Acute non traumatic cervical mielopathy in an adult patient: a challenging diagnosis. Case report.

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Research Article

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Abstract

Introduction

Acute cervical myelopathy is a challenging diagnosis. Spinal cord infarction is generally caused by aortic pathologies. In absence of a definite diagnosis, fibrocartilagineous embolism can be a cause of spinal cord ischemia.

Case presentation

Authors here presented a case report of a 42 years old female patient, suffering acute myelopathy in a stenotic cervical canal by anterior osteophytes.

She was admitted to our emergency department with chest pain and tetraparesis, manifesting with two acute episodes within 24 hours of each other, the second worse than the first. Traumatic, inflammatory, ischemic, infectious and compressive causes were excluded. Both neuroprotection therapies (administration of glucocorticoids, maintenance of mean arterial pressure) and surgical decompression of stenotic cervical canal were adopted. Follow-up was characterized by neurological improvement.

Conclusions

To our knowledge, the case here reported is the first of a suspected FCE in a stenotic CC surgically decompressed. FCE is generally an exclusive diagnosis; definite diagnosis could be only provided by spinal cord biopsy examination. Cervical canal decompression, by removing anterior osteophytes, probably contributed to SC adequate vascular perfusion through the anterior spinal artery and prevented secondary damage from medullary swelling.

Introduction

SCI is a very rare clinical occurrence and causes important permanent neurological disabilities[1, 12, 13]. Despite clinical efforts in prevention and treatment, clinical outcome is disabling[1]. Therapy is based on neuroprotection principles[2]. As well as in medullary contusion, cervical canal decompression surgery can play an important role. We wanted to apply these principles to a suspected case of FCE in a 42-year-old female patient suffering from a severe acute form of myelopathy.

Case presentation

A 42-year-old patient was admitted to emergency department for generalized malaise, chest attack pain and numb limbs. Symptoms began acutely without physical exertion and trauma. An aortic angio-CT scan did not document vascular lesions, in particular no dissecting lesions were documented. Brain and cervical spine CT scans, as well as cerebral angio-CT study, were performed but they did not reveal any disease. In the event of a cardiovascular problem, antiplatelet precautionary therapy was set up. Cervical spine MRI scan documented a stenotic canal, in particular C3-C4 C4-C5 C5-C6 anterior osteophytes, and

no evidence of myelopathy (Fig. 1A and 1B). Patient was admitted to Neurological Operative Unit and, 12 hours later onset, presented a complete *restitutio ad integrum* of the beginning symptoms (ASIA E). Rachicentesis was performed with CSF examination, in order to exclude inflammatory spinal cord injury; the only alteration was a mild protidorrachia. Infectious and oligoclonal bands CSF laboratory tests were negative.

Both medical history and following examinations documented absence of autoimmune, vasculitis and infectious diseases.

24 hours later clinical onset, patient began to present a further clinical worsening with tetraparesis: severe upper limbs diparesis in the distal segments, right lower limb plegia, pain in the four limbs, urinary retention (ASIA C). Sudden and persistent chest pain also appeared. A cervical spine MRI scan documented radiological modification: intra-medullary alteration signal of the posterior C4 cords evident in the sequences in T2 (Fig. 2A, 2B, 2C), suggesting ischemia. Moreover, MS was already present (Fig. 2D). Patient underwent anterior cervical canal decompression surgery. We performed C3-C4 C4-C5 C5-C6 discectomy and interbody cervical fusion by three cages (Fig. 3B). Following clinical course regularly took place. Slow but progressive clinical improvement was showing. At 3-months follow-up she came to the clinic, able to walk autonomously with unilateral support and to perform daily activities. Cervical MRI scan documented successful surgical outcome: satisfying cervical canal decompression and stability of the area of altered intra-medullary signal (Fig. 3A).

Discussion

Cervical myelopathy disease is generally characterized by a progressive worsening when caused by spondylar-disc-arthrosis. Acute cervical myelopathy etiopathological diagnosis is challenging[2].

In our case report, clinic acutely onset in two episodes: the second one causing marked neurological worsening if compared to the first one. Clinical history would suggest a cardiovascular event followed by spinal cord infarction. Nevertheless, patient is young; she does not have cardiovascular risk factors (hypertension, atrial fibrillation, heart disease) nor hypercoagulability pathological conditions. No imaging examination was positive for thrombo-embolic lesions; CT aortic angioCT scan study was negative for dissecting lesions.

