The Anatomy of a Great Poster

SGIM Conference 5/5/2011

Richard Gerkin, M.D.
Bridget Stiegler, D.O.
Christina Bergin, M.D.
What to Expect

• Purpose
• Elements
• Arrangement
  – Research Poster
  – Case Presentation Poster
• Construction
• Examples
• Presentation
• Judging/Evaluation
• Why would you take the time to enter a research or case presentation poster?

• Why wouldn’t you?

• What makes a poster great?
  – Upon first impression?
  – After reading?

• What makes a poster irritating?

• What elements are important when judging a poster?
Purpose

• To communicate medical/scientific research

• To illustrate key points in a visually stimulating and aesthetically pleasing manner

• To represent yourself and your work to peers and colleagues

• To participate meaningfully in a scientific meeting or conference
Elements

• Easy for evaluators to read, clean and uncluttered

• Well planned and meets the guidelines set forth by ACP/SGIM/governing board

• Attracts viewer’s attention, grabs a second look

• Concisely communicates results of investigation
Arrangement

**Timing**
- The viewer is able to *glean the message* in 3-5 minutes
- The viewer is able to *read the text* in 10 minutes

**Organization**
- The poster is organized in sections similar to a scientific article or oral presentation
- The poster describes and represents findings of either a scientific research project or a clinical case
Construction

• Use a template/software program
  – Internet search “poster template”
  – Online example of poster construction

• The rough draft process
  – 1st draft at least one month prior to conference
  – Multiple considerations
    • Font: ≥ 72 point title, ≥ 20 point text
    • Word count, prose style, grammar, fluidity, figure clarity, spelling
  – Print rough draft on letter sized paper to assess layout challenges
Research Poster

• Title
• Introduction
• Materials and Methods
• Results
• Discussion
• Conclusion
• References
• Acknowledgements
• **Title**: 2 lines or less  
  – ≥ 72 pt. type, legible at 25 feet  
  – Clear, concise, direct

• **Intro**: 200 words or less  
  – Define the issue  
  – Establish the purpose of your work  
  – Justify your experimental approach  
  – Provide a clear hypothesis

• **Materials and Methods**: approximately 200 words  
  – Use figures and tables to illustrate experimental design  
  – Use flowcharts to summarize timing of events  
  – Include photograph or labeled drawing  
  – Outline statistical plan
Research Poster, continued

**Results**: approximately 200 words
- Provide qualitative/descriptive results
- Present analyses that specifically address the hypothesis
- Refer to charts or images

**Discussion**: approximately 300 words
- Remind the viewer of the hypothesis
- Discuss if/why results were conclusive
- Point out relevance of findings to other published work
- Discuss limitations of the work
- Highlight future directions of the research
Research Poster, continued

**Conclusion**; approximately two sentences
- Concise summary
- Reminds viewer of relevance

**References**
- Approximately 5-10 citations
- Standard format

**Acknowledgement**
- Assistance and financial support
Case Presentation Poster

- Title
- Introduction
- **History of Present Illness**
- **Hospital Course**
- **Family History**
- **Social History**
- **Labs, Images, Studies**
- Discussion
- Conclusion
- References
Case Presentation Poster

• Introduction – briefly introduce the type of condition/disease process – pathogenesis, etiology, microbiology, epidemiology if relevant

• HPI – as in a classic academic history and physical. Age of patient, important past medical history, presenting complaint, events leading to presentation

• Hospital Course - pertinent positive and negative findings on physical exam, work up and treatment plan, involvement of consultants, clinical progress
Case Presentation Poster

• Family History

• Social History

• Pertinent Labs/Images
  – Visual additions attract and inform viewers more effectively than text
  – Details on graphs and photos should easily be viewed from six feet away
  – If you include a photo, add a thin gray or black border
  – If possible use digital, high quality photographs as many web images have poor printing resolution
Example Posters
It is estimated that 6-10% of school-aged children suffer from Attention Deficit Hyperactivity Disorder (ADHD). Treatments for ADHD include the use of stimulants, (methylphenidate, etc.) and psychotherapy(1). A significant number of patients treated with psychostimulants exhibit an inadequate response or cannot tolerate their use (2). The potential for abuse with the use of stimulants is a concern and is a factor in the quest for non-stimulants to treat the condition.

