

Case Report

Reproducible transient asystolic arrest during intramedullary reaming of the femoral canal: A case report

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ABSTRACT

The utilization of intramedullary devices in the surgical fixation of hip fractures is increasing. Although intramedullary devices offer many advantages in the treatment of these injuries, they are also associated with a unique set of potential complications, particularly during preparation of the femoral canal. Cardiac dysrhythmia resulting from reaming the femoral canal is rare and has not been previously described in detail in the literature. We present the case of a 69-year-old male with an infected right cephalomedullary femoral nail who underwent removal of hardware and experienced reproducible, transient asystolic cardiac arrest during reaming of the femoral canal and offer potential explanations for this event.

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1. Background

Hip fractures are one of the most common orthopaedic injuries sustained by geriatric patients worldwide.¹ While various methods of surgical fixation can be employed in the treatment of these injuries, the utilization of reamed intramedullary devices has significantly increased over the past few decades.² This has been attributed to the minimally invasive nature of the procedure, the biomechanical advantages offered by intramedullary devices, and the benefits provided in early post-operative rehabilitation.³ Although reamed intramedullary devices offer many advantages in the treatment of hip fractures, they are also associated with a unique set of potential complications. Most of these complications are technical in nature and can be easily avoided by using appropriate surgical technique.³ Rarely, serious systemic complications from intramedullary fixation can occur, particularly during preparation of the femoral canal, including fat embolization, acute respiratory distress syndrome, and even cardiac dysrhythmia.^{4,5}

2. Case presentation

A 69-year-old male with a history of remote myocardial infarction status post percutaneous coronary intervention and chronic atrial fibrillation managed with rivaroxaban underwent treatment of a right intertrochanteric hip fracture with a Depuy-Synthes Titanium Trochanteric Fixation Nail System (Warsaw, IN, USA) at an outside hospital. The patient's initial post-operative course was uneventful, but he presented to our clinic approximately 2 months after surgery with signs and symptoms concerning for periprosthetic infection.

On examination of the right hip, there was a 1 × 2 mm draining sinus tract at the distal third of the most proximal incision with associated erythema and swelling. Radiographs of the right femur demonstrated a healed intertrochanteric hip fracture with a well-positioned cephalomedullary nail without evidence of failure (Fig. 1). Significant degenerative changes of the hip joint were also appreciated on these films. CT scan of the right hip confirmed osseous union of the fracture without evidence of hardware failure or any significant fluid collection. Inflammatory labs were significant for a WBC 9.8 K/cmm, ESR 30 mm/hr, and CRP 10.9 mg/dL. Consequently, recommendations were to proceed with incision and debridement of the right hip, removal of hardware, and placement of antibiotic spacer in preparation for a staged total hip arthroplasty. Given the patient's cardiac history, referral to cardiology for pre-operative risk stratification and medical optimization was scheduled.

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Fig. 1. Pre-operative anteroposterior and lateral radiographs of the right femur demonstrating a well positioned cephalomedullary nail without evidence of failure, evidence of callus formation at prior fracture site, and significant post-traumatic degenerative changes of the right hip.

Transthoracic echocardiogram was performed in the cardiology clinic and demonstrated an ejection fraction of 50% with trace tricuspid valve regurgitation and patient was diagnosed with New York Heart Association (NYHA) Class II congestive heart failure. Revised Cardiac Risk Index (RCRI) score was calculated to be 2, conferring a 6.6% perioperative cardiovascular risk and no additional cardiac testing or intervention was recommended based on the American College of Cardiology and American Heart Association guidelines. In regards to the patient's anticoagulation regimen for chronic atrial fibrillation, patient was instructed to hold two doses of rivaroxaban prior to surgery with

recommendation to restart the medication on post-operative day 2 at the surgeon's discretion.