On the other hand, laboratory CSF tests by rachicentesis did not document any inflammatory or demyelinating diseases.

Myelopathy onset was diagnosed by cervical spine MRI, revealing an altered intramedullary signal area, following a hypothetic cardiovascular event. Cervical spine cord signal alteration was documented 12 hours later neurological symptoms onset, when the first embolic cleavage probably occurred. 24 hours later, as a consequence of a following probable embolic event, patient presented further acute neurological worsening: it produced a severe tethraparesis (ASIA C). Latency interval between the onset of the symptoms and the radiological evidence would suggest an ischemic problem.

Acute myelopathy by spine cord infarction would result spinal cord swelling inside a narrow cervical canal. Anterior triple discectomy decompression surgery would produce inside cervical canal pressure reduction, favoring medullary perfusion and secondary ischemic damage prevention.

FCE could contribute to explain the event[4, 5, 6]. As already proposed in other previous papers, spinal cord infarction is a rare disease and accounts for about 1% of all strokes[3, 7]. If no other obvious causes on imaging and laboratory tests are present, it can be a pathophysiological cause, albeit extremely rare. Its diagnosis is exclusive[8, 9, 10, 11].

Peaks in the incidence of FCE are made up adolescence and middle age[4, 12, 14]. Hypothesized mechanism is migration of material from the nucleus pulposus into the vessels that supply the medulla[2, 4, 5, 9, 10]. Typical presentation includes neck or lumbar pain, followed by neurological deficits, up to supply territory of the occluded vessel. More frequently, ASA closure brings to bilateral motor deficits with possible asymmetry and sensory deficit from involvement of the spino-thalamic tract[2, 3, 4, 8].

Unfortunately FCE diagnosis would be possible only with anatomopathological examination (and can be performed post-mortem)[14, 16]. CSF analysis is mandatory to exclude inflammatory, infectious and autoimmune pathological forms, as well as demyelinating diseases. As well as the already published cases, in our case the only data detected is a mild hyperprotidorrachia[8, 15].

24 hours later onset of symptoms MRI study documented spine cord swelling and an alteration signal in T2 sequences, confirming SCI.

Proposed algorithm for the FCE diagnosis is: establish the diagnosis of myelopathy and the level involved; exclude traumatic and compressive etiologies; exclude inflammatory etiologies; establish the diagnosis of medullary infarction; establish the possible diagnosis of FCE (related to minor spinal trauma or episodes of increased intradiscal pressure and/or Valsalva maneuvers, presence of disc degeneration, especially Schmorl's hernietion or close to the level of myelopathy and absence of cardiovascular risk factors)[4].

According to literature clinical practice, therapeutic strategies were: hypothermia; systemic hypertension; spinal fluid drainage; oxygen therapy; pharmacological neuroprotection; prevention of secondary ischemic damage[2].

Secondary damage prevention is provided by perfusion pressure adjustment (by increasing mean arterial pressure and/or reducing CSF pressure with the external lumbar drainage); in our case, cervical canal via anterior decompression protected spine cord from secondary damage, before second ischemic insult could determine more serious effect than the first embolism.

The question remains whether it would be more appropriate to operate soon after the first clinical manifestation, earlier than the second episode occurred, despite spine cord ischemia was not revealed by MRI scan and self-resolution of the tetraparesis in the following 24 hours.

Conclusions

In cases of spinal cord swelling as a consequence of infarction, principles of neuroprotection are valid as well as in cervical cord contusion in a stenotic canal. Decompression of the acute canal can allow the prevention of secondary ischemic damage in the ischemic penumbra thanks to the adjustment of the perfusion pressure.

Abbreviations

ASA= anterior spinal artery; CC= cervical canal; CSF=cerebrospinal fluid; FCE= fibrocartilagineous embolism; MRI= magnetic resonance imaging; MS= medullary swelling; SC= spinal cord; SCI= spinal cord ischemia; SCPP= spinal cord perfusion pressure; TSE= turbo spin echo; STIR= short TAU inversion recovery; GRE= gradient echo; DWI= diffusion weighted imaging.