Atomoxetine (Strattera™), known chemically as benzenepropanamine N-methyl-alpha-(2-methoxyphenoxy), hydrochloride, was approved for the treatment of ADHD in November 2002. Marketed as a non-stimulant alternative in the treatment of ADHD, atomoxetine is a specific norepinephrine reuptake inhibitor. Atomoxetine has little affinity for other noradrenergic receptors or for other neurotransmitter transporters or receptors (3).

In therapeutic doses, atomoxetine is rapidly absorbed from the gastrointestinal tract, with peak plasma level occurring in 1-2hours after ingestion. Atomoxetine is metabolized by aromatic ring hydroxylation, benzylic oxidation, N-Demethylation and by hepatic microsomal enzyme P4502D6. As a result of P4502D6 metabolism, pharmacokinetics is influenced by polymorphic expression (3).

To our knowledge, there are no other reports of Atomoxetine overdose in the literature. The purpose of this study was to describe the effects of isolated Atomoxetine overdose.

Methods

We conducted a retrospective chart review of all human exposure calls (~150,000 charts) for atomoxetine ingestions reported to our Poison Control Center (PCC) during the years immediately following the release of the drug. Of the ~150,000, 17 accounted for isolated atomoxetine ingestions.

Inclusion criteria were defined as any oral atomoxetine ingestion in human beings, irrespective of age, referred to our PCC. Chart review was conducted using Crystal Reports™ in Visual Dotlab™. The only exclusion criterion was the inability to meet the inclusion criteria.

Following a brief training of systematic chart review, reviewer’s blinded purpose of this study completed a standardized data collection sheet. A third reviewer, acting as a tie-breaker was used in cases of conflict between the two reviewers. Age, outcomes and signs and symptoms were recorded. Patients were followed for up to 24 hours or until cessation of signs and symptoms. The study received expedited review from the institutional board review for all patient identifiers were recorded. Patients were followed for up to 24 hours or until cessation of signs and symptoms. The study received expedited review from the institutional board review for all patient identifiers were removed and here were no interventions.

Results

Symptoms were reported in 9 of 17 patients. Symptoms included gastrointestinal upset, hyperactivity, drowsiness, throat irritation, dizziness, tremor, tachycardia and tremor. Ages ranged from 9 months to 28 years (mean of 15.6 years). Doses of Atomoxetine were 10 – 1,2000mg.

Symptoms were delayed as long as three hours in patients. All neurological symptoms were preceded by tachycardia. Tachycardia was reported in 58%, emesis was reported in 34% and 17% had agitation and received benzodiazepines. All symptoms resolved within 30 hours.

Discussion

Little is known about the effects of isolated Atomoxetine overdose. Sawant and Daviss report a case of a 15-year-old male with a 1,200 mg Atomoxetine ingestion. His hospital course was complicated by two grand mal seizures and QTc prolongation.

Atomoxetine, as the sole agent was presumed, from a pill count. However, the patient had access to Bupropion, Risperidone and Alprazolam as well.(4) To our knowledge there are no additional reports of Atomoxetine reported in the literature.

In our study about isolated Atomoxetine ingestions developed symptoms. Symptoms may have been delayed for up to three hours. All symptoms resolved in 30 hours. Some of the limitations of our study include the retrospective nature, small sample size, lack of conformation of ingestion and the patient’s self-reporting of ingestions.

We conclude that isolated Atomoxetine ingestions commonly result in toxicity and can be delayed for up to 3 hours post ingestion.

