiology, repetitive freezing and thawing of blood is a common cause of false positive rates as well as autoimmune disorders such as SLE or Rheumatoid arthritis. These tests however, are cheap to perform and are able to administer many true negatives in patients without HCV.

Conclusion

While very useful in areas with a high prevalence of HCV, in low prevalence areas up to 32% of anti-HCV positive results represent false-positive test. Prompt discussion of such possibility with the patient may help alleviate patient concerns.



New Jersey Chapter