Declarations

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2. Conflicts of interest/Competing interests: the authors report no conflict of interest concerning the materials or methods used in this study or the findinges specified in this paper. The authors declare that they have no competing interests.
3. Ethics approval: not applicable.
4. Consent to participate: written informed consent was obtained from the patient for publication of this case report and accompanying images.
5. Written Consent for publication: written informed consent was obtained from the patient for publication of this case report and accompanying images.
6. Availability of data and material: the data that support the findings of this case report are available from the corresponding author, upon reasonable request.
7. Code availability: not applicable.
8. Authors' contributions: Acquisition of data: DN; Conception and methodology design: DN and LA; Drafting the article: DN, AV, AB; Manuscript reviewing and editing: DN and GZ; Supervision: DN and LA; All authors have read and approved the final version of the manuscript.

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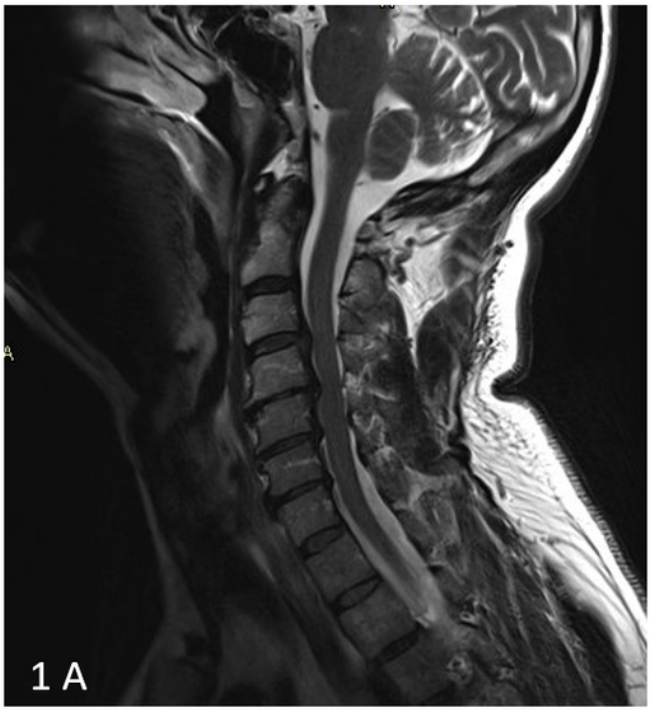
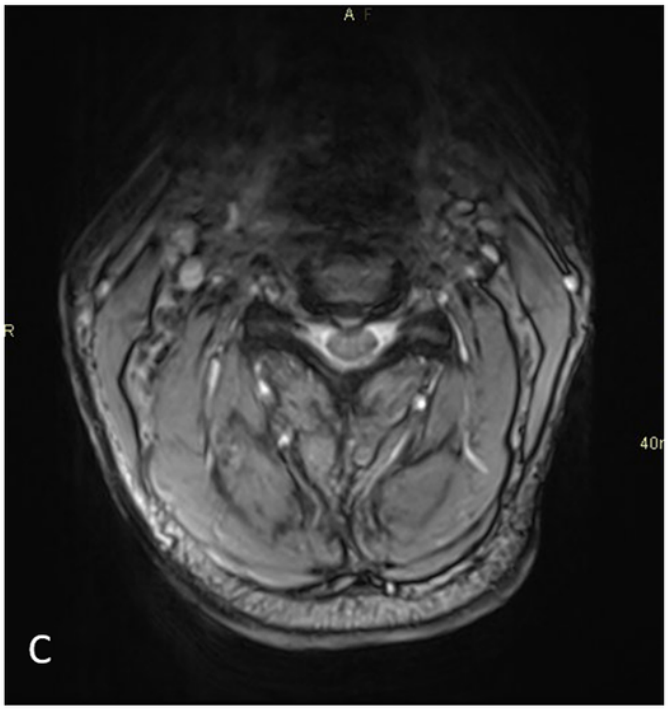
Figures





