

Case Report

A Case of Pituitary Apoplexy Following Total Hip Arthroplasty

Li R, Smith D, Sambaziotis C, Kim S*, Courtney J and Cobelli NJ

Department of Orthopedics & Rheumatology, Montefiore Medical Center, USA

*Corresponding author

Kim S, Department of Orthopedics & Rheumatology, Montefiore Medical Center, Bronx, New York, USA,

E-mail: SUKIM@montefiore.org

Submitted: 08 December 2013

Accepted: 05 January 2014

Published: 07 January 2014

Copyright

© 2014 Kim et al.

OPEN ACCESS

Abstract

Pituitary apoplexy following total knee or hip replacement is a rarely documented postoperative complication and is caused by the sudden enlargement of a preexisting pituitary adenoma. Patients with this condition commonly complain of severe headache, altered mental status, ophthalmoplegia, and visual changes secondary to the mass effect of the tumor on its surrounding structures. We present a case of a 64-year-old male who underwent elective total hip arthroplasty and developed postoperative pituitary apoplexy secondary to a pituitary adenoma that was subsequently surgically resected. We believe that early diagnosis and surgical treatment with a multidisciplinary team led to a favorable outcome for this potentially fatal and debilitating condition.

INTRODUCTION

Pituitary apoplexy following total knee or hip replacement is a rarely documented complication during the immediate postoperative period and is caused by the sudden enlargement of a pituitary adenoma secondary to infarction, hemorrhage, or both. Patients with this condition most commonly present with headache, visual deficits, ophthalmoplegia, hypopituitarism, altered levels of consciousness, or even death [1,2]. In the majority of patients there is no prior diagnosis of pituitary adenoma, which may make the diagnosis difficult and delay time to definitive management. Predisposing factors have been reported to include surgery (most commonly cardiac), spinal anesthesia, anticoagulant therapy, pregnancy, pituitary stimulation tests, and head trauma [2,3-5].

To our knowledge, there are no published reports of pituitary apoplexy in the U.S. following total joint arthroplasty. There were two reports out of the United Kingdom: one after a total hip and total knee replacement in two separate patients and one of pituitary apoplexy after total hip replacement in a patient who developed profound postoperative hyponatremia. Additionally, one case was reported after bilateral total knee replacement out of New Delhi as well as one case in a patient following a total hip arthroplasty included in a neurosurgery review. With appropriate and timely surgical intervention, the patients with reported long-term follow-up were all able to recover to near baseline vision and neurologic function [6,7].

CASE REPORT

In January 2011, a 64-year-old male underwent an elective left total hip arthroplasty for osteoarthritis of the hip. The patient had a past medical history significant for hypertension, hyperlipidemia, and coronary artery disease treated with angioplasty and two stents. He underwent stress testing several

months prior to surgery, which was negative, and he was deemed medically optimized for surgery by his private cardiologist. His clopidogrel was held 5 days prior to surgery. The patient received spinal anesthesia and had an uneventful intraoperative course with minimal reported blood loss and no hypotensive episodes. On postoperative day 3, the patient complained of substernal chest pain and was diagnosed with a non-ST elevation myocardial infarction (NSTEMI). He was treated with aspirin and a heparin drip and two days later was lethargic and complained of severe bifrontal headache. Physical exam revealed acute right-sided ptosis, anisocoria, ophthalmoplegia, and blurry vision with bitemporal hemianopsia. A head CT followed by a brain MRI revealed a 3.4 cm pituitary macroadenoma with mass effect on the optic chiasm and the left cavernous sinus (Figure 1). Laboratory studies demonstrated panhypopituitarism. He was found to be persistently hypotensive and was started on high-dose intravenous steroids. The patient was taken by the neurosurgery team for urgent transsphenoidal resection and decompression and his neurologic symptoms improved postoperatively. Upon his most recent follow-up with his neurosurgeon over 2 years

