Eur Spine J (2010) 19 (Suppl 2):S203–S205
DOI 10.1007/s00586-010-1372-3

CASE REPORT

Left iliac artery injury during anterior lumbar spine surgery diagnosed by intraoperative neurophysiological monitoring

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Received: 4 May 2009 / Revised: 28 February 2010 / Accepted: 7 March 2010 / Published online: 17 April 2010

Abstract Serious vascular injury is a rare, but potentially devastating complication during anterior lumbar spinal surgery. The authors describe the first reported case where vascular injury was detected by multimodality neurophysiological monitoring during an L3–S1 anterior lumbar interbody fusion. The case demonstrates the need for multimodality monitoring and the combined use of somatosensory-evoked potentials and motor-evoked potentials.

Keywords Anterior lumbar fusion · Arterial injury · Motor-evoked potentials · Somatosensory-evoked potentials

Introduction

Serious vascular injuries during anterior approaches for lumbosacral pathology are an uncommon, but potentially devastating, complication. Here, we report the first use of both transcranial electrical motor-evoked potential (MEP) and somatosensory-evoked potential (SEP) monitoring to identify vascular compromise in the setting of anterior lumbar spinal surgery.

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Case

History

A 45-year-old female presented with an extensive history of lumbar back pain. She had previously undergone laminectomies and discectomies at L4–5 and L5–S1, followed by an L3–S1 posterior instrumented fusion with PEEK interbody spacers at L3–4 and L4–5 done 15 months prior to being seen in our clinic. None of these procedures provided long-term pain relief.

Diagnostics and plan

A CT myelogram demonstrated that no bony fusion had occurred. She did not have gross instability on flexion–extension plain radiographs. Because of the severity of her pain, an anterior lumbar approach was planned to remove the interbody spacers and perform anterior lumbar interbody fusions (ALIFs) from L3–S1 with femoral ring allografts (FRA).

Operative and post-operative course

With the assistance of general surgery, a left paramedian retroperitoneal approach was performed in the supine position. The aorta, left iliac artery and vein were all identified and mobilized. L3–S1 left segmental vessels were ligated. A Syn-frame was used for retraction utilizing multiple blades of varying widths and depths. During the time of retraction, the first occurrence of neurophysiological signal change occurred with both left abductor hallucis MEP and left tibial nerve SEP being absent. Release of traction led to immediate recovery of responses (Fig. 1). At L3–4, the non-fused PEEK cage was removed, discectomy completed and FRA inserted.

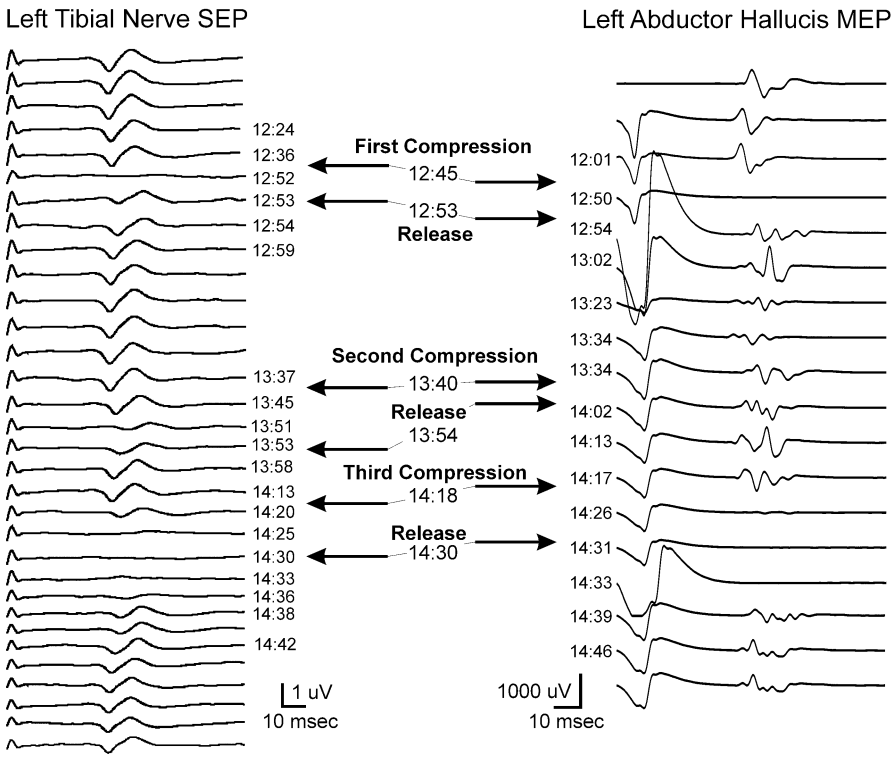

Fig. 1 Left lower extremity SEP and MEP signals. The three compression events are demonstrated, with both SEP and MEP signal amplitude decreases noted in the first and third event. During the second compression, only SEPs diminished