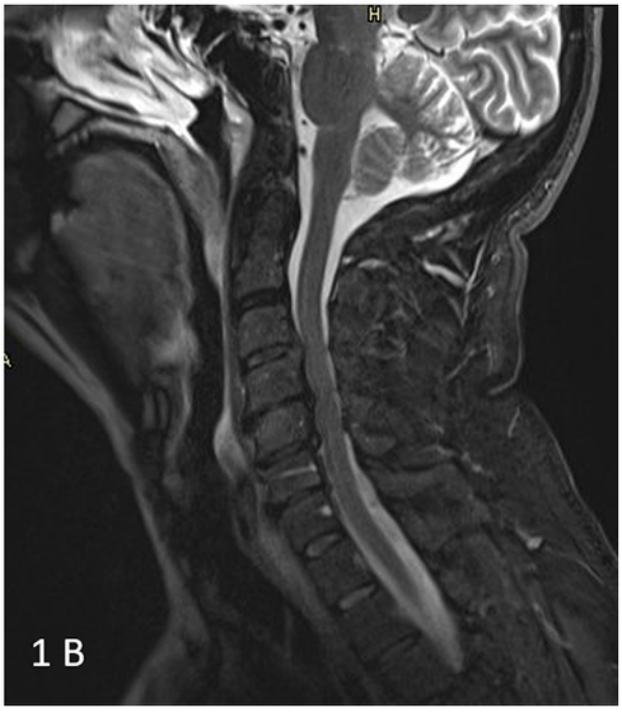
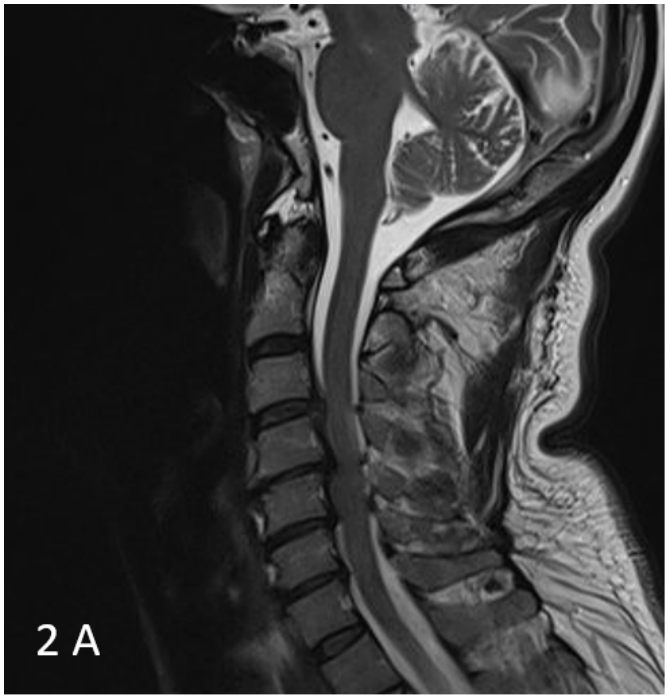
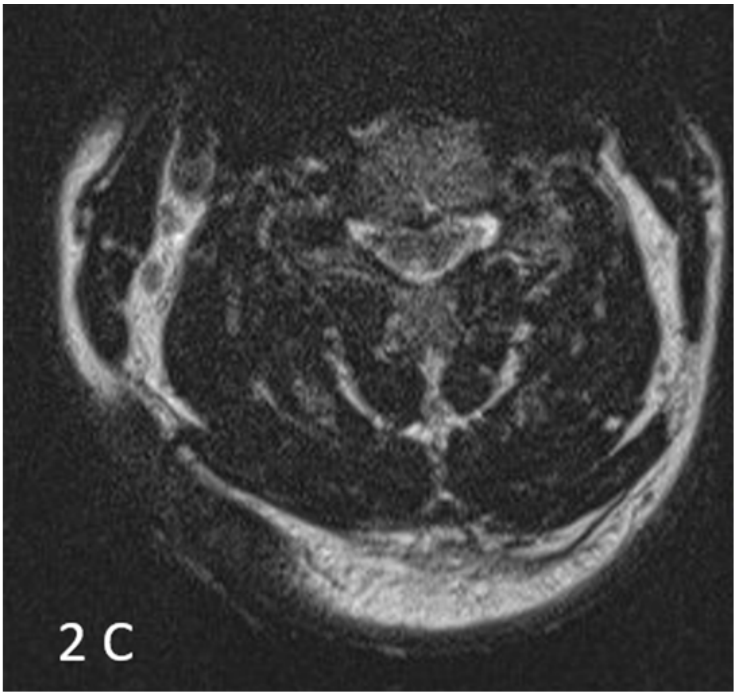
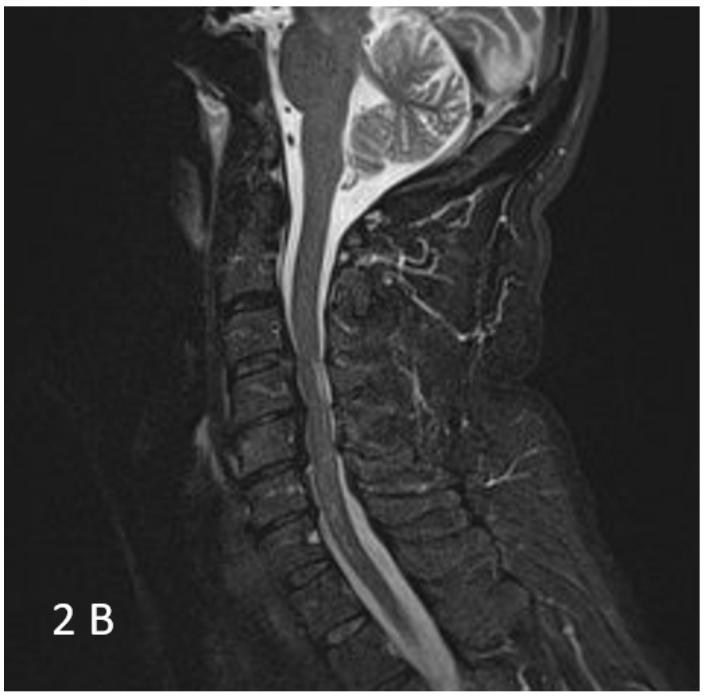


