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GRAND ROUNDS

Major neurological deficit following anterior cervical decompression and fusion: what is the next step?

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Abstract

Introduction Major neurological deficit following anterior cervical decompression and fusion (ACDF) is a rare event, with incidences of up to 0.2 % now reported. Post-operative MRI is mandatory to assess for ongoing compression of the cord. In the past, the deficit has often been attributed to oedema or over-zealous intra-operative manipulation of the cord. Reperfusion injury is a more recent concept. We describe a case of acute cervical disc prolapse with progressive neurology, and the difficult decision making one is faced with when the neurological deficit continues to deteriorate post ACDF.

Materials and methods A 30-year-old male was referred from the Emergency Department with acute left arm paraesthesia and left leg weakness. A cerebrovascular accident was ruled-out with a CT of the brain, and later an MRI of the cervical spine revealed a large C6/7 disc prolapse with significant compression of the spinal cord. A C6/7 ACDF was performed, but post-operatively the patient could no longer move his lower limbs. An urgent MRI was obtained which showed removal of the disc fragment, cord signal changes and the suggestion of ongoing cord compression. In part, this was due to his narrow cervical canal. The decision was made to proceed to posterior decompression and stabilisation, although cord reperfusion injury was one of the differential diagnoses considered at this stage.

Results Post-operatively the patient's neurology started to improve over the next 48 h. He was discharged from in-patient rehabilitation at 2 months post-surgery and by 3 months he had returned to work. Latest follow-up revealed normal function with only mild paraesthesia in the T1 dermatome of his left arm.

Conclusion The management of patients in whom a neurological deficit has increased post-operatively is difficult. Urgent MRI scan is mandatory to assess for epidural haematoma which may need further decompression. Cord reperfusion injury is a diagnosis of exclusion. The difficulty the clinician faces is in interpreting the MRI for 'acceptable' decompression, and therefore excluding the need for further surgery.

Keywords Cord compression · Anterior cervical decompression and fusion · Reperfusion injury · Surgical outcome

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Case presentation

A 30-year-old male was referred from the Emergency Department with sudden onset of left leg weakness and left

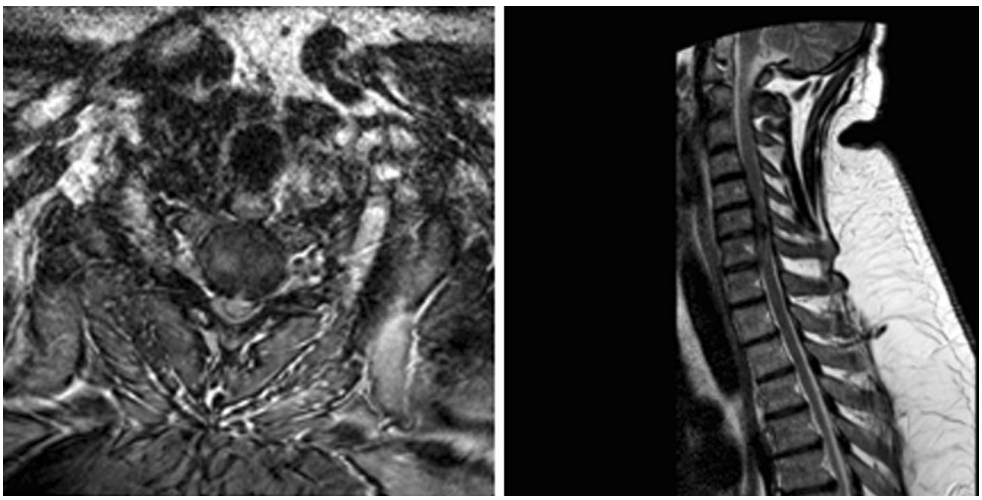

Fig. 1 Sagittal and axial MRI showing massive C6/7 disc prolapse

arm numbness. He was referred to our Spinal Unit after exclusion of a cerebrovascular accident as the cause of his symptoms.

On referral to the on-call team an urgent MRI of the whole spine was arranged the same day. At this stage the neurology in the lower limbs was: power in the left leg—3/5 in L2 and L3 myotomes and 3–4/5 in L4 and L5; whereas in S1 it was 4/5. On the right leg power was 5/5. There was altered sensation in the left leg to both pin prick and light touch from L1–S1. There was no neurological deficit in the upper limbs. The peri-rectal examination was normal consistent with an American Spinal Injury Association (ASIA) Impairment Scale (AIS) grade D [1]. There were also signs of cord compression in the form of hyper-reflexia in the legs and clonus.

