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Case Report

Global instability following primary total knee arthroplasty in patient with osteoarthritis and backache due to missed thoracic meningioma: our experience

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ABSTRACT

Recurvatum or hyperextension instability with bladder dysfunction and impeded walking after total knee arthroplasty due to a thoracic meningioma that was not detected on preliminary neurological evaluation (clinco-radiological) in an osteoarthritic patient with complaint of backache, muscle weakness and claudication. Magnetic resonance imaging (MRI) whole spine revealed a thoracic mass at the level D2-3. Complete resection of spinal mass was done. The knee was revised with rotating hinge prosthesis following spinal surgery. The diagnosis requires an algorithmic approach to identify the cause and best available management options. It is strongly suggested that thorough neurological examination of whole spine should be done preoperatively if lumbar imaging is unconvincing or is incompatible with the history and clinical examination to prevent such serious complications.

Keywords: Total Knee arthroplasty, Meningioma, Osteoarthritis, Recurvatum instability

INTRODUCTION

Instability is one of the most frequent cause of failure of total knee arthroplasty (TKA) and/or revision surgeries.[1,2] Recurvatum Instability following primary total knee arthroplasty is rare and can be very disabling.[3] The etiology can be complex and related to the interaction of several factors; localized or systemic.[3,4] Localized factors such as a severe osseous deformity, including genu valgum, laxity of capsuloligamentous structures, inadequate balancing of soft tissues intra-operatively and trauma, either during or after surgery may play a role. Systemic factors contributing to instability are most often related to generalized ligamentous laxity, neuromuscular disorders, charcot neuro-arthropathy and inflammatory arthropathy. There is a paucity of published literature concerning the same. We herein present a case of a 77 year old female with recurvatum instability with muscle weakness, backache and bladder dysfunction following primary TKA due to thoracic meningioma that was missed on initial neurological evaluation, who was managed adequately and recovered well. Reports on neurological deterioration with laxity of capsuloligamentous structures due to a missed compressive lesion in the spinal cord following TKA has not been reported before in the literature to the best of our knowledge. The present case is reported because of its rarity and to highlight the fact that thorough and complete neurological examination is critical prior to total knee arthroplasty in patients with knee osteoarthritis who also have backache.

CASE REPORT

In 2014, a 77-year old lady presented to the outpatient clinic of Shalby hospital with knee/leg pain with no clear

radiculopathy, bladder dysfunction and progressive walking disability for more than 6 months following primary total knee arthroplasty. She reported pain over the popliteal region while on walking. There was no history of any traumatic event. She felt knee instability (sensation of buckling, shifting or giving way of joint) and muscle weakness on weight bearing. She complained of the knee feeling wobbly or unstable and persistent discomfort. She had significantly more knee pain and activity limitations following TKA. The condition had begun 6 month before after primary TKR with slight weakness of the muscles of the knees and pain. It had increased gradually so that weight bearing was became an arduous task. Clinical examination revealed lower-extremity motor weakness bilaterally (average motor grading 2/5), patellar and Achilles reflexes were exaggerated and anal sphincter tone was found to be reduced equivocal planters. Bladder disturbance/dysfunction was found. She was evaluated using the Western Ontario and McMaster Universities Arthritis Index (WOMAC) score. Examination of the knees revealed good alignment, shifting to recurvatum on weight bearing/ stance phase. There was no local tenderness. The flexion was 112.5 degree, hyperextension extended to 26.5 degrees (Figure 1). There was varus and Valgus laxity. The total WOMAC score was 49.3±7.1. Weight bearing radiographs taken in the post operative period showed well aligned TKR (Figure 2).

Figure 1: Recurvatum (clinically).

No loosening of the components was appreciated. Patellar tracking was found to be normal. Medical history of the patient revealed that she had visited to an orthopaedic surgeon 7 month earlier with complains of lower backache radiating to lower limb with tingling sensation, pain in the right knee with stiffness and impeded walking. She also felt cramping of one or both legs, brought on when walking and relieved in sitting .Her pain became worse with activity and frequently worsens over the course of the day. There was lateral laxity with weakness of muscles.