References

Gamma-aminobutyric acid (GABA) deficiency as a cause for refractory status epilepticus in alcohol withdrawal

Introduction: Status epilepticus is a neurological disorder resulting in high morbidity and mortality. While most seizures have historically been treated with Gamma-aminobutyric acid (GABA) agonists drugs such as benzodiazepines and barbiturates, there are cases where GABAergic drugs are not effective. These involve INH related pyridoxine (vitamin B6) deficiency leading to intractable seizures or pyridoxine dependent seizures seen in the perinatal period where GABA levels or binding affinity of glutamate decarboxylase (GAD) are low. Most of the data on seizures related to pyridoxine dependent seizures is documented in neonates but the literature of such a phenomenon is sparse in the adult population. We present a case of refractory status epilepticus in an alcoholic man terminated by high dose pyridoxine after failure of high dose infusions of multiple GABA agonists.

Case:

HPI: A 53 YO heavy ethanol user with remote history of seizures of unknown etiology not previously treated with antiepileptics. He was admitted initially with ataxia and diplopia. He denied fever, chills, sweats, headache, neck pain, nausea, vomiting, dyspnea, tremor, slurred speech, or one-sided weakness. His last drink was more than 48 hours previous and he denied any withdrawal symptoms. His last drink was more than 48 hours previous and he denied any withdrawal symptoms.

Hospital course: 24 hours after admission, he had eye flickering and right facial twitching, which progressed to a generalized tonic clonic seizure. He was intubated for airway protection, placed on a 24 hour EEG monitor, and given multiple medications:

- Lorazepam (24 mg IV)
- Fosphenytoin (2 grams IV)
- Midazolam infusion
- Pentobarbital drip to induce coma
- Levetiracetam
- Valproic acid infusion
- Pyridoxine 5 grams IV bolus.

The seizures were refractory to all of these medications. GABA and pyridoxine deficiency were suspected and the patient was given Pyridoxine 5 grams IV bolus. This resulted in termination of clinical seizures and epileptiform activity on EEG. The patient remained seizure free for 72 hours but unfortunately developed multiorgan failure and the family chose to withdraw care.

Discussion: The prevalence of specific vitamin deficiencies including B6 has been reported in up to 50% of chronic alcoholics and should be considered in alcoholics with refractory seizures. This may be directly related to the amount of alcohol consumptim rather than malnutrition alone. The bioactive form of Vitamin B6, pyridoxal-5-phosphate (PLP) is a cofactor for the synthesis of GABA, the inhibitory neurotransmitter implicated in epilepsy and seizures. It exists as gamma-aminobutyrate in body fluids and is synthesized from the excitatory neurotransmitter glutamate via the enzymes GABA transaminase and L-glutamic acid decarboxylase (GAD) using PLP as a cofactor.

The mechanism of seizures during a low pyridoxine and GABA state may include a shift in the equilibrium between glutamate and GABA, downregulation of GABA_A receptors, reductions of GABA-mediated inhibition, activity of GAD, binding affinity to GABA_A and benzodiazepine sites, and GABA in CSF. The use of pyridoxine in theory should increase GABA synthesis and activity of GAD, increasing the binding affinity of GABA for its receptors, rendering GABAergic drugs more effective. However, some studies have shown contradictory results in CSF glutamate and GABA and seizures may actually involve raised piopecolic acid levels.

Conclusion: Regardless of the mechanism, as health professionals we should be aware that nutritional deficiencies like B6 should be considered as an etiology for refractory seizures, especially in chronic alcoholics and those taking bioniazid. We should have a high index of suspicion for B6 deficiency and have pyridoxine available if the traditional GABA agonists are ineffective in the termination of status epilepticus.