The MRI scan showed a large disc prolapse at C6/7, which caused almost complete occlusion of the spinal canal (Fig. 1). The disc component appeared to be acute on the MRI, rather than a chronic calcified disc. Later the same night the patient reported no further deterioration in his neurology and re-examination was performed. Although he complained of numbness and tingling in the arms, neurological examination was normal. Power in the lower limbs was documented as the same as previous examinations and there was ongoing altered sensation in the left leg to touch and pin prick from L1–S1. Repeat perineal examination confirmed ongoing normal sensation and anal tone. A decision was therefore made to perform a C6/7 anterior cervical decompression and fusion (ACDF) the next morning, rather than operate overnight.

The next morning the patient was noted to have developed right leg symptoms (MRC grades: 3–4/5 in L2–S1) and on the left the weakness had progressed (MRC grades: 1–2/5 L2 and L3, 3–4/5 more distally). He also had

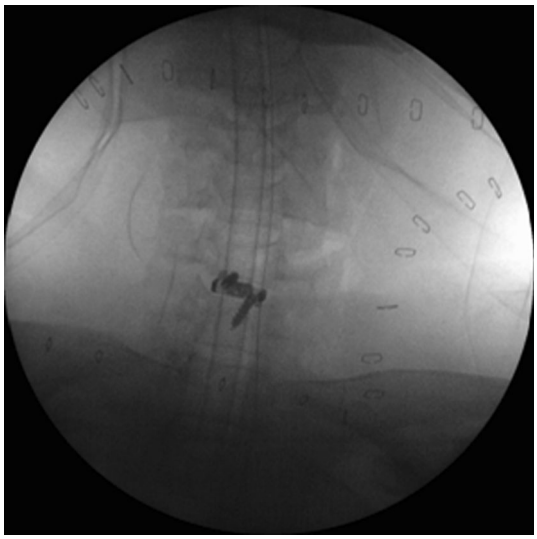

Fig. 2 Intra-operative AP radiograph of ACDF

generalised decreased sensation in the lower limbs. He had pins and needles in his finger tips, but otherwise still no neurological deficit in the upper limbs (AIS grade C). The deterioration in the neurology had only occurred just prior to the ward round (at 0745) with the regular consecutive neurological examinations performed overnight documenting no change in the neurology at 0600. We therefore proceeded to theatre first on the emergency list that morning for C6/7 ACDF (described below) (Fig. 2).

At the earliest opportunity, once extubated, we assessed his neurology. At this point he was found to have no power in either lower limb and clonus. However, there was improvement in the paraesthesia in his hands, there was normal neurology on examining the upper limbs, and there was improved sensation in the lower limbs. The patient was

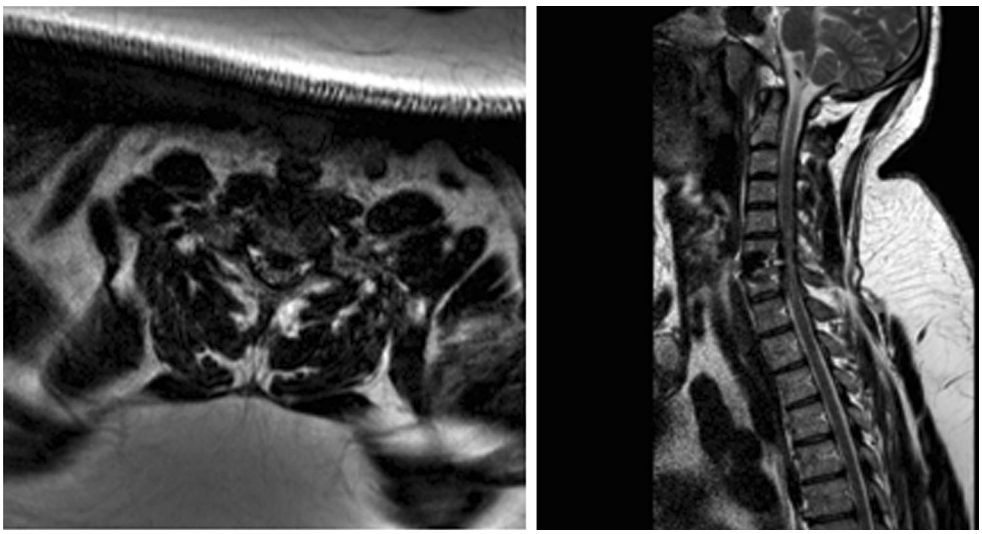

Fig. 3 Post-operative MRI (axial and sagittal) showing partial restoration of CSF flow around cord and cord signal changes, but concerns over ongoing compression

catheterised after his surgery and he could feel the catheter tug, but no formal perineal examination was documented in the notes to confirm sacral sparing.