The patient was taken back to the operative theatre in February 2017 with an American Society of Anesthesiologists (ASA) score of 3E. Prior to induction of anesthesia, the patient was noted to be in atrial fibrillation without rapid ventricular response. An arterial line was placed for intra-operative hemodynamic monitoring and general endotracheal anesthesia was induced without complication utilizing propofol, fentanyl, lidocaine, and succinylcholine. After routine preparation of the extremity, a standard posterior approach to the right hip that incorporated the previous surgical incision was performed from the lateral decubitus position on a pegboard. Systematic debridement of infected tissue and removal of the cephalomedullary screw and femoral nail were uncomplicated. The hip was dislocated and femoral head was excised in standard fashion. A box osteotome and femoral canal finder were used to enter the intramedullary canal of the femur and an 8 mm starting reamer was placed into the femoral canal over a ball-tipped guidewire. As soon as the reamer was powered, the patient entered an apparent asystolic cardiac arrest. Consequently, the reamer was removed and the patient's heart rhythm immediately returned to baseline atrial fibrillation without any intervention from the anesthesia team (Fig. 2). Given the transient nature of the abnormal rhythm and the patient's immediate conversion back to baseline atrial fibrillation after the reamer was removed, the asystolic arrest reading was thought to be artifact secondary to interference. Consequently, we proceeded to ream the femoral canal once again while carefully monitoring the patient's hemodynamic status. The patient entered asystolic arrest immediately after the reamer was powered for the second time. Furthermore, the patient's pulse simultaneously ceased on the arterial line tracing demonstrating that the event was genuine. Similar to the first event, the patient's rhythm immediately returned to baseline atrial fibrillation and the pulse tracing on the arterial line returned on removal of the reamer.

Given the patient's cardiac history and discussion with anesthesia team, we collectively decided to abort the procedure at this time. Conversation was had with the patient's family member and the decision was made to definitively treat the patient's periprosthetic infection with a girdlestone amputation (Fig. 3). The wound was closed in standard fashion, the patient was extubated, and transferred to the surgical intensive care unit in stable condition. Total operative time was one hour and 29 min. Estimated blood loss was 600 ml and total intravenous fluids given were 1000 ml crystalloid.

The patient remained hemodynamically stable in atrial fibrillation without rapid ventricular response throughout the post-operative period. Intra-operative cultures ultimately grew *anaerococcus vaginalis* and the patient was started on intravenous vancomycin for 6-week course on recommendations from the infectious disease team. A discussion involving the surgical and anesthesia teams was had with the patient and his family regarding the potential of future re-operation with a temporary

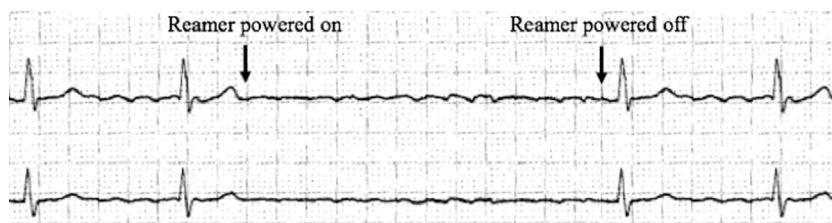


Fig. 2. Example of intra-operative electrocardiogram tracing demonstrating transition from atrial fibrillation to asystolic cardiac arrest with powering of the femoral reamer and return to baseline atrial fibrillation when reamer was powered off.



Fig. 3. Post-operative anteroposterior radiograph of the pelvis demonstrating successful removal of hardware and right girdlestone amputation.

cardiac pacemaker or transvenous pacing, but the patient has elected to not pursue additional surgical intervention.

3. Discussion

The incidence of intra-operative cardiac dysrhythmias during elective orthopaedic surgery is rare and etiology is often unknown. In 2002, Gallagher and Wilkinson published a small case series involving four patients who experienced severe intra-operative bradycardia and even transient asystolic cardiac arrest during total knee arthroplasty whenever high pressures were applied to the distal femoral diaphysis with a hammer.⁵ Similar observations were seen in an additional two patients in their series during reaming of the femoral intramedullary canal. In either scenario, cessation of the applied stimulus resulted in the immediate reappearance of electrocardiogram complexes and pulse oximetry waveforms in each patient, which was similar to the patient presented in this report.⁵ All patients in their series ultimately underwent uneventful completion of the surgical procedure and demonstrated no adverse symptoms in the post-operative period.

