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Commentary

Reperfusion Injury (RPI)/White Cord Syndrome (WCS) Due to Cervical Spine Surgery: A Diagnosis of Exclusion

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ABSTRACT

Background: Following acute cervical spinal cord decompression, a subset of patients may develop acute postoperative paralysis due to Reperfusion Injury (RPI)/White Cord Syndrome (WCS). Pathophysiologically, this occurs due to the immediate restoration of normal blood flow to previously markedly compressed, and under-perfused/ischemic cord tissues. On emergent postoperative MR scans, the classical findings for RPI/WCS include new or expanded, and focal or diffuse intramedullary hyperintense cord signals consistent with edema/ischemia, swelling, and/or intrinsic hematoma. To confirm RPI/WCS, MR studies must exclude extrinsic cord pathology (e.g. extramedullary hematomas, new/residual compressive disease, new graft/vertebral fracture etc.) that may warrant additional cervical surgery to avoid permanent neurological sequelae.

Methods: In the English literature (i.e. excluding 2 Japanese studies), 9 patients were identified with postoperative RPI/WCS following cervical surgical procedures. For 7 patients, new acute postoperative neurological deficits were appropriately attributed to MR-documented RPI/WCS syndromes (i.e. hyperintense cord signals). However, for 2 patients who neurologically worsened, MR studies demonstrated residual extrinsic disease (e.g. stenosis and OPLL) warranting additional surgery; therefore, these 2 patients did not meet the criteria for RPI/WCS.

Results: The diagnosis of RPI/WCS is one of exclusion. It is critical to rule out residual extrinsic cord compression where secondary surgery may improve/resolve neurological deficits.

Conclusions: Patients with acute postoperative neurological deficits following cervical spine surgery must undergo MR studies to rule out extrinsic cord pathology before being diagnosed with RPI/WCS. Notably, 2 of the 9 cases of RPI/WCS reported in the literature required additional surgery to address stenosis and OPLL, and therefore, did not have the RPI/WCS syndromes.

Key words: Reperfusion Injury (RPI), White Cord Syndrome (WCS), Diagnosis of Exclusion, Cervical Spine Surgery, Myelopathy, Quadriplegia Cord Injury, Residual Cord Compression

INTRODUCTION

Definition, Pathophysiology, and Etiology of Reperfusion Injury (RPI)/White Cord Syndrome (WCS) Following Cervical Spine Surgery

RPI/WCS is typically defined as an acute spinal cord injury that follows cervical spine surgery where postoperative MR studies document intrinsic cord edema/ischemia, swelling, and/or hemorrhage in the absence of significant new/residual extrinsic pathology. Pathophysiologically, this occurs because spinal cord decompression due to cervical surgery acutely restores normal blood flow

to previously under-perfused/ischemic cervical spinal cord tissues.[1-8] In theory, such acute cord decompression, and re-expansion disrupts the blood-spine barrier allowing for "rush-in reperfusion", a "…triggered cascade of reperfusion injuries…", and potentially, "…oxygen-derived free radical damage" [Table 1].[2] Other etiologies include; small artery or anterior spinal artery occlusion, "…microthrombi, and altered perfusion due to internal recoil of the spinal architecture following decompression", "… direct trauma from blood flow itself or by the oxygen free radicals…", or "…lipid peroxidation of the neuronal membrane…as a main cause in the secondary injury-induced degenerative cascade."[1-5,7,8] In a rat model, RPI/WCS resulted in the "grey matter …(being)..dislodged when acute cord compression was "released".[6]

RPI/WCS A Diagnosis of Exclusion: Classical Postoperative MR Findings for RPI/WCS

RPI/WCS is a diagnosis of exclusion following multiple cervical surgical procedures; anterior cervical discectomy/fusion (ACDF), anterior corpectomy/fusion (ACF), laminectomy with/without fusion, laminoplasty, or other cervical procedures including those for tumor resection. Postoperative MR studies best document the classical intrinsic cord changes diagnostic for RPI/WCS.[1,2] Immediate (30 minutes up to 4-6 postoperative hours) postoperative MR studies typically show acute focal/diffuse hyperintense T2 weighted intramedullary cord edema/swelling in the absence of extrinsic pathology [Table 1].[1-8] Further, MR's may rule out (i.e. exclude) RPI/WCS if they document new postoperative surgical disease (e.g. extramedullary hematoma, new/residual extrinsic cord compression (e.g. residual disc, stenosis, or OPLL (ossification of the posterior longitudinal ligament)), direct intraoperative cord trauma/mechanical insult (i.e. "overzealous manipulation"), postoperative graft/vertebral fracture/displacement, other factors) that warrants additional treatment/surgery.[1,8]

