NEUROLOGICAL SEQUELAE FOLLOWING ANAESTHETIC RECOVERY AFTER BILATERAL TOTAL KNEE REPLACEMENT – TWO CASE REPORTS

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ABSTRACT

BACKGROUND
Thromboembolism is a common and serious complication of joint replacement surgery. Cognitive decline occurs in 5-29% of patients, undergoing major orthopaedic surgery. Many studies show that systemic embolism may occur in the absence of venous-arterial shunts leading to cognitive dysfunction and neurological sequelae.

METHODS
We present two cases of neurological consequences occurring post bilateral TKR. Cases were successfully done under Combined Spinal Epidural Anaesthesia. Steroids were not used intraoperatively.

RESULTS
Both cases had neurological complications following completion of surgery. First case had posterior circulation TIA while the other had a right upper motor neuron facial palsy.

CONCLUSION
We suspect both as cases of cerebral fat microembolism in the absence of any venous-arterial shunt.

KEYWORDS
Neurological, Anaesthetic, Knee Replacement.

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INTRODUCTION: Thromboembolism is a common and serious complication of joint replacement surgery. Without deep venous thrombosis prophylaxis, reported incidences are 50% in patients undergoing total hip replacement and 84% in total knee replacement patients. Incidence with deep venous thrombosis prophylaxis has been reported to be 2-12%.[1] Pulmonary embolism is seen to occur in 20% of post THR and 7% of post TKR patients.[1] Additionally, cognitive decline occurs in 5-29% of patients undergoing major orthopaedic surgery.[2,3] Various studies have shown detection of intraoperative cerebral microembolism using transcranial Doppler[4,5] and MRI[6,7] during orthopaedic surgery, and have presumed these to be lipid in nature. Further, it is postulated in many studies that systemic embolism may occur due to venous-arterial shunts in patients.[8] However, there are evidences to show that systemic embolism may occur even in absence of venous-arterial shunts, leading to cognitive dysfunction and neurological sequelae.[9] Here, we report 2 cases of neurological complications post bilateral TKR.

Case Report I: A 70-year-old female was scheduled for bilateral TKR. On preoperative checkup, the patient was a controlled hypertensive with no other co-existing illness. She had a history of cataract surgery done two years back. Routine investigations like haemogram, TLC, DLC, KFTs, LFT, random blood sugar, PT (INR), PTTK and ECG were within normal limits. Echocardiography was also normal with ejection fraction of 60%.

In the operating room, routine monitors were attached (blood pressure, heart rate, ECG, SpO_2) and two 18G IV lines were secured. Combined spinal epidural block was performed in sitting position, with Inj. bupivacaine 0.5% heavy 2.2 mL and Inj. fentanyl 10 µg. Patient was made to lie supine and sensory level of T_{10} was achieved. Tourniquets were sequentially inflated in both lower limbs starting with right knee. Cemented prosthesis was used. The intra-operative period was uneventful and total tourniquet time of both limbs was 1 hour 30 minutes and tourniquet pressure of 290 mmHg.

At the time of dressing, the patient complained of numbness and weakness of tongue with abnormal sensations in tongue and slurring of speech. The patient was conscious, oriented with GCS of 15/15. Baseline parameters

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were normal with BP=144/70 mmHg, HR=65/min, SpO_2=98% on O_2 and normal ECG tracing. Chest was bilaterally clear. Sensory level of the block was L_1 and she could move both ankles (Bromage scale score 2).

On neurological examination, there was no loss of sensation over the face. Power in both upper limbs was 5/5. Deep tendon reflexes were normal. Cranial nerves examination showed 12th nerve paresis on (R) side with deviation of tongue towards same side. She had mild ataxia of cerebellar type. Instillation of 100% oxygen and observation of vital parameters were continued. Neurological deficits recovered within 1 hour suggesting posterior circulation TIA. She was shifted to postoperative room. MRI brain was also done within 2 hours. Findings in MRI showed a patchy area of ischemia in the cerebellum & lower brainstem in the form of hypointensities on T1W, hyperintensities on T_2 and FLAIR and diffusion restriction on DWI (Fig. 1, 2). Saline bubble echocardiography was done on the patient to rule out any venous-arterial shunts such as patent foramen ovale, but came out to be negative. Remaining hospital stay of two weeks was uneventful.

