External iliac artery thrombus masquerading as sciatic nerve palsy in anterior column fracture of the acetabulum

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ABSTRACT
We report a case of ischemic neuropathy of the sciatic nerve in a patient with an anterior column fracture of the acetabulum operated by ilioinguinal approach. It resulted from occlusion of the blood supply to the sciatic nerve. There were no signs of a vascular insult until ischemic changes ensued on the 6th postoperative day on the lateral part of great toe. The patient underwent crossover femoro-femoral bypass grafting and there was a complete reversal of the ischemic changes at 6 months. The sciatic nerve palsy continued to recover until the end of 1 year; by which time the only deficit was a Grade 4 power in the extensor hallucis longus (EHL) and the extensor digitorum longus (EDL). There was no further recovery at 2 years followup.

Key words: Anterior column fracture of acetabulum, external iliac artery thrombosis, ischemic neuropathy, sciatic nerve palsy
MeSH terms: Acetabulum, thrombosis, sciatic nerve diseases, sciatic neuropathy

INTRODUCTION
Earliest descriptions of an arterial thrombosis following acetabular fracture fixation through an ilioinguinal approach were given by Probe and Letournel. Formation of collateral circulation prevents the development of clinically evident thrombus and it remains a rare entity. Until the present date and to the best of our knowledge, there have been five reports of external iliac artery thrombus arising in the setting of an acetabular fracture in English literature, however none of them presented as a sciatic nerve palsy. Though ischemic neuropathy of the sciatic nerve has been reported previously in literature, it has never been reported as a complication of acetabular surgery through the anterior approach.

Case Report
A 61 year old male patient who suffered a seizure and sustained fracture of his right acetabulum and the right proximal humerus was referred to our hospital after 15 days. He was being managed conservatively at some peripheral setup. He was a known case of type II diabetes mellitus, hypertension and epilepsy. Neurovascular assessment was normal. Roentenograms (Anterioposterior pelvis, Judet views) and computed tomography (CT) with reconstructions revealed that he had a fracture of the anterior column of the acetabulum and the quadrilateral plate along with central dislocation of the femoral head [Figure 1a and b]. Haematological investigations revealed a low HDL/LDL ratio.

The fracture site was accessed through an ilioinguinal approach. We used a Schanz screw as a joystick to pull the femoral head laterally to reduce the fracture. Though we did use reduction clamps, we made sure that we do not produce prolonged compression with them. Vessels were retracted on pen-rose drain to facilitate adequate visualization of the fracture site. Anatomical reduction was achieved and the fracture was fixed using a 14 hole reconstruction plate (Synthes; Synthes India Pvt. Ltd., India) spanning from the internal iliac fossa to the superior pubic ramus [Figure 1c]. The artery was palpated during the procedure and prior to closure and had good volume and pulsations.

In the recovery room, the neurovascular status was checked again and was found to be normal. On the
second postoperative day, the patient developed foot drop. Examination revealed weakness in the muscles of the anterior compartment, attributable to the peroneal division of the sciatic nerve. On the 6th postoperative day, he started developing ischemic changes on the lateral part of his great toe [Figure 2]. Vascular surgery consultation was taken and CT angiography for lower extremity was performed, which revealed occlusion in the right external iliac artery [Figure 3a]. The patient was taken up for surgery; intraoperatively a large thrombus occluding the external iliac artery was identified. Cross over femoro-femoral bypass grafting with expanded polytetrafluoroethylene graft (GORE-TEX, WL Gore and Associates Inc., USA) was performed. The patient was put on low molecular weight heparin for 3 days and was switched over to warfarin which was titrated to an international normalized ratio of 2-3 at day 3. Postoperatively, an angiogram was obtained and the graft was found to be patent [Figure 3b].

The patient was given an ankle-foot orthosis and was allowed partial weight bearing with a walker at 6 weeks and full weight bearing at 10 weeks. Nerve conduction velocity and electromyography studies were performed at 1, 3 and 6 months after the onset of the palsy to assess recovery. Six months after the onset of palsy, the ischemic changes reversed completely and muscle strength in the right leg recovered to 4/5 in the peroneus tertius and 3/5 in the tibialis anterior [Figure 4a]. At 1 year the strength of the peronei and the tibialis anterior were 5/5 each and that of Extensor Hallucis Longus and Extensor Digitorum Longus was 4/5 each [Figure 4b]. At 2 years followup, the patient did not show any discernable recovery from that at 1 year.

**Discussion**

Mechanism of injury in most acetabular fractures is either a strong impact by femoral head onto the acetabulum or...
a direct lateral blow to the ilium. With such an impact the external iliac and proximal femoral arterial segments are subjected to traction. The medially coursing internal iliac and the inferior epigastric vessels can undergo tethering, resulting in intimal lesions and plaque rupture, which may give rise to thrombotic complications. With ilioinguinal approach these vascular structures are at further risk of thrombogenesis due to their inadvertent handling during the surgical procedure. The risk amplifies when this approach is used in patients who are high risk for thrombogenesis.

In the present case, the patient was a diabetic, epileptic, hypertensive and atherosclerotic male who was managed in traction for 15 days prior to undergoing surgery. He was on antiepileptic medication (phenytoin), which is known to cause osteoporosis. His lab investigations revealed hyperlipidemia and postoperative CT angiography showed sclerotic changes in many vessels. It is therefore conceivable that when he sustained an epileptic seizure, he suffered a traumatic intimal lesion and/or rupture of an atherosclerotic plaque, leading to activation of the coagulation cascade. The development of sciatic nerve palsy is attributable to dislodgement of an embolus from the thrombus which settled in vasa nervorum of the sciatic nerve. Concomitant small vessel vascular disease further predisposed the patient to the development of sciatic neuropathy. The development of ischemic changes on the lateral aspect of the great toe is attributed to occlusion by thrombus of an already compromised blood supply.

Although sudden onset sciatic nerve palsy is most commonly caused by trauma or iatrogenic injuries the possibility of a vascular insult as a cause of the neuropathy should not be ignored. In our case, the peroneal division of the sciatic nerve was affected which resulted in weakness of muscles of the anterior compartment of the leg and the patient developed foot drop. The same has been reported in literature by various authors. Comparison of cross section of both the divisions shows that the peroneal division has fewer and thicker nerve bundles than the tibial division, where the nerve bundles are greater in number and thinner, thus the vasa nervorum have to supply larger fibers in peroneal division as compared to the tibial division. Any ischemic insult to the vasa nervorum thus manifests itself more easily in the peroneal division.

CONCLUSION

Although rare, the possibility of a vascular disorder should be considered in a case of abrupt onset sciatic neuropathy, especially in patients who are at high risk, even though there may not be classical manifestations of a vascular insult. We urge caution in using the ilioinguinal approach in this subset of patients and recommend to use an alternative approach such as Stoppa’s approach, which allows adequate exposure of the anterior column and avoid dissection around the vessels or there inadvertent handling.

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