Frequency of Reperfusion Injury (RPI)/White Cord Syndrome (WCS)

Seichi et al. (2014) Frequency of RPI/WCS Following Laminoplasty

In 2004, Seichi *et al*. evaluated the frequency of RPI/WCS in 114 patients with severe cervical myelopathy undergoing laminoplasty.[5] They prospectively compared the preoperative vs. 3 week postoperative MR studies looking for new or increased intramedullary hyperintense cord signals indicative of ischemia/edema; they further correlated these findings with new postoperative neurological deficits.[5] "Seven patients (6.1%) showed postoperative abnormal expansion of the T2 high-signal intensity area (*in the cord),* and 3 of the 7 were asymptomatic"; the other 4 patients (3.5%) with symptoms had varying degrees of new postoperative myelopathy, and/or motor cord/root deficits with appropriate MR findings for RPI/WCS.[5] However, methodological limitations in this study make it inappropriate to generalize the high 3.5% frequency of RPI/WACS to all cervical surgeries. Limitations of this study included; (1) it involved a non-randomized single cohort of patients, (2) there was no control group, (3) it involved just a small number of patients, (4) patients underwent a single cervical operation (laminoplasty), and (5) these operations were performd by just a small group of surgeons.

Frequency of RPI/WCS Best Identified by Seven Cases in the Literature

Several authors defined the RPI/WCS syndrome as "rare", involving only a small number of cases found in the English literature (i.e. omitting Khan *et al*. 1973 case, and 2 additional Japanese studies) that met the MR-documented inclusion criteria) [Table1].[1-4,6-8] In 2013, Chin *et al*. noted that;"… paralysis (*with RPI/WCS*) is extraordinarily rare".[2] Khan *et al*. (2017) later noted: " Non-traumatic cervical disc herniation is rarely the cause of acute quadriparesis, with the earliest case reported in literature as recent as 1973. Since then, a further nine cases have been added in the English literature, and two cases have been described in the Japanese language."[4] Wiginton *et al*. in their 2019 study also acknowledged that RPI/WCS was a "rare complication of cervical spine decompression…", and further observed it had been reported in just 3 prior cases.[7] Critically, however, the two individual cases respectively reported by Bailey *et al*. and Chin *et al*. did not actually have RPI/WCS. In fact, their postoperative MR scans demonstrated new extrinsic spinal lesions attributed to residual stenosis (one case), and OPLL (one cases); both warranted additional cervical surgery [Table 1].[1,2] In short, very few valid cases of RPI/WCS have been accurately reported in the English spinal literature. Therefore, most postoperative patients with new neurological deficits following cervical spine surgery more than likely have other extrinsic pathologies responsible for their new findings [Table 1].[1-8]

Treatment Options for RPI/WCS

Conservative Management

Conservative treatment options for patients with RPI/WCS include; admission to intensive care units for neurological monitoring, elevation of mean arterial pressures (MAP) (i.e. averaging 85-95 mm HG) to perfuse a compromised/ischemic cord, and the administration of steroids (varying regimens from Decadron to High Dose/Trauma Protocol of Methylprednisolone).

Select Additional Surgical Decompression for RPI/WCS

Zhang *et al*. offered several explanations as to why RPI/WCS injuries occurred in their 3 OPLL patients [Table 1].[8] First,

Table 1: Postoperative reperfusion injury (RPI)/white cord syndrome (WCS) causing acute deficits following cervical spine surgery.