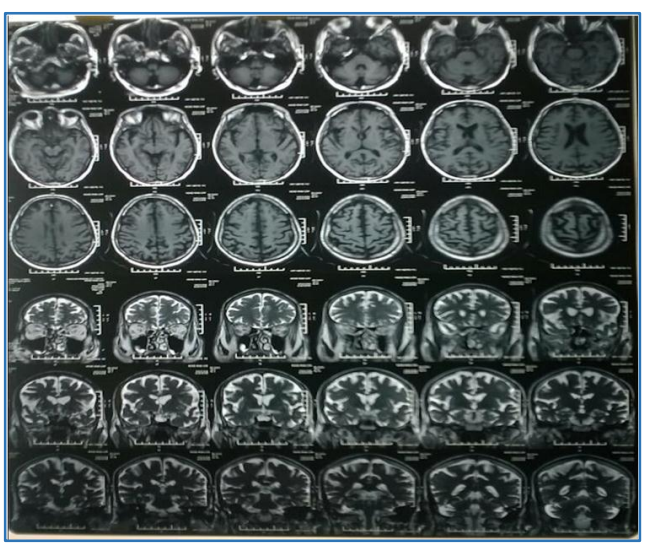

Fig. 1

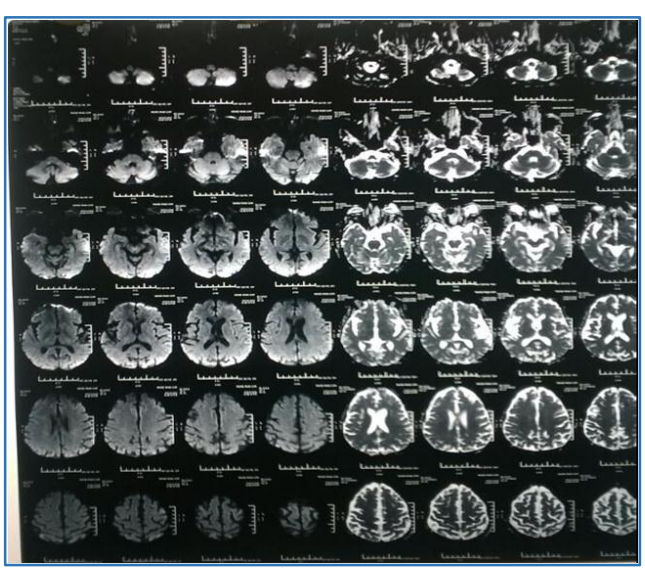

Fig. 2

Case Report II: An 80-year-old male was posted for bilateral TKR. On preoperative checkup, the patient was known diabetic and hypertensive, with controlled blood sugar and blood pressure. Routine investigations were within normal limits. Echocardiography was also normal showing ejection fraction of 58%.

Routine monitoring (BP, HR, ECG, SpO_2) was stared in operating room and two 18G IV cannulas were secured. Anaesthesia was administered using combined spinal epidural technique using Inj. bupivacaine 0.5% heavy 2.4 mL and Inj. fentanyl 10 ug. Sensory level up to T_8 was achieved. Steps of surgery were similar as previous case. Total tourniquet time was 1 hour 50 minutes and pressure was 280 mmHg. Intraoperative period was uneventful. Patient was shifted to the postoperative recovery room after surgery.

About 12 hours postop, the patient was noticed to have deviation of angle of mouth towards left side. All vital parameters like BP, HR, SpO_2, ECG were normal.

On neurological examination, patient was conscious and alert. Pupils were equal and reacting. Cranial nerve examination showed right upper motor neuron type of facial paresis. There were no other deficits. MRI brain done within 3 hours of onset showed areas of ischemia in right posterior frontal region as shown by T_1 hypointensity, T_2 and FLAIR hyperintensity and diffusion restriction on DWI (Fig. 3).