An urgent MRI was arranged to assess for inadequate decompression of the disc, or epidural haematoma causing cord compression. He was given 16 mg oral dexamethasone. Given that he had preserved sensation bilaterally in his legs, our differential diagnosis included reperfusion cord injury; however, this is a diagnosis of exclusion at this stage.

The MRI revealed adequate removal of the disc prolapse, but concern that there may still be some compression of the cord due to a constitutionally narrow canal (Fig. 3). There was also increased signal in the cord on the T2-weighted images suggestive of cord injury. The case was discussed with a senior colleague and a neurology opinion was arranged given the incomplete picture of normal upper limb neurology, despite compression at the C6/7 level. Following a review by the Neurology team, the differential diagnosis was thought to be either: cord reperfusion injury, persistent compression, anterior cord infarct, or new onset of compression secondary to post-surgical oedema.

The consensus, based on the various discussions held between ourselves, the Neurologist and the Consultant Radiologist, was that there was ongoing compression on the spinal cord, although it was less compared to the pre-operative scan. The decision to do a posterior decompression and instrumentation was made based upon the above discussions, to give the cord the best chance to recover. Neurological examination following the second procedure revealed normal examination in the upper limbs, with sensation present in both lower limbs to touch and pin prick. Motor power was 0/5 throughout both legs, but perineal sensation and anal tone was normal (AIS grade C).

At this stage it may have been useful to perform a further MRI of the cervical spine to radiologically confirm circumferential decompression of the cord. However, in our department this is not routine practise, and given that we could confirm this on visual inspection during posterior decompression, we did not feel it clinically necessary at this stage.

Over the next 48 h he had some improvement in the power in both his legs, and again sensation was preserved in the lower limbs. Our feeling at this point was that he had an element of reperfusion injury combined with surgical oedema to the cord. He was referred to our local Spinal Cord injury unit for further rehabilitation and the steroids were reduced over 48 h on their advice to prevent infection.

Procedures

Anterior cervical decompression and fusion

A routine ACDF at C6/7 was performed without any significant complications (Fig. 2). The patient was positioned supine with the head kept in a neutral position. Inline immobilisation was performed at the time of induction and the positioning was done carefully by the operating surgeon. We did not put any sand bags under the shoulders to prevent hyperextension of the cervical spine and inadvertent compression of the cord during patient positioning. We also asked the anaesthetist to keep the

mean arterial pressure at 90 mmHg during the procedure to prevent any hypoperfusion of the cord.

A standard Smith-Robinson anterior approach was performed at the level of C6–7. The decompression was performed with the help of the microscope. The posterior longitudinal ligament was excised. We were careful during the surgery not to manipulate the cord and we used small Kerrison Rongeurs. Three disc fragments were taken out from the canal and the cord was seen to be floating upwards towards the vertebral bodies. The decompression was further checked with the knobbly feeler and there was no significant anterior compression at the end of the procedure.

A size 6 cage and size 16 screws were placed. Image intensifier was used to confirm satisfactory position of the implants and at the end of the procedure there was good haemostasis (Fig. 2).

Posterior decompression and stabilisation

Posterior decompression and instrumented stabilisation were performed in the standard way using a Mayfield clamp to align the head and neck. Inline intubation was maintained and blood pressure was again monitored closely, with the mean arterial blood pressure maintained at 90 mmHg.

A posterior midline approach was made and decompression of C5–7 performed with C5–7 lateral mass screws placed under image intensifier. The cord was found to be swollen and inflamed, and post decompression it was pulsatile.

Immediately post-surgery there was no improvement in the muscle power in his legs and he was transferred to a monitored bed where his blood pressure was actively maintained with intravenous fluids, and he was placed on flat bed rest.

Outcome/follow-up

The patient's neurology slowly improved over the next 48 h and by day two post-surgery the motor function in his legs had risen to MRC grade power 3/5 in L2–4 on the right, 4/5 distally, and a flicker of movement in L5 and S1 on the left (1/5). There was normal neurology in the upper limbs (AIS grade C). He was transferred to the Spinal Injuries rehabilitation unit at this stage.