Author year	Case report or series pathology	Surgery postop complication treatment	Postop complication postop MR treatment	Follow-up treatment	Treatment and outcomes
Chin *et al.*[2] 2013	59-yo M C4-C5, C5-C6 ACDF 7 mos severe myelopathy MR Large C5C6 disk-marked cord compression/myelomalacia	Intraoperative SEP and MEP C4-C6 Graft - PEEK Cages-C5-C6 graft placed-sudden decreased MEP graft C4-C5 placed-MEP lost grafts replaced, plated	Hydrocortisone 100 mg IV during surgery-HDS given postop C6 level quadriplegia postop MR/CT residual C5 bony compression	NOT RPI/WCS* Due to residual stenosis 2nd surgery C5 corpectomy C4-C6 graft not RPI/WCS*	Postop 2nd surgery severe residual quadriparesis rehabilitation mild improvement 16 months later
Zhang *et al.*[8] 2013	Three cases ACF postop paralysis 2M; 1F: Ages 41–61 ACF: C5, C5/6, and C6/C7	Postop all 3 quadriplegic postop MR (3) no clot-cord edema two complete deficits resolved 30 min-4 h-HDS	3rd patient -24 h HDS residual deficit, incomplete 2nd surgery-laminoplasty C3-C6 after 24 h improved in 1 week	Diagnosis; RPI/WCS acute cord edema	Conclusion: Early diagnosis HDS surgical decompression if needed-laminoplasty
Bayley *et al.*[1] 2015	C67 ACDF: 30 yo M; Preop MR large disk, severe compression	Postop paraplegic MR C6/C7 residual stenosis- cord compression	2nd Surgery: Posterior decompression/fusion	Etiology deficit: NOT RPI/WCS* due to residual stenosis	At 3 mos return to work -residual T1 sensory changes on left
Khan *et al.*[4] 2017	C56 ACDF 36 yo M 2–3 h incomplete quadriparesis Frankel Grade C	Preop MR: High cord signal C5-C6-Significant cord compression from acute disc given HDS	Surgery in 8 h of postop intact until 3 days later acute deterioration incomplete quadriplegia	Postop MR High T1/T2 cord signals-edema patchy hemorrhage no extrinsic compression	Diagnosis: Consistent with RPI/WCS
Giammalva *et al.*[3] 2017	64-year-old M ACDF C34-C4/C5-C6 preop mild grip weakness	Preoperative MR T2 high signal at C34 and severe cord compression due to discs C34/C56	No intraoperative complications postoperative severe quadriparesis	Postop MR Increased hyperintensity in cord T2 study at C56 level	Treatment HDS partial Improvement
Vinodh *et al.*[6] 2018	C3 intradural/extradural metastatic ductal carcinoma-paraparesis for 1 month	Preoperative MR; focal T2 high signal in cord at C3-C4 with stenosis laminectomy C2-5, posterior fusion C1, C2-C5, C6	Postop quadriplegic on ventilator postop MR: New T2 hyperintensity/edema from C3 to brain stem	MR findings - no residual extrinsic cord compression/consistent with RPI/WCS given HDS	6 Week postop MR: High T2 intramedullary cord signal C3-brain stem unchanged
Wiginton *et al.*[7] 2019	41 yo M progressive myelopathy	MR severe cord compression C1 high cord signal	C1 laminectomy, total loss SEP/MEP: Resolved at closing quadriplegic postop no technical cause	Immediate postop MR - T2 increased signal in cord at C1	Treatment: >>MAP 95 mm Hg HDS-improved in h/normal mos later

CS: Cervical stenosis, CSM: Cervical spondylotic myelopathy, Post: Posterior, Dec: Decompression, Lam: Laminectomy, M: Male, F: Female, Postop: Postoperative, ASA: Anterior spinal artery, Comp: Complication, WCS: White cord syndrome, F: Fusion, HP: Hemiparesis, ARI: Acute reperfusion injury, CCC: Chronic cord compression, ACDF: Anterior cervical decompression fusion, RIPC: Remote ischemia preconditioning, SCI: Spinal cord injury, AD: Adverse events, Preop: Preoperative, RPI: Reperfusion injury, ACF: Anterior corpectomy/fusion, UE: Upper extremities, BLE: Bilateral lower extremities, R: Right, L: Left, yo: Year old, Quad: Quadriparetic/quadriplegic not RPI/WCS*: Not reperfusion injury or white cord syndrome, SEP: Somatosensory evoked potentials, MEP: Motor evoked potentials, MAP: Mean arterial blood pressure, mos: Months, yrs: Years, wk: Weeks, HDS: High dose steroids, h: Hour

they thought that; "…damage to the spinal cord (*occurred)* during removal of extensive adhesions anterior to the dura...", that they largely attributed to OPLL. Two further hypotheses were that; "… the space provided by (the) corpectomy is relatively small compared to that provided by posterior decompression", and therefore, "....after mesh grafting the once opened spinal canal was again closed".[8]

