International Journal of Research in Orthopaedics
Suresha B et al. Int J Res Orthop. 2017 Sep;3(5):1081-1083
http://www.ijoro.org

DOI: http://dx.doi.org/10.18203/issn.2455-4510.IntJResOrthop20173946

Case Report

Silent meningioma: a cause of quadriperesis in total knee arthroplasty

Suresha B.[1], Chandrashekar P.[2]*, Nataraj H. M.[2]

Department of Orthopaedics, [1]Subbaiah Institute of Medical Sciences, Shivamogga, [2]Sakra World Hospital, Bangalore, Karnataka, India

Received: 21 July 2017
Accepted: 10 August 2017

***Correspondence:**
Dr. Chandrashekar P.,
E-mail: shekharbones@yahoo.com

ABSTRACT

Total knee arthroplasty (TKA) is a treatment for end stage osteoarthritis. Majority of them in the older age group with associated co-morbidities and increased the risk for stroke. Asymptomatic meningiomas may become symptomatic and presents with variable symptoms depending on the size and location of the tumour. We report a case of a 58 years old lady who underwent bilateral TKA under combined spinal epidural anaesthesia with an uneventful post-operative course with post-operative analgesia. Patient developed dysphasia and weakness in upper and lower limbs. Following computed tomography of the brain revealed a meningioma in the falx region. Patient treated with steroid and underwent elective surgery for meningioma without any complications. Asymptomatic brain tumours can become symptomatic after surgery and urgent initiation of neurologic evaluation and strong recommendation to initiate intracranial pressure management oriented therapy in time.

Keywords: Quadriperesis, Meningioma, TKA

INTRODUCTION

Total knee arthroplasty (TKA) has been a standard procedure for patients with late-stage osteoarthritis of the knee. Older patients who undergo more complex procedures like TKA tend to be at greater risk for stroke, even if the nominal risk of stroke is low. Following TKA, the number of high-risk patients who are undergoing those procedures implies that the overall risk for stroke following TKA is also increasing.[1] Perioperative stroke in joint arthroplasty patient is usually attributed to a history of non-coronary heart disease, emergency surgery than elective surgery, general anaesthesia than regional anaesthesia, and an intraoperative arrhythmia or other alterations in the heart rate during surgery.[2]

Meningiomas are benign, slow growing tumours with a long initial asymptomatic phase.[3] The neurological deficit seemed to be related to the tumour location and size, and the severity of the peritumoural oedema. The considerable peritumoural oedema, probably related to their cellular morphology and ability to release vasoactive substances.[4]

To our knowledge, there are no documented cases of a meningioma causing quadriperesis in post TKA. Here we report an uncommon cause of quadriperesis in a case of silent meningioma following TKA.

CASE REPORT

A 58 year old lady with history of pain in both the knees and deformity since few years was diagnosed to have osteoarthritis and planned for bilateral TKA. Patient not had any medical co-morbidity. All preoperative investigations were within normal limits. Fitness for surgery was taken from the respective departments.

Patient underwent bilateral total knee replacement (Figure 1) with combined spinal epidural anaesthesia with postoperative epidural analgesia. Epidural analgesia was continued up to 3rd day of postoperative period. On

5th post-operative day patient developed neurologic symptoms comprising of dysphasia and weakness in upper and lower limbs. However, sensation was intact. There were no symptoms of headache, vomiting, convulsions, facial asymmetry, visual diminution and bowel bladder involvement. All the vital parameters were within normal limits.

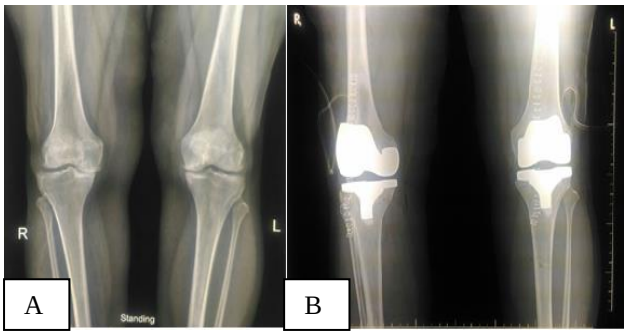

Figure 1: a) Pre-operative radiographs showing arthritic changes. B) Post-operative radiographs with implant in-situ.