Figure 1

A and 1 B. Sagittal T2-weighted TSE and STIR (Fig. 1 A, 1 B) image of the cervical spine acquired at the onset of the symptoms, demonstrated multiple disc protrusions and spinal canal narrowing, without signal alteration of the spinal cord.







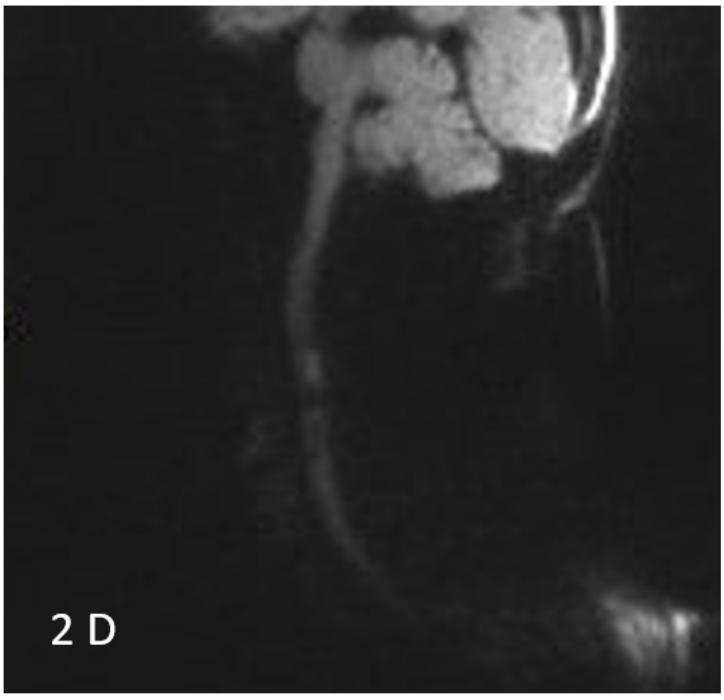


Figure 2

A, 2 B, 2 C, 2 D. Sagittal T2 weighted TSE (Fig. 2 A) and STIR (Fig. 2 B), axial T2-weighted TSE (Fig. 2 C) and sagittal DWI (Fig. 2 D) images of the cervical spine acquired two days after and which showing signal alteration of the posterior spinal cord at C4, characterized by diffusion restriction.