In one of Zhang *et al*.'s patients, following a C4-C6 anterior corpectomy/fusion, the patient was immediately quadriplegic.[8] The patient was diagnosed as having the RPI/WCS based upon the emergent postoperative MR findings of: "…spinal cord edema, and no hematoma formation" [Case #7, Table 1]. When the patient's deficit failed to resolve after 24 hours of high dose steroids, a C3-C6 laminoplasty was performed; at surgery, they found an extremely swollen cord which they determined was now adequately "decompressed".[8]

Case Summaries

Here, we reviewed each of the 7 patients undergoing 5 anterior and 2 posterior procedures, who developed immediate postoperative MR findings consistent with RPI/WCS [Table 1].[3-7] However, postoperative deficits for the remaining 2 patients were attributed to MR-documented extrinsic disease (respectively stenosis, and OPLL) that required additional surgery; these were, therefore, not attributable to RPI/WCS [Table 1].[1,2]

Risk of Reperfusion Injury After Posterior Cervical Decompression

Following posterior cervical decompressive surgery, the following two patients developed acute postoperative RPI/WCS.[6,7]

Case #1

In Vinodh *et al*. (2018), a 51-year-old female presented with 1-month of increasing paraparesis, and urinary dysfunction [Table 1].[6] Her neurological deficit was attributed to metastatic ductal carcinoma stemming from the C3 vertebral body with both intradural-extramedullary, and extradural extension. On the T2 - weighted MR scan, there was hyperintensity within the cord. On the T1 contrast study, the intradural-extramedullary, and extradural tumor homogeneously enhanced. She underwent a C2 to C5 laminectomy for excision of a well-defined tumor mass with an instrumented C1/C2 - C5/C6 posterior fusion. Acutely postoperatively, she exhibited a complete C3 motor/sensory quadriplegia accompanied by respiratory distress requiring intubation. The emergent postoperative CT documented a normal fusion construct, but the MR confirmed an RPI/WCS characterized by increased "…cord edema extending to the lower brain stem" in the absence of new extrinsic pathology. Despite high dose steroids for 3 weeks, she exhibited no neurological recovery. Further, the 6-week postoperative MR demonstrated the same degree of C3-brain stem /cord edema as seen on the initial postoperative examination [Table 1].[6]

Case #2

A 41-year-old male in the Wiginton *et al*. study (2019) presented with increasing myelopathy (e.g. 4/5 motor function in the upper and lower extremities accompanied by bilateraal Hoffman's signs, and diffuse hyperreflexia with clonus) [Table 1].[7] The T2 weighted MR demonstrated severe, chronic C1 stenosis/cord compression (i.e. more pronounced posteriorly than anteriorly), and a high signal within the C1 cord consistent with chronic myelomalacia. Suddenly intraoperatively, while performing a full C1 and partial C2 laminectomy, the somatosensory evoked (SEP), and motor evoked potentials (MEP) dropped out. Immediately, the mean arterial pressure (MAP) was raised from 85 to 95 mm HG, and intraoperative fluoroscopy was performed to rule out a structural lesion/reason for the acute deterioration; there was, however, no "subluxation or malalignment". Although the monitoring changes recovered to baseline prior to closure, the patient initially awakened with a near-complete 1/5 motor quadriplegia; however, within minutes, it began to resolve. The STAT postoperative T2-weighted MR revealed no residual extrinsic cord compression, but showed a new increase in the size of the preoperative chronic intramedullary hyperintensity at the C1 level. They determined this represented just mild postoperative expansion of the focus of myelomalacia seen on the preoperative study; it was simply being better visualized following the decompression, and did not reflect a new cord injury. For five days, the patient's MAPs were maintained at > 90 mm Hg, and he was given steroids (Dexamethasone 10 mg IV q6h). Within several postoperative weeks, he fully recovered neurological function.

Risk of RPI/WCS After Anterior Cervical Surgery

2 Cases of RPI/WCS After Anterior Cervical Diskectomuy and Fusion (ACDF)

In two cases, following ACDF, patients developed RPI/WCS confirmed on postoperative MR studies.