During this process, a second neurophysiological signal change occurred with a decreased amplitude and increased latency of the left tibial nerve SEP. Again, release of traction restored the signal (Fig. 1). In a similar fashion, the PEEK cage was removed and disc space prepared at L4–5. The FRA spacer was sized and inserted. At this point, a third neurophysiological change was noted in that there was a loss of both left abductor hallucis MEP and left tibial nerve SEP. No spontaneous EMG changes were noted at this time or during either of the two previous MEP and SEP changes (Fig. 1). Distal iliac artery pulsations were evaluated and felt to be diminished. On examination, the adventitia of the left iliac artery was found to have been trapped by the L4–5 spacer edge and pulled partially into the interspace when the FRA was inserted. Using a high speed burr, the artery was released while preserving the structural integrity of the FRA spacer. A 2-mm arterial laceration was primarily repaired with a 4-0 prolene suture. The MEP and SEP signals both recovered after the iliac artery was released.

We completed the L5–S1 discectomy and interbody fusion without additional signal loss. Post-operatively, the patient had intact distal pulses and ankle/brachial indices were normal 12 days post-operatively.

Discussion

The majority of arterial injuries related to ALIFs are thought to be secondary to prolonged retraction leading to stagnation of arterial flow and eventual thrombosis [1]. This is a rare example where the injury included both compression and an arterial laceration, which was noted intraoperatively with concomitant neurophysiologic changes, but fortunately repaired expeditiously without neurologic sequelae. Faciszewski reported only one arterial (aortic) laceration in 1,223 patients [2].

The incidence of any form of vascular injury during ALIF surgery ranges from 0 to 18.1% in literature [1]. Left common iliac venous lacerations are most common. Arterial injury is significantly more rare [3–6]. Brau reported an incidence of common iliac artery (CIA) injury of only 0.45% in their series of 1,315 anterior procedures [7]. In the 16 reported cases of CIA injury in literature, 15 were left sided and most were at the L4–5 level [1]. CIA injuries may be increasing as more mini-open ALIF procedures are done. Kulkarni reported an incidence of 5.2% of arterial injury in these operations [8]. Such a report highlights the utility of monitoring for such injuries.

The complications of a left iliac arterial injury include loss of limb or fatal rhabdomyolysis [1]. Left lower limb SaO_2 monitoring has been recommended as an additional preventive measure. In one study that examined SEPs and SaO_2 monitoring in anterior lumbar procedures, they found that 57% of patients had reductions in SEPs and SaO_2 secondary to the retraction required for L4–5 exposure; these changes resolved after removal of the retractors [9]. No reports presently report MEP changes in this situation.

In our case, three occurrences of neurophysiological signal changes occurred associated with retraction of the iliac artery. In two, both the MEP and the SEP signals became absent in parallel with a fall in the lower extremity SaO_2. In one occurrence, only the tibial nerve SEP changed, but was not eliminated, without any concurrent change in MEP. Though only speculative, this may have been due to less severe retraction of the iliac artery during the second event. It should be noted that upper extremity MEP signals were unchanged, indicating that anesthesia-related changes were not the source of signal loss. The absence of spontaneous EMG recordings supported a vascular versus neurologic cause. Both signals recovered with release of the artery and the patient suffered no long-term consequences.

As MEPs and SEPs continue to become the modalities of choice for electrophysiological monitoring during spine surgery, the importance of recognizing events that can alter or eliminate these electrophysiologic signals grows in importance. This report suggests that vascular compromise should be among the pathologies considered when a decrement in MEPs or SEPs occurs. Combined with evaluation of the EMG record, distal pulses and SaO_2 values of the relevant limb, neurophysiologic monitoring with MEPs and SEPs can rapidly determine the presence of a significant vascular insult and most importantly alert the surgeon to the need for intervention.

Conclusion

SaO_2 limb desaturation has been correlated to increased SEP signal changes in the setting of anterior lumbar retraction. Vascular injury is a rare, but highly morbid complication. The use of a variety of intraoperative monitoring tools can increase the likelihood of rapid diagnosis and treatment. This case report illustrates that both MEPs and SEPs may be beneficial in detecting neurological changes related to retraction in anterior lumbar spinal procedures.

Conflict of interest statement None of the authors has any potential conflict of interest.

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