Figure 2: Radiograph (AP and lateral View) showing TKR (primary).

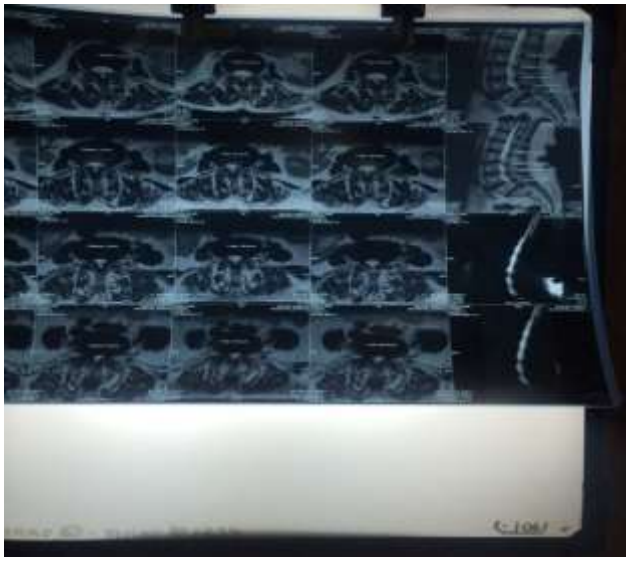

Figure 3: Magnetic resonance imaging (MRI) of lumbar spine: of lumbar spine showed diffuse disc bulge with ligamentum flavum and facetal hypertrophy at L3-L4 causing narrowing of both lateral recesses. There was grade II anterolisthesis of L5 over S1 with bilateral pars interarticularis defects at L5. Paracentral disc bulge with severe ligamentum flavum and facetal hypertrophy at L4-L5 causing spinal canal stenosis.

The roentgenograms of the knee (AP and lateral view) showed osteoarthritis with marked diminution of medial joint space and osteophytes; lumbar spine (AP and Lateral view (flexion and extension)) showed features of lumbar spondylosis, spondylolisthesis (grade II) with decreased L3, L4, L5 and S1 disc space. Magnetic resonance imaging (MRI) (Figure 3) of lumbar spine showed diffuse disc bulge with ligamentum flavum and facetal hypertrophy at L3-L4 causing narrowing of both lateral recesses. There was grade II anterolisthesis of L5

over S1 with bilateral pars Interarticularis defects at L5. Paracentral disc bulge with severe ligamentum flavum and facetal hypertrophy at L4-L5 causing spinal canal stenosis. Her previous medical history was unremarkable. Laboratory tests were normal.

She was prescribed precautions, short course of non-steroid anti-inflammatory (NSAID) drugs, physical therapy and rest. This treatment produced pain relief. Two weeks after the conservative therapy she reported that she had difficulty walking, increased knee pain with stiffness and leg pain with muscle weakness and episodes of numbness and tingling. Orthopaedic Surgeon recommended Total knee replacement (TKR) for knee pain and stiffness (osteoarthritis); conservative treatment for backache with features of degenerative spondylosis/spondylolisthesis with features of radiculopathy. Cemented TKR was done under spinal anesthesia (Figure 2). Two week after TKR the patients reported that the pain spread into the posterior aspect of knee with progressive muscle weakness and difficulty walking. The pain occurred during weight bearing and even at rest. The patient described that she had difficulty walking due to pain and muscle weakness. She developed bladder dysfunction (incontinence).

Figure 4: Magnetic resonance imaging (MRI) of dorsal spine: showed evidence of a well defines oval, intradural extra medullary lesion measuring approximately 2.2 x 1.1 cm at the level of D2-D3.

