2014 Minnesota Academy of Pediatrics Poster Competition Winners

Each year, the American Academy of Pediatrics encourages its state chapters to invite medical students and residents to take part in a scientific poster competition. Residents and students submitted posters for consideration at the Minnesota chapter’s annual meeting in Minneapolis in June. Both of the state’s pediatric training programs (the University of Minnesota’s and Mayo Clinic’s) were well-represented.

Posters were judged by practicing pediatricians, pediatricians from the state’s academic medical centers, and the students’ and residents’ peers. “Poster Rounds” were conducted for the peer-judging process. Criteria used by judges included clinical relevance, originality, and written and visual presentation. A “People’s Choice” award winner was also selected. Special thanks to Andrew Olson, M.D., from the University of Minnesota for coordinating the competition. Congratulations to all of the participants on their excellent work.

MEDICAL STUDENT CLINICAL VIGNETTE WINNER

Amantadine Toxicity in an Adolescent with End-Stage Renal Disease

BY MEGAN PETERSON AND CHRISTIAN HANNA, M.D., UNIVERSITY OF MINNESOTA

Amantadine is an antiviral drug used against influenza A and as a treatment for Parkinson’s disease. Recently, it has been used for treatment of psychotropic-induced weight gain in children and adolescents. The drug’s full mechanism of action is not entirely understood, but it is known to have dopaminergic, antimuscarinic and anti-NMDA-glutamatergic properties. Amantadine toxicity most often affects the cardiac and central nervous systems.

Presentation: Our patient is a 16-year-old female with bipolar disorder and subsequent end-stage renal disease (ESRD) secondary to lithium toxicity requiring chronic hemodialysis. Because of weight gain from the antipsychotics, she was prescribed amantadine. She presented with acute worsening of tremors, weakness, gait problems and vision loss, as well as acute hypoxia noted at dialysis the morning of admission. She had been admitted to the hospital with similar symptoms on two separate occasions during the previous month without a clear etiology of those symptoms. Her vision loss was attributed to corneal edema with an unknown cause. During her admission, she was intubated and required respiratory support for two weeks as well as continuous renal replacement therapy. She also had new onset arrhythmia and seizure episodes during her stay in the intensive care unit. Her urine drug screen from admission was positive for amantadine.

A diagnosis of amantadine toxicity was made after eliciting further history from the patient’s mother. The patient had been taking doses higher than recommended for ESRD patients for five months prior to presentation. Her symptoms gradually improved with supportive care and cessation of amantadine.

Conclusion: Our patient developed amantadine toxicity because she was prescribed much higher doses of the drug than recommended for patients with ESRD. Amantadine accumulates in patients with abnormal kidney function, and the amount of the drug removed by hemodialysis is small.

Reports have described effects of amantadine toxicity on the cardiac and central nervous systems. One report described an acute respiratory distress syndrome (ARDS)-type presentation in an adult with ESRD. Since we did not find any specific etiology for our patient’s respiratory status, it is very likely that our report describes the first case of ARDS secondary to amantadine toxicity in children. It also may be the first case of amantadine toxicity in children with ESRD.

This case emphasizes the importance of drug dose adjustment in patients with renal impairment, in addition to good communication between the physicians and other health care providers involved in a patient’s care. MM
MEDICAL STUDENT RESEARCH/QUALITY IMPROVEMENT WINNER

Reduction of Pediatric Head CT Rates in a Community ED: A Preliminary Report

BY THUY DUONG NGUYEN-TRAN AND JEFF LOUIE, M.D., UNIVERSITY OF MINNESOTA

Head injury is a common reason for emergency department (ED) visits, and a head CT (HCT) is frequently used to evaluate for traumatic brain injury. Unfortunately, there is no national benchmark data for pediatric HCT rates; studies have estimated rates to be between 5% and 70%. A 2012 study by Menoch et al. found the HCT rate was 29% for head injuries at two tertiary pediatric emergency departments. However, the rate of children who receive a HCT and require neurosurgical intervention is less than 1%. Thus, it raises concern that some children with head injury are exposed to unnecessary radiation, which could increase their risk for malignancy.