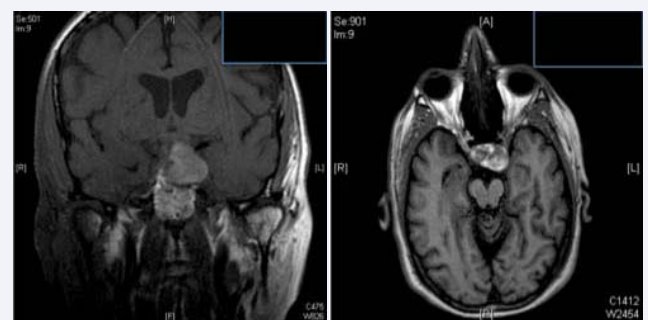


Figure 1 Coronal and Axial MRI showing a pituitary macro adenoma 3.4 cm in size.

after surgery, his visual fields have improved to 100% and 90% in his left and right eyes, respectively, and he is on chronic pituitary substitution for panhypopituitarism.

DISCUSSION

The name pituitary apoplexy was first coined in 1950 by Brougham et al. who recognized the clinical syndrome in 5 cases [1]. The exact mechanism behind the development of pituitary apoplexy is still poorly understood to this day. Various theories have been presented to explain the pathophysiology of this condition but none have been proven to date.

One proposed mechanism is that a preexisting pituitary adenoma outgrows its blood supply leading to ischemia, followed by hemorrhage [1]. A second hypothesis is that as the tumor grows, the diaphragma sellae compresses the arterial blood supply resulting in ischemic necrosis of the adenoma. It has also been proposed that intrinsic vasculopathy may exist in pituitary tumors that make them more prone to undergo ischemia or hemorrhage [3]. Based on previous operative findings and autopsy reports, it is inconclusive whether the primary condition behind pituitary apoplexy is initial infarction followed by secondary hemorrhage, or if some only have ischemic infarction and others undergo primary hemorrhage [5]. The general consensus amongst the orthopaedic literature is that postoperative pituitary apoplexy is caused by some combination of intraoperative or postoperative hypotension, anticoagulation, and/or microemboli leading to infarction [6-8].

In the case report of pituitary apoplexy following bilateral total knee replacement by Khandelwal et al. in 2005, the cause of pituitary apoplexy was thought to be postoperative systemic anticoagulation with heparin leading to hemorrhage of the pituitary gland, which has also previously been reported in patients who underwent cardiac bypass [8]. They hypothesized that the administration of low molecular weight heparin following surgery was likely the main cause of pituitary apoplexy, as the patient became symptomatic 12 hours following its administration. Their secondary hypothesis was that arthroplasty surgery is associated with particulate microemboli (fat, air, marrow, or cement) that may enter the cerebral circulation leading to this condition [8].

Goel et al. believed that although there was no evidence of intraoperative or immediate postoperative complications following their case of total hip arthroplasty, there may have been a transient episode of hypotension postoperatively leading to a vascular insult to the pituitary adenoma. In their second case of a total knee arthroplasty, the authors believed that the occurrence of myocardial infarction and neurologic complications within a short timeframe of each other suggested microembolism may have caused both events [6].

Thomason et al. reported a similar case of pituitary apoplexy following total hip arthroplasty in a patient who underwent a contralateral total hip replacement just ten months prior without complication. The authors proposed that the complication occurred only after the second surgery because the patient began taking clopidogrel and aspirin after developing a forefoot embolism, which increased his risk for intracerebral hemorrhage. The authors also focused on the importance of recognizing the

physiologic effects of hypopituitarism. If unrecognized, there is a greatly increased risk of perioperative hypoglycemia, hypothermia, water intoxication, and respiratory failure. Therefore, if the diagnosis of pituitary adenoma is known before surgery, replacement substitution therapy including steroid coverage with hydrocortisone should be planned for and readily available postoperatively [7].

The patient presented in the current case possessed several predisposing factors that may have led to his pituitary apoplexy. Undergoing a total hip or knee arthroplasty may be an independent risk factor for this condition. Administration of spinal anesthesia may have caused a transient reduction in systolic blood pressure leading to low perfusion pressure to the pituitary gland [3]. His myocardial infarction and hypotension in the early postoperative period may have also led to an ischemic event in the pituitary gland. The occurrence of an NSTEMI and pituitary apoplexy in close proximity to one another may have been caused directly by microemboli from surgery leading to infarction. Lastly, the administration of a heparin drip and aspirin after the diagnosis of NSTEMI, as well as his prior clopidogrel medication, may have made him more susceptible to a hemorrhagic event in the tumor postoperatively.