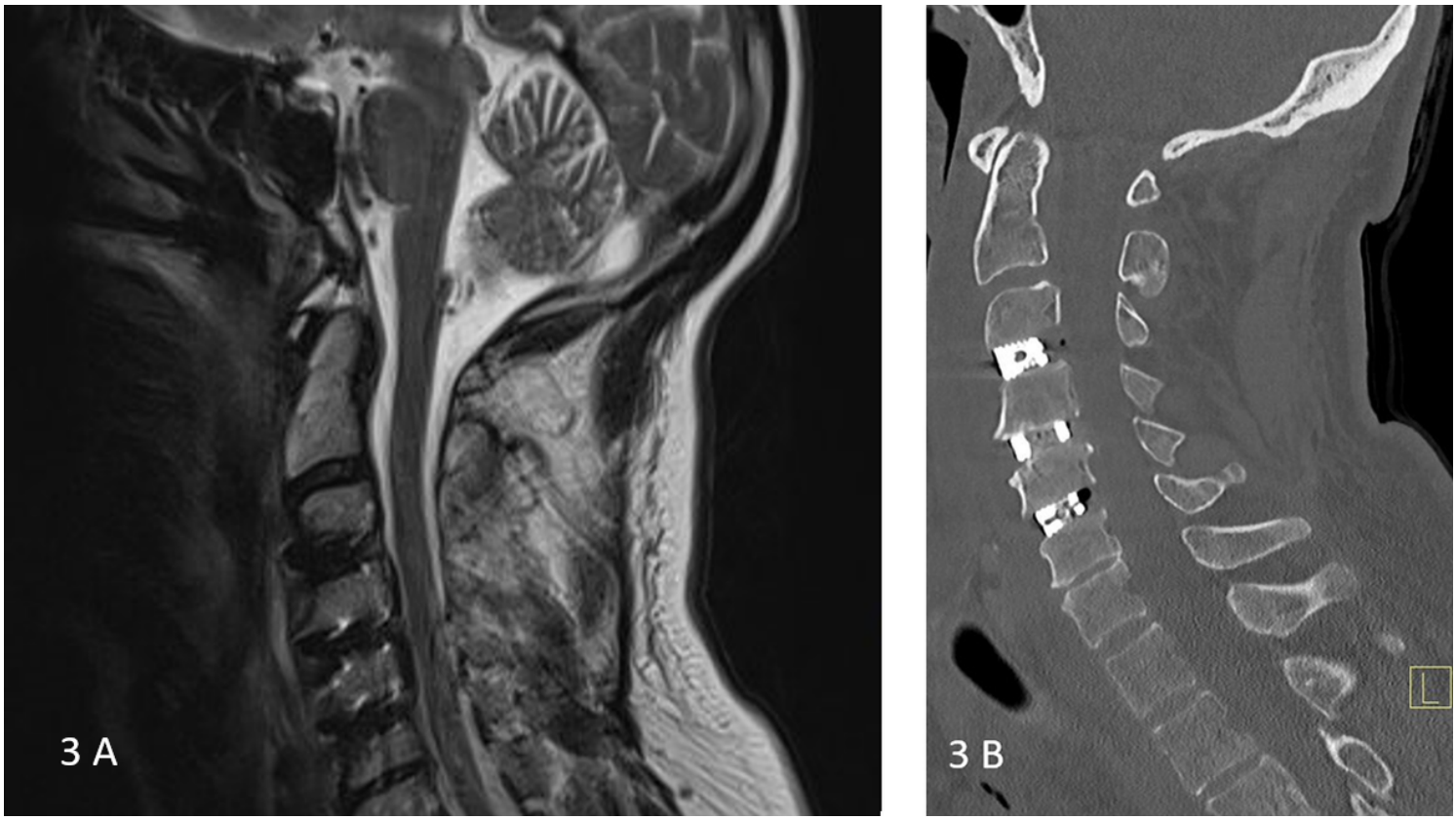


Figure 3

A and 3 B. Follow up T2 weighted TSE image on MRI showing the post-ischemic changes in spine (Fig. 3 A) Sagittal CT the day after the surgery. A triple discectomy and interbody cervical fusion was performed (Fig. 3 B).

Supplementary Files

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