Gallagher and Wilkinson postulated that the cause of asystolic arrest in these patients was secondary to the transmission of pressure into the hip joint and the abdominal cavity resulting in a parasympathetic mediated reflex bradycardia: the Gallagher-Wilkinson reflex.⁵ Reflex bradycardia is traditionally associated with the well described carotid sinus reflex. In this pathway, baroreceptors in the carotid sinus sense increases in systemic blood pressure and utilize the parasympathetic nervous system to diminish mean arterial pressure by decreasing cardiac output through a reduction in the heart rate.⁶ Although a specific pathway linking the femoral canal and the heart has not been identified, experimental animal studies have demonstrated that the femur experiences massive increases in pressure during reaming of the intramedullary canal.⁷ Whether this supraphysiologic increase in intramedullary pressure with reaming results directly in the activation of the parasympathetic nervous system or indirectly through transmission of pressure to visceral organs, either theory would offer a logical explanation for the asystolic arrest observed in the patient presented in this report.

Alternatively, an intra-operative cardiac event such as a myocardial infarction could offer another potential explanation for the asystolic rhythm observed during reaming of the femoral canal. Acute coronary syndromes occur as the result of the

development of a luminal thrombus on a ruptured atherosclerotic plaque, coronary vessel spasm, or rapid increases in myocardial oxygen consumption.⁸ In the perioperative period, metabolic factors secondary to surgical trauma, administration of various anesthetics, and rapid volume shifts can precipitate acute coronary syndrome in the susceptible patient.⁸ As the patient in this report did have a fairly extensive pre-operative cardiac history including remote myocardial infarction, NYHA class II congestive heart failure, and chronic atrial fibrillation, he was certainly susceptible to sustaining an intra-operative cardiac event despite a pre-operative RCRI score of 2. The asystolic rhythm that was observed in this case, however, was transient, reproducibly associated with powering of the femoral reamer, and resolved without any intervention from the anesthesia team. An intra-operative cardiac event, such as a myocardial infarction, would be expected to have produced persistent deficits that would have necessitated more extensive intervention prior to resolution making this a less likely explanation.

Fat embolism is another possible source for asystolic rhythm observed during preparation of the patient's femoral canal. Fat embolization syndrome is classically described as a triad of pulmonary, neurological, and cutaneous manifestations that occurs within 24–72 hrs of a traumatic long bone fracture or surgical intervention.⁹ Clinical manifestations of the syndrome are variable and can range from mild tachypnea to multisystem organ failure and treatment is supportive.^{4,9} Although the signs and symptoms of fat embolization syndrome have been well described in the literature, the pathophysiology of this process remains less clear.^{4,9} The prevailing theory is that pressurization of the femoral canal causes intravasation of intramedullary contents including marrow fat into systemic circulation.^{4,9} Marrow fat embolization is thought to elicit systemic effects at end organ systems through a variety of mechanisms including the mechanical obstruction of terminal vessels, release of humeral mediators, and changes in cellular osmolality.^{4,9} The transient nature of the asystolic rhythm occurring only during reaming of the femur in this patient along with the absence of any of the traditional symptoms of fat embolization in the post-operative period, however, make this syndrome a less likely explanation.

4. Conclusion

The definitive etiology for the transient, reproducible, intra-operative asystolic rhythm observed in this patient during pressurization of the femoral canal remains unknown. Although an intra-operative cardiac event or fat embolization could potentially have caused this cardiac dysrhythmia, the paucity of other symptoms classically associated with these syndromes in this patient along with the absence of any residual symptoms in the post-operative period make these explanations less likely. As the cause of the asystolic rhythm was unclear intra-operatively, the patient was stable, and the case was at a safe stopping point, we collectively decided to abort the placement of antibiotic cement spacer in fear of eliciting a persistent cardiac dysrhythmia. In retrospect, we believe that the etiology of this cardiac dysrhythmia occurred secondary to a parasympathetic mediated response that has been previously described briefly in the literature as the Gallagher-Wilkinson reflex. Further studies are needed to corroborate the observations presented in this report and to shed more light on the etiology of this event, but in the meantime, we recommend surgeons remain vigilant for this phenomenon during preparation of the femoral canal.

Conflict of interest

None.

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