Case #3

In 2017, Kahn *et al*. evaluated a 36-year-old male who presented with 2 weeks of vague cervicalgia, and the acute onset of an incomplete quadriparesis over just 2-3 hours (e.g. motor deficit 2-3/5 in the upper/lower extremities with incomplete sensory dysfunction: ASIA/Frankel Grade C) [Table 1].[4] The preoperative cervical MR demonstrated a large C5-C6 ventral-central disc herniation with marked stenosis/cord compression. There was an accompanying "subtle" increased T2 intrinsic cord signal seen just above the C5-C6 level consistent with cord edema.

The patient was placed on the high-dose steroid trauma protocol of Methylprednisolone (30 mg/kg bolus followed by 5.4 mg/kg/hour over 24 hours), and brought to surgery 8 hours following the onset of symptoms. A C5-C6 ACDF was routinely performed using an iliac autograft with a plate. Notably, at surgery, there was evidence of prior trauma. Immediately postoperatively, the patient's motor status improved to the 3/5 level in the upper and lower extremities. However, 3 days later, the patient acutely became fully quadriplegic, requiring immediate ventilatory support. Although the cervical X-rays showed the fusion construct was intact, the STAT MR revealed an intramedullary hemorrhagic cord infarction with edema maximal at the C5-C6 level (hyperintense on T1 and T2 weighted signals) without extrinsic cord compresion. The contrast study showed "patchy enhancement with luxury perfusion" in the cord. The patient's deterioration was attributed to a RPI/WCS injury characterized as a; "…delayed ischemic/reperfusion injury,…(*due to)* restoration of blood flow through the anterior spinal artery". Within a year, the patient's motor function improved to the 4-/5 level in all four extremities. Although he continued to have a relative C4 pin level, he regained vibratory/position appreciation, and no longer required a ventilator.

Case # 4

A 64-year-old male in Giammalva *et al*. 2017 study underwent a C3-C4 and C5-6 ACDF for severe cervical cord compromise [Table 1].[3] On the preoperative MR there were significant disc herniations at both levels, but only a high intrinsic singal was seen within the cord at C3-C4. Although the surgery went smoothly, the patient awakened with a new severe motor quadriparesis with sensensory preservation. The immediate postoperative MR documented a new "increased hyperintensity" in the cord at the C5-C6 level without any extrinsic lesion compressing the cord. Despite the administration of high-dose steroids, and significant rehabilitative efforts, the patient demonstrated only partial long-term recovery of motor funtion.

2 Cases of ACDF With Residual Extrinsic Cord Compression Requiring Additional Surgery; These Patients Did Not Have the RPI/WCS Syndrome

In two case reports, the diagnosis of RPI/WCS should have been excluded; both patients' new postoperative deficits following ACDF were due to MR-documented residual extrinsic cord compression requiring secondary surgery (e.g. stenosis and OPLL respectively) [Table 1].[1,2]

Case # 5

Bayley *et al*. (2015) performed a C6-C7 ACDF in a 30-year-old male who presented with left upper extremity paresthesias accompanied by left leg weakness; the MR demonstrated a large C6-C7 disc herniation [Table 1].[1] The surgeons characterized the performance of the C6-C7 ACDF as "routine". However, immediately postoperatively, the patient was quadriplegic. Here, although the postoperative MR showed diffuse/marked intramedullary hyperintensity/cord swelling at the C6-C7 level, there was also the "suggestion"of ongoing cord compression" (e.g. stenosis with incomplete decompression). Notably, following secondary surgery which included a laminectomy for decompressionn with fusion, the patient started started to improve within 48 hours; 3 months later, he regained normal function except for mild residual paresthesias in the left T1 dermatomal distribution. In summary, this patient had residual stenosis following the initial C6-C7 ACDF warranting an additional posterior decompression, and did not have the RPI/WCS syndrome.

Case # 6

A 59-year-old male in the Chin *et al*. (2013) study underwent a two-level C4-C5/C5-C6 ACDF [Table 1].[2] When the C5-C6 graft was placed, the motor evoked potentials (MEP) decreased, and did not recover even when the graft was removed. When the second interbody graft was placed at the C4-C5 level, MEP were now completely lost. This prompted removal of the second graft. When the wake-up test showed he was only able to move the arms, both interbody grafts were quickly replaced along with a plate so that he could undergo postoperative testing. The patient awakened with an incomplete quadriplegia (e.g. full motor deficit with sensory preservation). The postoperative MR and CT studies; "...raised concern for residual bony compression mostly behind the C5 body, and the edges of C4 and C6."[2] When Epstein reviewed these studies, there appeared to be marked residual/unresected OPLL that contributed to severe on-going anterior cord compression with ventral tethering-stretching of the cord. Notably, this "extrinsic compression" ruled out the RPI/WCS syndrome. Although the patient underwent an urgent/emergent secondary complete C5 corpectomy with a C4-C6 strut fusion, he failed to improve over the next 16 postoperative months.