Blood investigations revealed normal electrolyte level and other possible metabolic causes were ruled out. ECG and ECHO were done to rule out cardiac causes. A computed tomography of the brain showed an isodense lesion, suggestive of meningioma in the falx region projecting bilaterally more on left side, measuring 4.6×4.5×5 cm and associated with marked parenchymal oedema around the tumour with little mass effect (Figure 2). The patient was immediately referred to neurologist and neurosurgeon for management of the intracranial tumour. Intravenous steroid (IV dexamethasone 4 mg 12 hourly) was started and later converted oral steroid in tapering dose. Oral antibiotics were continued till tapered dose of steroid stopped. The response to therapy was good and patient condition improved neurologically. Patient managed to ambulate with a walking frame and discharged from the hospital. Patient was scheduled for neurosurgical check-up and underwent elective surgery for meningioma with uneventful events.

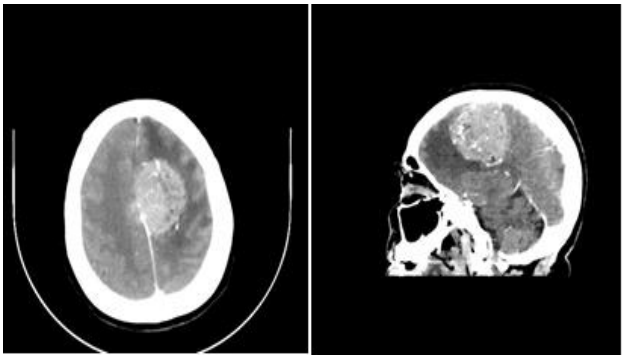

Figure 2: An isodense lesion in the falx region associated with marked parenchymal oedema around the tumour with little mass effect.

DISCUSSION

Meningiomas are benign, slow growing tumours and may cause a slow increase in the intracranial pressure with mild or no symptoms for a long time with a long initial asymptomatic phase, and may remain silent until the patient's sudden death.[3] Only 3%–6% of clinically detected asymptomatic meningiomas later become symptomatic.[5] When symptomatic, intracranial meningiomas present a wide variety of symptoms arising from the compression of adjacent structures, direct invasion of or reactive changes in the adjacent brain tissue, and obstruction of cerebrospinal fluid pathways, cortical veins, or major venous sinuses.[6] The neurological deficit seemed to be related to the tumour location and size, and the severity of the peritumoural oedema. The considerable peritumoural oedema, probably related to their cellular morphology and ability to release vasoactive substances.[4] and also cause structural damage to the cerebral endothelium, and the severity of cerebral oedema seems to be related to the size of the tumour due to cellular disruption over a wide area.[7]

Slowly growing intracranial tumours may go unnoticed because solid growth initially displaces blood and cerebrospinal fluid from within the skull and there is no absolute increase in intracranial contents or pressure. When this readily movable compartment is displaced, further small increases in size of tumour will lead to compression of solid tissue within the skull. The compliance decreases exponentially[8] and an extra millilitre of cerebrospinal fluid, blood or tumour can now be accommodated only at the expense of a significant rise in intracranial pressure. As the compensatory reserve decreases, the patient reaches such a precarious state that he becomes vulnerable to the normally tolerable insults of everyday life, any of which may result in acute decompensation with mass displacement of brain or brainstem, and a dramatic presentation of an intracranial lesion. As was first recognised by Monroe-Kellie, such as the interdependence between the parenchymatous, cerebrospinal fluid, and vascular compartments within the skull that the culprit is irrelevant, and an increase in any of the components is equally damaging.[9]

It seems that these asymptomatic tumours were approaching the limits of their compensatory mechanisms before operation, but as they would have had a normal Glasgow coma score (GCS) before operation, it is unlikely that intracranial pressure was critically raised.[4] They become symptomatic once there disturbance in the equilibrium within the skull.