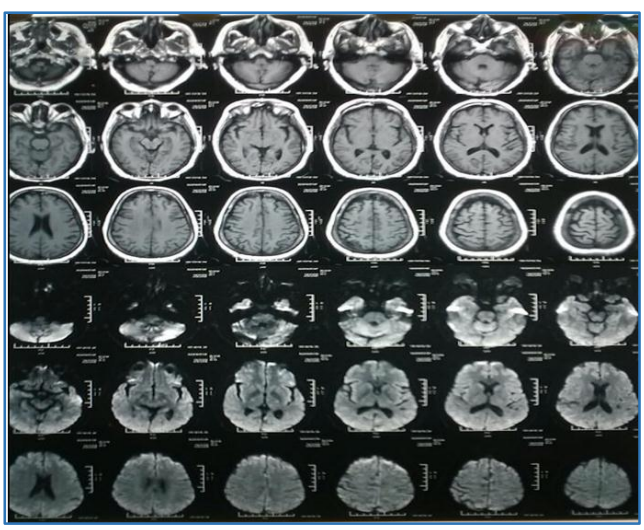

Fig. 3

Saline bubble echocardiography was done on this patient as well to rule out paradoxical systemic embolism during or after surgery, which came out to be negative. The patient was put on followup under neurologist. He was started on antiplatelet medication by the neurologist. There was no other event reported. He was discharged from hospital after 2 weeks and recovered from facial palsy after about 4 weeks.

DISCUSSION: Brain embolisation occurs in 40-60% patents undergoing joint arthroplasty.[4,5,7,8,9,10] Many studies report the occurrence of paradoxical cerebral fat embolism in patients of long bone fractures and joint arthroplasty having patent foramen ovale.[11,12,13]

But in patients without any venous-arterial shunts, there are reports in literature showing that large number of fat emboli rapidly cross the pulmonary circulation (less than 4 hours) in joint arthroplasty[9] as well as in femoral fractures.[14] An experimental study on dog also suggests that fat emboli traverse the lung vasculature rapidly within 3 hours of femoral nailing.[15]

Histological examination of patients with embolism after long bone fractures, femoral nailing or joint arthroplasty has shown pulmonary microvasculature occlusion with fat emboli. These emboli are believed to reach systemic circulation through pulmonary precapillary shunts or directly cross the pulmonary capillary bed.[13] Microvascular lodging of fat globules also produce local ischemia, release of inflammatory mediators, vasoactive amines and platelet aggregation. There is also a release of free fatty acids as chylomicrons. Acute phase reactants such as C-reactive protein cause chylomicrons to coalesce and create physiological reactions.[12]

The lipid nature of these emboli has been clinically co-related to be a cause of post-operative delirium and cognitive dysfunction seen in 44-55% of post-operative elderly orthopaedic patients, especially after hip fracture surgery.[16,17] A case series shows presence of cerebral micro emboli causing neurological dysfunction after long bone fractures detected by transcranial Doppler.[17] A case of acute brain fat embolisation after total hip replacement in the absence of patent foramen ovale leading to mortality of the patient, has been reported in which histological examination of brain showed approximately 5000 emboli/cm^3 of brain tissue.[9] These emboli were found to be identical to those in brain specimens of non-survivors of cardiopulmonary bypass which have been named as small capillary arteriolar dilatations (SCADs) by the author.[9,18]

In our patients, we too suspect that cerebral fat microembolism during knee arthroplasty led to neurological dysfunction even in the absence of patent foramen ovale.

Another issue to be highlighted is that we did not use steroid for prophylaxis of fat embolism in our cases. In literature, we found many reports indicating that corticosteroids may help to reduce complications like fat embolism syndrome in joint replacements and long bone fractures.[19] Reduced levels of IL-6 in blood in patients given three doses of hydrocortisone for prophylaxis of fat embolism syndrome has been shown in one of the studies. Possibly steroids act through inhibition of plasma complement activation, decreasing WBC aggregation and release of toxins. This reduces the capillary leakage and damage to platelet endothelium by reducing free fatty acids.[19]

It should be highlighted that as mentioned in one study there was no difference in the incidence of deep vein thrombosis or pulmonary embolism between general and regional anaesthesia for TKR patients when chemical prophylaxis was administered.[20]

CONCLUSION: Our patients illustrate neurological complications shortly after bilateral knee replacement surgery. We suspect them as cases of cerebral fat microembolism in the absence of any venous-arterial shunt. It may be a possibility that using steroid prophylaxis could have prevented such complication, but this advocates further research to weigh the risks and benefit of steroids.

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