There was no history of significant injury. She gradually developed genu recurvatum on weight bearing. Brace were recommended but not relieved. She reported to the Shalby Hospital for further assessment and management. After thorough clinical examination, MRI of Lumbar spine with screening studies of the entire spine was recommended. NCV/EMG/EP was advised. MRI showed evidence of a well defines oval, intradural extra medullary lesion measuring approximately 2.2 x 1.1 cm at the level of D2-D3 (Figure 4). It was seen in the anterior and right lateral position and is displacing the thoracic cord to the left. It was hyperintense to the muscle on T2W1 image and isointense to hyperintense on TIWI. There was no extension into the neural foramen. Findings were most likely sign of Meningioma.

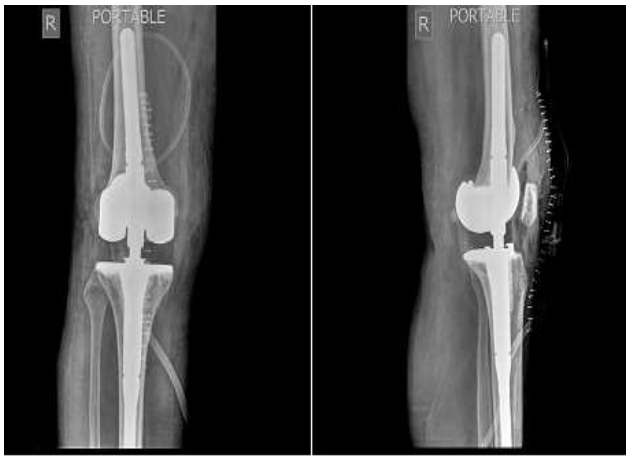

Figure 5: Radiographs showed (AP and lateral view): cemented link rotating hinge prosthesis (Nexgen RHK zimmer; implant size: femur D; tibia-3; insert D20; Patella 29; tibial stem 11/100; femoral stem 14/100).

Figure 6: Latest Follow up (~ 2.2 years): a) radiographs: no subsidence or loosening of prostheses; b) clinical: significant functional improvement with full correction of deformity.

The EMG-NCV study showed sensory motor Predominantly Axonal type of Poly-Neuropathy involving all four limbs. In view of the severe disability/difficulty walking, with gross laxity, revision total knee arthroplasty (TKA) was planned. After clinco-radiological evaluation, Joint replacement team of Shalby Hospital decided to perform revision TKR once the neurological problem has resolved. As a result, the patients were promptly refereed to department of neurosurgery for same. She underwent microsurgical exploration. The spinal cord was decompressed and gross resection of the tumor done. Histopathological examination revealed Meningioma, WHO grade-I. Motor deficit improved post-operatively (From 2/5-4/5).

Three month following the resection of meningioma, she reported that she was able to participate in all functional and recreational activities (WOMAC score improved from 49.3±7.1 to 26.0±13.8). Still she had posterior knee pain and deformity on weight bearing. The Shalby Arthroplasty team has decided to perform revision

surgery. The posterior capsule, lateral and medial ligament was found to be lax. There was no sign of infection and /or loosening of implant. The knee was revised to a cemented link rotating hinge prosthesis (Nexgen RHK Zimmer; Implant size: Femur D; Tibia-3; Insert D20; Patella 29; Tibial Stem 11/100; Femoral stem 14/100) (Figure 5).The knee was stable under stress (Valgus/ varus). The range of motion was from 0 to 120 degree flexion. No hyperextension could be appreciated even if the force applied. Her postoperative period was completely uneventful. She has started "weight bearing as tolerated" after surgery and was discharged on fourth day. She was painfree, had good ambulatory capacity and showed significant functional improvement at the latest follow up (Figure 6).