References:
Meniscal Allograft Shrinkage

**Background:** The meniscus carries out several functions in the knee to maintain its normal biomechanics. The meniscal deficient knee typically progresses to become painful and develop early arthritic changes compared to knees with intact menisci. Meniscal allograft transplantation (MAT) has been shown to be a viable treatment option for select patients with symptomatic meniscal deficiency. The ability of the meniscal allograft to restore the functions of the native meniscus depends on the allograft being appropriately sized. Inappropriately sized meniscal grafts may lead to an incongruent joint and abnormal contact forces. Shrinkage of the meniscal allograft can lead to it being substantially smaller than the native meniscus. Too small of a graft may pull on the repair site leading to increased rates of retears. Several papers have described shrinkage of the meniscal allograft after transplantation, but the degree and rate of shrinkage has not been quantified. The questions we looked to answer with this study were what is the frequency of meniscal allograft shrinkage and how much shrinkage typically occurs following MAT? We hypothesized that a measurable amount of shrinkage occurs in all meniscal allografts after transplantation.

**Methods:** A prospective review of 25 consecutive patients with meniscal deficient knees undergoing MAT was performed. Seventeen of the patients were male and eight were female with an average age of 30.1 years (range 15-45 years). MAT was performed as an isolated procedure in all patients. The medial meniscus was replaced in 15 patients utilizing the double bone plug technique for fixation of the horns, and the 10 lateral menisci had their horns secured with a bone bridge. The grafts were fixed peripherally to the capsule using an inside out method. All grafts used in the study were cryopreserved and sized using AP and lateral plain radiographs. The senior author (TC) performed all of the procedures. All 25 meniscal allograft recipients had magnetic resonance imaging (MRI) scans of the operative knee at one month (range 25-31 days) and a minimum 6 months (range 6-8 months) after the procedure. Meniscal volume was calculated using a laser-based non-contacting 3-D coordinate digitizing system (3-DCDS) to acquire the 3-D geometry of the meniscus. Meniscal volumes were compared between the 1 month MRI and the 6 month MRI to determine the amount of volume change over the 5 month period. Student t-test was used to determine a statistical significance between the time periods.

**Results:** Twenty-one of the 25 transplanted meniscal allografts showed a measurable decrease in volume of at least 2%. The average volume shrinkage from 1 month to 6 months was 7% with a range of 0 to 22%. One meniscus had greater than 15% shrinkage during the study; however, 8 of the 25 menisci showed 10% or greater shrinkage over the study period. No statistical difference was identified comparing the joint side, gender or age of the patients, or donor age or sex. Although not statistically significant, grafts not in anatomical position tended to have increased shrinkage. In addition, suturing the grafts to the peripheral capsule in a posterior to anterior manner led to less shrinkage, but this finding was not statistically significant.

**Discussion:** The aim of our study was to quantify the amount and frequency of meniscal allograft shrinkage. We found that at least 2% of shrinkage occurred in 84% of the grafts we studied with an average shrinkage of 7%. A concern is the fact that 32% of the grafts had 10% or greater shrinkage. Ten percent of shrinkage may be the limit that can be tolerated before contact forces are too high. The cause of shrinkage is still unknown, and further studies will need to be done to determine the exact cause. We did find that anatomical placement of the meniscal allograft tended to show less shrinkage. We also found a trend towards less shrinkage when the meniscal allograft was sutured to the meniscocapsular remnant in a posterior to anterior direction.
**Colonoscopy Bowel Prep: 1 day vs. 2 day in Patients at Risk for Slow Bowel Transit**

J. Reggie Thomas, DO; Richard Gerkin, MD; Bilal N Khan, MD; Sherri Thomas, DO; Nooman Gilani, MD, FACG

Department of Gastroenterology, Carl T. Hayden VAMC, Phoenix, AZ, USA

**Introduction:**
- About 1/3 of incomplete colonoscopies are the result of poor bowel preparation
- There is a paucity of data regarding whether or not patients with DM, constipation, or those on narcotics benefit from a more extensive bowel regimen
- The aim of this study is to evaluate the efficacy of two different strategies of polyethylene glycol solution administration for bowel preparation prior to colonoscopy