Two months following the surgery he was discharged from the rehabilitation unit without a catheter and mobilising independently. Three months following surgery he had returned to work where he is an office-based engineer.

He was seen in the out-patient clinic 9 months post-surgery where his only complaint was of paraesthesia in the left T1 dermatome, but normal sensation and power. There was normal neurology in his lower limbs (AIS grade E).

Historical review of the condition, epidemiology, diagnosis, pathology, differential diagnosis

Post-operative major neurological deficits following anterior cervical spine surgery are well described in the literature, with the earliest papers now being nearly 50 years old [2–5]. This is now an uncommon event following ACDF with the incidence described as being up to 0.2 % [6–8]. We could only find one case report on the subject detailing the difficult decision making needed when faced with this problem in clinical practise [9].

We describe a case of acute cervical disc prolapse at C6/7 with rapidly deteriorating neurology pre-operatively. Interestingly our patient had very little neurology in his upper limbs, which is not what one would necessarily expect with a C6/7 disc prolapse. This, combined with the pre-operative progression, made interpreting his post-operative neurological findings difficult. The upper limbs were still spared, but there were signs of cord compression in the form of hyper-reflexia and ankle clonus. Was this simply progression of his pre-operative status, or was there ongoing compression of the cord?

The AIS is a neurological classification system developed by ASIA and is determined following examination in a standardised way. It has been revised since its first publication and updated by the 'International standards for neurological classification of spinal cord injury' (ISNCSCI) [1]. The classification is determined by the degree of residual motor and sensory function below a spinal cord lesion. It relies on a thorough systematic examination of the patient and there is a standardised form that can be placed in the clinical notes to help with this. On retrospective review of this case, documentation of examination findings was limited and not complete as per the ASIA/ISNCSCI guidelines after the first procedure.

Specifically in this case, following the first surgery, perineal sensation was not clearly documented, rather a reliance on the finding that the sensation of the catheter being tugged was present along with sensation in both legs. On review of the case this has been highlighted by the senior authors to the trainees in the department to improve standards of care. However, the following day, a complete dictation of all the events and the rationale behind each stage of the decision making was made, with a full typed record placed in the notes. This is a good standard of care to adopt during these difficult cases.

Differential diagnoses at this stage include incomplete decompression, either of the disc, or posterior longitudinal ligament (PLL); epidural haematoma; cord oedema from

manipulation intra-operatively, reperfusion injury or vascular compromise/ischaemia. An urgent MRI is required at this stage to exclude ongoing compression, but the difficulty is interpreting it in the pressured hospital environment when faced with limb paralysis.

Work has been done on assessing post-operative compression by the PLL and the importance of this has been refuted in a case series of 33 patients [10]. They found that on MRI performed following ACDF there was no evidence that the PLL was causing compression, even when it had not been excised. Intra-operatively we had taken down the PLL and so this was not the concern, it was more the constitutionally narrow canal. An MRI following the completion of both procedures would have been beneficial to confirm adequate decompression radiologically, and would have helped in our ongoing reasoning for the cause of this patient's symptoms.

With regards to complications, Cramer et al. [6] describe the incidence of epidural haematoma as up to 0.01 % over all spinal levels (causing 40 % of all major neurological deficits), although they did not stratify this further. Earlier studies had reported slightly higher rates of 0.9–1.3 % [4, 5]. Interestingly, Fountas et al. [7] did not find epidural haematoma as a cause for any of their post-operative cord injuries in 1,015 cases, hypothesising that the deteriorating neurology was due to surgical manipulation of the cord because high signal was identified on the post-operative MRI scan. If there is a large haematoma causing ongoing compression, then few would argue that urgent re-operation is necessary.

There has been a retrospective case series review of chronic compression [11] which found a transient cervical palsy incidence of 5.7 %. They again hypothesised that the cord was undergoing a reperfusion phenomenon, with all their neurological deficits recovering spontaneously. They cited age, anterior decompression, and ossification of the PLL as risk factors. Our case differs in that it was an acute disc prolapse in a young patient.

Interestingly, we could only find one case report on this subject [9] where a similar situation to ours was faced. Their post-operative MRI raised concern over ongoing bony compression at C5, and so they elected to revise their surgery through a revision anterior approach. However, they hypothesised that their patient's cord injury may have also been due to reperfusion syndrome, something they named 'White Cord Syndrome'. Unfortunately their patient did not obtain a full recovery.