The recommended management for an acute pituitary apoplexy with profound neurologic symptoms is early surgical decompression and high dose intravenous steroids [6,9]. Missed or delayed diagnosis may result in permanent visual and neurologic changes, and may potentially be fatal [10]. A retrospective review of 35 patients who underwent transsphenoidal hypophysectomy for pituitary apoplexy showed that overall, surgery improved visual acuity deficits in 86%, visual field deficits in 76%, and ocular paresis in 91%, with a significant improvement in visual acuity and visual fields in those who underwent surgery within 8 days of presentation [4]. The authors also advocated for surgical decompression over nonsurgical management, citing that conservative management may increase the chances of recurrence [1,4]. Their results supported the previous findings by Bills et al. who found that surgery resulted in improvement in visual acuity deficits in 88%, visual field deficits in 95%, and ocular paresis in 100% of their 37 patients. In their study, those who underwent surgery within a week of apoplexy had significantly greater recovery in their visual acuities [9]. At his most recent follow-up, our patient demonstrated almost full improvement in his visual fields bilaterally compared to initial presentation.

CONCLUSION

The diagnosis of pituitary apoplexy after total joint arthroplasty may be difficult to make given its extremely rare presentation and patients' lack of prior diagnosis of pituitary adenoma in the vast majority of cases. It is important to recognize predisposing factors as well as the perioperative events hypothesized to lead to this condition including spinal anesthesia, hypotension, and systemic anticoagulation or antiplatelet therapy. In addition, it is important to recognize the classic symptoms of pituitary apoplexy, which include headache, visual changes, and altered mental status, and to pay careful attention to laboratory studies that reveal panhypopituitarism so that early diagnosis may be achieved. Surgical intervention

should be performed on a diagnosed pituitary apoplexy within 1 week in order to improve long-term visual function and prevent mortality. We believe that our early recognition and treatment of this patient with a multidisciplinary team led to his successful outcome from this potentially devastating condition.

REFERENCES

1. BROUGHAM M, HEUSNER AP, ADAMS RD. Acute degenerative changes in adenomas of the pituitary body with special reference to pituitary apoplexy. *J Neurosurg.* 1950; 7: 421-439.
2. Cardoso ER, Peterson EW. Pituitary apoplexy: a review. *Neurosurgery.* 1984; 14: 363-373.
3. Lennon M, Seigne P, Cunningham AJ. Pituitary apoplexy after spinal anaesthesia. *Br J Anaesth.* 1998; 81: 616-618.
4. Randeve HS, Schoebel J, Byrne J, Esiri M, Adams CB, Wass JA, et al. Classical pituitary apoplexy: clinical features, management and outcome. *Clin Endocrinol (Oxf).* 1999; 51: 181-188.
5. Semple PL, Jane JA Jr, Laws ER Jr. Clinical relevance of precipitating factors in pituitary apoplexy. *Neurosurgery.* 2007; 61: 956-961.
6. Goel V, Debnath UK, Singh J, Brydon HL. Pituitary apoplexy after joint arthroplasty. *J Arthroplasty.* 2009; 24: 826-829.
7. Thomason K, Macleod K, Eyres KS. Hyponatraemia after orthopaedic surgery - a case of pituitary apoplexy. *Ann R Coll Surg Engl.* 2009; 91: W3-5.
8. Khandelwal M, Chhabra A, Krishnan S. Pituitary apoplexy following bilateral total knee arthroplasty. *J Postgrad Med.* 2005; 51: 155-156.
9. Bills DC, Meyer FB, Laws ER Jr, Davis DH, Ebersold MJ, Scheithauer BW, et al. A retrospective analysis of pituitary apoplexy. *Neurosurgery.* 1993; 33: 602-608.
10. David NJ. Pituitary apoplexy goes to the bar: litigation for delayed diagnosis, deficient vision, and death. *J Neuroophthalmol.* 2006; 26: 128-133.

Cite this article

Li R, Smith D, Sambaziotis C, Kim S, Courtney J, et al. (2014) A Case of Pituitary Apoplexy Following Total Hip Arthroplasty. *Ann Orthop Rheumatol* 2(1): 1007.