**Methods:**
- A retrospective review of all colonoscopies done between March 1 and June 30, 2009
- One day regimen = one day of clear liquid diet + 4L PEG solution + 20mg bisacodyl 1 day prior to the procedure
- Two day regimen = two days of clear liquid diet + 2L PEG solution two days prior and 4L PEG solution + 20mg bisacodyl one day prior to the procedure
- Demographic data collected: age, BMI, narcotic use, constipation, DM, HgbA1C, prep quality, cecal and total procedure time, test completeness, procedure indications and polyps detected
- Chi square testing and logistic regression were used for categorical variables
- T-tests and linear regression were used for continuous variable

**Results:**
- 300 Colonoscopy procedures reviewed (96% men)

**Conclusion:**
- Patients with a higher BMI were found to have inadequate bowel cleansing and more incomplete exams
- More incomplete colonoscopies were seen in the patient group using two-day bowel prep regimen
- Use of a two-day bowel regimen did not result in more adequate bowel cleansing in those at risk for slow bowel transit
Judging and Evaluation

• Who will be judging your poster
  – Attending physicians, unfamiliar with author

• What kind of questions you will be asked
  – Preparation is key, anticipate questions
  – Have friends and colleagues tell you what their questions would be
  – Any style of question is possible

• Breakdown of points system

• Example judging sheet
## Rating Scale

<table>
<thead>
<tr>
<th>Value</th>
<th>Anchor</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Outstanding</td>
<td>Virtually flawless, with negligible weaknesses</td>
</tr>
<tr>
<td>2</td>
<td>Excellent</td>
<td>Very strong, but with some minor weaknesses</td>
</tr>
<tr>
<td>3</td>
<td>Very Good</td>
<td>Strong, but with some moderate weaknesses</td>
</tr>
<tr>
<td>4</td>
<td>Good</td>
<td>Weak, but with some moderate strengths</td>
</tr>
<tr>
<td>5</td>
<td>Acceptable</td>
<td>Very weak, with some minor strengths</td>
</tr>
<tr>
<td>6</td>
<td>Unacceptable</td>
<td>Inadequate</td>
</tr>
</tbody>
</table>
## Content Areas: Research Poster

<table>
<thead>
<tr>
<th>Area</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presentation</td>
<td>Was the content logically arranged, easy to read, clearly written, and visually effective?</td>
</tr>
<tr>
<td>Significance</td>
<td>Does the study help to understand and/or improve human health?</td>
</tr>
<tr>
<td>Rationale</td>
<td>Were the reasons for the study clear? Was the study justified?</td>
</tr>
<tr>
<td>Research Design</td>
<td>Was the study designed to clearly and directly answer the research question?</td>
</tr>
<tr>
<td>Analysis/Interpretation</td>
<td>Was the analysis proper and did the conclusions flow from the research question and data?</td>
</tr>
<tr>
<td>Originality</td>
<td>Was the approach to the research question unique and original?</td>
</tr>
</tbody>
</table>
# Content Areas: Case Report

<table>
<thead>
<tr>
<th>Area</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presentation</td>
<td>Was the content logically arranged, easy to read, clearly written, and visually effective?</td>
</tr>
<tr>
<td>Significance</td>
<td>Does the report help to understand and/or improve human health?</td>
</tr>
<tr>
<td>Discussion</td>
<td>Did the poster effectively “mine” all available information from the case?</td>
</tr>
<tr>
<td>Originality</td>
<td>Were the choice and approach to the case unique and original?</td>
</tr>
</tbody>
</table>
Practice Judging

• Independent Scoring
• Group Discussion
• Group Scoring
The Presentation

• Know your poster inside and out, be able to present a synopsis without reading from the poster (two minutes or less)

• Maintain eye contact with the judges during your evaluation

• Brainstorm potential questions ahead of time

• Practice presenting to multiple people, request that they ask you questions

• Be prepared to discuss any tests or laboratory findings that are inconsistent

• Know the limitations of your study type

• Be well versed on both sides of concepts that may be controversial

• Know your references; do not list a reference that you have not read
Thank you for your attention and participation