Reperfusion Injury After Anterior Cervical Corpectomy and Fusion (ACCF)

Three patients in Zhang *et al*. series, undergoing multilevel anterior corpectomy/fusions for OPLL, exhibited postoperative RPI/WCS [Table 1].[8] Interestingly, one of the three patients underwent a secondary laminoplasty to "decompress" a diffusely swollen cord due to the RPI/WCS (e.g. without other new/residual extrinsic pathology).

Summary of 3 OPLL Cases with RPI/WCS with Specific Presentation of Case #7 Who Required an Additional Posterior Decompression

Zhang *et al*. (2013) reported 3 patients with OPLL, ranging from 41-61 years of age; all 3 developed acute postoperative

quadriplegia (e.g. within 30 minutes-4 hours) following anterior cervical corpectomy and fusion procedures (ACCF: C(5), C(5-6), and C(6-7)) [Table 1].[8] In all cases, the surgeons noted that operative dissection required extensive release of adhesions between the dura, OPLL, and the herniated discs. As soon as the new postoperative deficits were recognized, all 3 patients were given high-dose methylprednisolone for 24 hours. They all also underwent STAT MRI scans that demonstrated diffuse cord edema consistent with acute RPI/WCS syndromes without focal residual extrinsic cord lesions. For 2 patients undergoing ACCF at the C5-C6 and C6-C7 levels, their postoperative deficits appeared within 30-40 minutes, but resolved spontaneously within 2 hours. The MR scan in the first patient had shown no extrinsic lesiion; rather it demonstrated a high signal in the cord with swelling. The MR for the second patient documented a small ventral hematoma (i.e. insufficient to be considered surgical) at C5 and; "…obvious spinal cord edema with no high intensity (*signal*) in the spinal cord".[8] However, the third patient who had undergone a C5 corpectomy (C4-C6 ACCF), despite the administration of steroids for 24 hours, continued to exhibit an incomplete quadriparesis. As her MRI showed; "…spinal cord edema and no hematoma formation…", the surgeons decided to perform a C3-C6 laminoplasty to provide additional posterior "decompression". At surgery, they encountered an extremely "swollen" cord; the second postoperative MR demonstrated a decrease in both the intrinsic cord edema, and high intramedullary cord singal. She recovered full neurological function one week later.

CONCLUSION

Pathophysiologically, the RPI/WCS occurs following acute cervical spinal cord decompression when normal blood flow is restored to previously under-perfused/ischemic spinal cord tissues. To establish the diagnosis of RPI/WCS, postoperative MR studies must demonstrate new or expanded, focal or diffuse hyperintense cord signals on T2 weighted images indicative of cord edema/swelling with occasional additional intramedullary hemorrhages.

However, RPI/WCS is a diagnosis of exclusion. Postoperative MR studies must rule out new/residual extrinsic causes of cord compression; (e.g. extramedullary hematomas, residual/new disc/stenosis/OPLL/other, graft and/or vertebral fracture/dislocation, direct cord injury due to overzealous intraoperative dissection, amongst other factors) to determine if further surgery is warranted.[1-8] In our review of the English literature, and based upon Epstein's review of postoperative MR studies, RPI/WCS was accurately diagnosed in 7 of 9 cases.[3,4,6-8] Notably, analysis of 2 cases originally identifed as having RPI/WCS were excluded as they exhibited new/residual postoperative "extrinsic disease" (e.g. stenosis and OPLL respectively) that the authors' themselves determined warranted (i.e. and performed) additional surgery.[1-2] Although there are likely a few other studies in the literature that we failed to identify, the main point of this commentary still holds; there are very few cases of RPI/WCS reported in the literature.[3,4,6-8] In summary, the diagnosis of RPI/WCS is one of exclusion, and should not be invoked until residual extrinsic cord compression, and the need for secondary surgery have been definitively ruled out.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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Conflicts of interest

There are no conflicts of interest.

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