Objective: Our study’s purpose was to determine the HCT rate in a pediatric population at a community ED and identify factors that could help reduce HCT rates while still maintaining provider comfort and patient safety. In 2009, a prospective study by Kuppermann et al. validated prediction rules that identified children at low risk of clinically important traumatic brain injuries (cTBI) and would obviate the need for a HCT. These prediction rules included severity of injury and certain signs and symptoms such as loss of consciousness, palpable skull fracture and impaired mental status. These prediction rules were developed into a guideline for evaluating head injuries and discussed with providers at a community ED that also serves as a Level III trauma center.

Methods: Our retrospective study looked at patients from birth to 18 years of age who were evaluated in the ED in 2012 for a head injury or concussion or who had a HCT, focusing on HCT rates before and after provider education and application of Kuppermann’s guidelines.

Results: The ED had a total volume of 28,072 visits, 5,727 of which involved patients younger than 18 years of age. Head injury or concussion comprised 5% of the pediatric visits. The overall HCT rate for pediatric patients was 42%. The guidelines were discussed with providers in July 2012, and there was an initial decrease in the HCT rate for three months (to an overall low of 25%), after which rates began to rise. Additionally, variables commonly not documented were GCS (66%), vomiting (20%) and severe headache (28%).

Conclusion: These data gave us a baseline for HCT rates and identified key factors that require improved documentation. Our next goal is to implement PDSA (Plan-Study-Do-Act) cycles after each educational intervention. Our educational bundles will include interventions to improve awareness and use of the guidelines such as standardized EMR templates for head injuries and pocket handouts. We hope to standardize the evaluation of pediatric head injuries and decrease the use of HCT while optimizing patient safety and provider comfort.

PEDIATRIC RESIDENT RESEARCH WINNER

Improving the Consistency of Screening for Syphilis and Human Immunodeficiency Virus in Expectant Mothers

BY LINDSEY YOCK, M.D., KRISTI BOLDT, M.D., WILLIAM CAREY, M.D., CHRISTOPHER COLBY, M.D., AND THOMAS BOYCE, M.D., MAYO CLINIC

Objective: We sought to standardize our approach to prenatal screening so that the maternal syphilis (VDRL) and human immunodeficiency virus (HIV) status of every expectant mother would be determined and documented during the second trimester, with postpartum testing of the mother or newborn if the initial screening were declined.

Methods: We convened a workgroup with representatives from the obstetrics, pediatrics, neonatal medicine, infectious diseases and legal departments. Members adhered to the DMAIC (define, measure, analyze, improve and control) framework of quality improvement. We measured our baseline performance and found that VDRL and HIV status were absent from 20% and 9% of maternal records, respectively, among 100 consecutive newborns admitted to our NICU. Using causal tree analysis, we identified critical barriers to screening and documentation of serologies. We then developed standardized process improvements that targeted these deficiencies. In the control phase, we re-measured our performance to determine whether these interventions were successful.

Results: Six months after implementation of the interventions, VDRL and HIV status were absent from 9% and 0% of maternal records, respectively.

Conclusion: A formal QI approach enabled our institution to improve the rates of maternal VDRL and HIV screening and documentation.
Mycoplasma has several diverse presentations including atypical pneumonia, hemolytic anemia and bullous myringitis. Mycoplasma is also one of the leading causes of erythema multiforme major. There are several case reports correlating mycoplasma infection and incomplete Kawasaki disease. This case report further supports that link. Other case reports describe annular skin lesions as the initial presenting feature of incomplete Kawasaki disease, further expanding the index of suspicion needed when evaluating a child with rash and fever.

Case: A 9-year-old previously healthy, immunized boy presented with seven days of fevers, purulent conjunctivitis, pharyngitis, cough, mucositis and rash. His only drug exposures were to ibuprofen and over-the-counter cold medicine. On admission, he was afebrile and his vital signs were normal. He had purulent bilateral conjunctivitis and extensive mucositis involving his mouth and urethra; bilateral, tender anterior cervical lymphadenopathy; and numerous targetoid plaques with central bullae and peripheral pallor ranging in size from 1 to 3 cm covering his body and extremities. He was also noted to have painful dactylitis of his fingers.