DISCUSSION

The present report showcased the appropriate method for identifying, diagnosing, and treating the patient with knee osteoarthritis with spinal tumor. This is a significant academic topic in the realm of orthropaedics (arthroplasty). The patient reported neurological deterioration following total knee arthroplasty. Though Reports of neurologic complications after TKA are available there is controversy about the incidence of and predisposing factors to the development of neurological deficit after total knee arthroplasty (TKA).[5-7] The present patient had history suggestive of cord or nerve root compression (compression-related lumbar symptoms) before operation. The primary surgeon decided not to push through with the decompressive surgery of lumbar spine on the basis of an inconclusive lumbar spine MRI and lower extremity motor weakness. He decided to treat the patient conservatively for lumbar disc herniation and canal stenosis symptoms. She was treated with primary total knee arthroplasty for right knee osteoarthritis under spinal anesthesia. She did very well for few days until she began to develop progressive walking disability, knee instability, deformity, and bladder dysfunction. The actual cause for such neurological deterioration was unknown. On admission to our institute, she underwent screening magnetic resonance imaging (MRI) of the whole spines and was found to have severe thoracic compressive lesions (meningioma). Authors speculated that a silent Meningioma aggrevate neurological deficit/symptoms following spinal anesthesia used for primary TKA. Although actual cause is unknown, various postulations have been made to delineate the proper mechanism of neurological worsening in this setting.

Concentrated injected local anesthetic induced neurotoxicity, spinal coning and compression effect of engorged epidural veins has been proposed as a potential cause of neurological deterioration.[8-10] However, the exact pathophysiological mechanism could not be clearly elucidated. Thus, if imaging findings do not correlates to the patient's complaints or the results of the neurological examination, with team of spinal surgeon, authors suggest, should perform a thorough neurological examination. Had the primary surgeon/anaesthestic done thorough neurological screening before surgery, patient would not have undergone repeated surgeries and neurological deterioration.

Though the exact cause of recurvatum instability in the present case was not clear, it was thought to be the weakness of lower extremity and laxity of capsuloligamentous structures owing to thoracic Meningioma as she have not achieved complete neurological recovery (power: 4/5; laxity of ligaments; bladder dysfunction) .The quadriceps weakness poses to instability symptoms (buckling, hyperextension) as the quads tend to maximally contracts concentrically to lock the knee during the gait cycle (heel strike) to compensate for a weak extensor apparatus and thus avoid instability. Weak extensor mechanism with laxity of capsuloligamentous complex in association with deformity was challenging to treat. Still there was considerable controversy concerning the optimal treatment in these uncommon conditions as there is paucity of evidence based literature favoring any particular or specific treatment.

Treatment options include augmentation, ligament reconstruction or imbrications, or use of more constrained prosthesis.[11,12] Augmentation would be the answer if there was deficient bone and hence a loose extension gap. Tensioning of collaterals (medial collateral (MCL) and lateral collateral ligament in extension) or ligament imbrications/reconstruction would be a better option in case of weak posterior tissue (soft tissue) or in view of absence of any malalignment/loosening of component. Owing to incomplete/partial neurological recovery and generalized ligamentous laxity the authors doubt about the success of these procedures. The authors reasserted that she had poor soft tissue quality, thus increasing the risk of further stretching leading to recurrence risk of recurvatum. In view of her advanced age, severe deformity, global instability about the knee and partial neurological recovery authors preferred to use rotating-hinge total knee prosthesis. She had achieved an improvement in functions, pain and range of motion of the knee. No subsidence or loosening of prostheses was observed at the latest follow-up. The positions of the components were considered optimal in her knee.

CONCLUSION

Revision with rotating-hinge total knee prostheses can provide substantial improvement in WOMAC scores, pain and overall range of motion of the knee in the present case. We believe this salvage procedure should be reserved for patients with global ligamentous instability and neurological deficit or loss of muscle control. We suggest that in addition to the preoperative history review, a detailed neurological examination is critical, especially in osteoarthritic patients with symptoms or signs suggestive of root or cord compression. If any metastasis and /or spinal tumor are diagnosed before

surgery, it should be dealt with priority. Furthermore, this report may serve as a warning sign and/or red alert for the anaesthetic also highlighting the importance of thorough neurological examination of patients undergoing spinal anesthesia both in the pre-anesthetic check-up and postoperative examination in the recovery period. Otherwise the patient may developed complete paraplegia following spinal/epidural anesthesia possibly as a consequence of cord compression by a previously undiagnosed/or unrecognized spinal tumor.

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