Symptoms and signs may include seizure disorders, raised intracranial pressure sign, classic early morning headaches, focal neurological deficits, such as motor and sensory disorders, ataxia, language dysfunction, cranial neuropathies, psychomotor symptoms, and behavioural disturbances.[6]

There are only a few reports of asymptomatic brain tumours complicating the postoperative course of non-neurosurgical patients.[4,10,11] Also, few reports of meningioma presenting as stroke in cardiac surgery.[12] The present case is of a meningioma manifested as stroke in post-operative period of TKA. The standard literature search done did not yield a single case of asymptomatic meningioma becoming symptomatic in the immediate post op period following TKA.

Computed tomography or magnetic resonance imaging of the brain should be performed immediately upon suspicion of neurological insult in the early postoperative course. The CT scan of the brain confirmed the diagnosis by revealing the classical features of an isodense lesion in the falx region projecting bilaterally more on left side, measuring 4.6×4.5× 5 cm and associated with marked parenchymal oedema around the tumour with little mass effect.

Anti-oedema therapy with intravenous dexamethasone, prolonged antibiotic therapy, cautious fluid and electrolyte management are recommended in cases of focal neurological deficit with less severe deterioration of consciousness level. The role of dexamethasone in reducing peritumoural blood brain barrier permeability and inhibition of vascular endothelial growth factor action is well reported in the literature.[13]

CONCLUSION

In conclusion, asymptomatic brain tumours can be precipitated after surgery. When become symptomatic urgent initiation of neurologic evaluation and strong recommendation to initiate intracranial pressure management oriented therapy in time.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Aubrun F, Marmion F. The elderly patient and postoperative pain treatment. Best Pract Res Clin Anaesthesiol. 2007;21(1):109–27.
2. Mortazavi SMJ, Kakli H, Bican O, Moussouttas M, Parvizi J, Rothman RH. Perioperative stroke after total joint arthroplasty: prevalence, predictors, and outcome. J Bone Joint Surg Am. 2010;92(11):2095–101.
3. Nakasu S, Nakasu Y, Fukami T, Jito J, Nozaki K. Growth curve analysis of asymptomatic and symptomatic meningiomas. J Neurooncol. 2011;102(2):303–10.
4. Razis PA, Robinson DL, Alberry R. Clinical presentation of “silent” meningiomas after general anaesthesia. Br J Anaesth. 1995;74(3):335–7.
5. Yoneoka Y, Fujii Y, Tanaka R. Growth of incidental meningiomas. Acta Neurochir (Wien). 2000;142(5):507–11.
6. Whittle IR, Smith C, Navoo P, Collie D. Meningiomas. Lancet Lond Engl. 2004;363(9420):1535–43.
7. Harris DN, Oatridge A, Dob D, Smith PL, Taylor KM, Bydder GM. Cerebral swelling after normothermic cardiopulmonary bypass. Anesthesiology. 1998;88(2):340–5.
8. Miller JD, Garibi J, Pickard JD. Induced changes of cerebrospinal fluid volume. Effects during continuous monitoring of ventricular fluid pressure. Arch Neurol. 1973;28(4):265–9.
9. Fraser AC, Goat VA. Unrecognised presentation of a meningioma. Delayed recovery after general anaesthesia presenting as a sign of intracranial pathology. Anaesthesia. 1983;38(2):128–31.
10. Giordano F, Urciuoli RM, Ragazzi P, Griva F, Cossandi C, Fontanella M, et al. Sudden presentation of intracranial meningioma after elective general anesthesia. J Neurosurg Anesthesiol. 2001;13(2):138–42.
11. Young HA, Smith H, Gregory SA, Gleave J. Intracranial meningioma unmasked by elective general anesthesia: report of three cases. Neurosurgery. 1984;14(3):328–31.
12. Chow HK, Yousafzai SM, Ugurlucan M, Canver CC. Silent meningioma – a rare cause of stroke in post-cardiopulmonary bypass patients. Arch Med Sci AMS. 2010;6(2):176–280.
13. Heiss JD, Papavassiliou E, Merrill MJ, Nieman L, Knightly JJ, Walbridge S, et al. Mechanism of dexamethasone suppression of brain tumor-associated vascular permeability in rats. Involvement of the glucocorticoid receptor and vascular permeability factor. J Clin Invest. 1996;98(6):1400–8.

Cite this article as: Suresha B, Chandrashekar P, Nataraj HM. Silent meningioma: a cause of quadriperesis in total knee arthroplasty. Int J Res Orthop 2017;3:1081-3.