On admission, his labs were remarkable for an elevated erythrocyte sedimentation rate of 128 mm/h and an elevated C-reactive protein of 78.8 mg/L. His white blood cell count, hemoglobin, platelet count, alanine aminotransferase and albumin were normal. He did have a sterile pyuria (5 white blood cells per high power field) attributed to his mucositis. His mycoplasma IgG was negative and his mycoplasma sputum PCR was positive.

His exam findings were consistent with erythema multiforme major caused by active mycoplasma infection. Despite treatment with azithromycin and supportive care, he was persistently febrile on hospital day four, so a transthoracic echocardiogram was obtained that showed coronary artery ectasia and left anterior descending artery dilatation to 5.8 mm (Z-score 2.5) with no frank aneurysm formation.

A diagnosis of incomplete Kawasaki disease was made and high-dose aspirin and IV immunoglobulin were administered. At follow-up, his skin lesions had improved and his coronary artery dilatation had stabilized.

Discussion: This case illustrates the elusive nature of Kawasaki disease. Once the etiology is better understood, there will undoubtedly be a decline in late or missed diagnoses. In the meantime, pediatricians must have a high index of suspicion for this diagnosis even if there is another explanation for erythema multiforme, given the growing association between mycoplasma infection and incomplete Kawasaki disease. MM
Glut-1 deficiency syndrome occurs as a result of impaired glucose transport across the blood-brain barrier. Mutations in the Glut-1 gene are responsible for a spectrum of phenotypes thought to be dependent on the level of glucose transporter function preserved in vascular endothelial cells. Characteristic symptoms include infantile seizures, microcephaly, ataxia and developmental delay. A ketogenic diet is an effective treatment in most patients, as ketones bypass the transporter deficiency and diffuse across the blood-brain barrier to fuel the brain's metabolism.

**Case:** A 34-month-old female with a history of recurrent complex febrile seizures and developmental delay presented with worsening seizure activity. At 14 months of age, the patient was diagnosed with febrile seizures. At the time, work-up included multiple normal electroencephalograms (EEG) and brain magnetic resonance imaging (MRI). Over the next year, she experienced four seizure events with peri-ictal ataxia despite treatment with anti-epileptics including levetiracetam and oxcarbazepine. As the seizures subsequently increased in frequency to three times per week, the patient's mother distinguished a prodrome in which the patient appeared “drunk” with an abnormal gait that was indicative of an impending seizure.

The patient was admitted for work-up of increased frequency and severity of seizures. EEG, MRI and numerous serum assays including serum glucose were unremarkable. Because of concern for a metabolic disease, a lumbar puncture was performed. Cerebral spinal fluid (CSF) glucose was 34 ng/dL and serum glucose was 97 ng/dL with a ratio of 0.36, well below the normal ratio of 0.67.

The patient initially required intravenous dextrose to prevent neuroglycopenia and associated symptoms of restlessness, ataxia and altered levels of consciousness. In order to begin a ketogenic diet, the patient needed to be weaned from the dextrose. Because of the recurrence of symptoms and parental concern, weaning from IV dextrose required a stepwise approach over three days.

The patient was then started on a ketogenic diet with gradual increases in the fat-to-protein plus carbohydrate ratio. A therapeutic ketogenic diet resulted in resolution of seizure activity with transient side effects of nausea and vomiting. Genetic testing for SLC2A1 mutations were pending at the time of submission.

**Discussion:** The hallmark finding of Glut-1 deficiency syndrome is hypoglycorrhachia, with the mean CSF glucose: plasma glucose ratio of 0.37. Approximately 500 cases have been reported since the syndrome was first described in 1991. However, it may be underdiagnosed given its rarity and complex clinical and genetic features. In this case report, it appears that the correct diagnosis was masked by febrile illnesses, which induced anorexia and hypoglycemia, exacerbating underlying hypoglycorrhachia and triggered seizure activity. Following establishment of a ketogenic diet with avoidance of fasting, the patient's seizure activity and gait disturbances should continue to improve.