Dumont et al. [12] have written a comprehensive overview of the pathophysiologic mechanisms of both acute and secondary cord injuries. They state that there is direct damage to the capillaries and venules from the initial trauma, which disrupts the semi-permeable membrane of the vascular walls. Ischaemia caused by direct pressure on the cord leads to a drop in pH which, combined with release of the cord compression, leads to a period of hyperaemia. With return of vascular flow to the cord, highly reactive oxygen and nitrogen metabolites formed during the period of ischaemia caused localised tissue damage [12–14].

There is loss of the normal local homeostatic mechanisms because of damage to the semi-permeable vessel walls and the astrocyte footprints of the blood–brain barrier [12, 15]. Further cell injury is therefore generated by the resulting electrolyte imbalances. There is also an accumulation of excitatory neurotransmitters, in particular glutamate, which are also cytotoxic, and this is referred to as 'excitotoxicity'. The resulting nerve cell damage from all these mechanisms could account for the deterioration in neurological status in those patients who do not have ongoing external compression of the cord.

Diagnosis of reperfusion injury is unfortunately still a diagnosis of exclusion, with external compression of the cord needing to be ruled-out first. At present there are no diagnostic tools which can conclusively state that a patient has reperfusion injury. It has been shown, in principle, that spinal cord pressures at the zone of injury could be measured [16]. However, further work is needed to define 'normal' pressure, and how altering the pressure can improve patient outcomes.

There has also been work investigating the use of MEPs and SSEPs (motor and somatosensory evoked potentials) in rabbits where cord ischaemia and reperfusion injury were simulated [17]. The authors did identify changes in the SSEPs and MEPs during the reperfusion phase, but unfortunately these changes were prone to significant variability. This limits their usefulness in clinical practise at this stage, and further work is needed to improve their sensitivity and specificity.

Finally, the measurement of microvascular flow and the presence of free radicals in the microvascular circulation at the level of injury may be a diagnostic tool, but samples would be technically difficult to collect, as would the measurement of flow. As yet, we are unaware of any described techniques to aid in the diagnosis of cord reperfusion injury.

Rationale for treatment and evidence-based literature

We took the view that we should proceed to further posterior decompression in order to give this patient the best possible outcome. However, there are some who would also say that surgery was not indicated given the MRI findings and that a watch-and-wait course of action should be taken. The difficulty is in assessing what is 'satisfactory decompression of the cord'; there are no clear definitions in

the literature. Following the anterior surgery, based on the MRI findings of there being no clear circumferential cerebrospinal fluid column around the cord, we felt there may be ongoing compression and therefore elected to proceed to posterior surgery. During posterior laminectomy, it is easier to visualise the cord and assess for adequate decompression at the time of surgery. However, these are our own arbitrary evaluations. A post-operative MRI would have been useful at this stage.

Further surgery is also supported by Flynn who in 1982 [4] described major neurological deficit occurring in 53 out of 311 patients (17 %) immediately on waking from the general anaesthetic. Of those that they explored, 57 % improved; whereas in those they did not explore, only 28 % improved. Unfortunately they do not stratify further the reason for exploration or intra-operative findings because it was a questionnaire-based study, although their results did include 'no aetiology and intra-operative trauma'. However, these figures would suggest re-operation is justified.

The use of steroids is a controversial subject following both acute spinal cord injury and reperfusion injury. Dumont et al. [18] summarise the evidence for their use very well in their follow-up article on the treatment of reperfusion injury. There are several studies that show conflicting results, despite the potential benefits from their anti-inflammatory properties. Unfortunately, no study has been able to clearly show a benefit of their use with a standardised regimen. Concerns have also been raised due to the possible side effects of gastric ulcers and concurrent infection. Similar findings have also been described for the use of steroids following cord reperfusion injury after thoraco-abdominal aneurysm repair, with no clear improvement in outcome being seen [19].

We initially started dexamethasone based on the limited evidence that suggests that steroids can be beneficial. There are some regions internationally who still advocate their use. However, when referring to our regional spinal cord injury centre, we were advised to stop these because their overall feeling was that the evidence is limited, and the risk of severe complications outweighs the possible benefits that steroids may bring.

Ultimately, these are the dilemmas that we as spinal surgeons face when appraising post-operative neurological status in these difficult situations. Thorough examination of the patient with clear documentation in the notes is mandatory at every stage of the decision making. Spinal cord reperfusion injury is ultimately a diagnosis of exclusion, with no single diagnostic test. As clinicians we are required to ensure that the benefits of any intervention performed outweigh the risk of further complications.

Conflict of interest None.

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