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Posterior Ring Apophysis Separation (PRAS): A relatively rare clinical entity in adolescents & young adults

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Herniated Lumbar Disc (HLD) may be associated with separation of the posterior ring apophysis of the adjacent vertebral body. It is often difficult to diagnose radiologically and may be confused with calcification of the posterior vertebral structures. We report a 32 years old male, presenting with a history of low back pain radiating to the left lower limb and associated with tingling sensation and paresthesia. Magnetic Resonance Imaging (MRI) of lumbosacral (LS) spine revealed left L5-S1 lateral disc herniation compressing the exiting nerve root. CT scan with sagittal reconstruction of LS spine revealed PRAS of upper lip of S1 vertebra. L5 partial laminectomy and microdiscectomy with removal of the loose bony fragment was done. Post-operative MRI demonstrated decompression of the spinal canal and the neural foramen. We report this case to familiarize the clinicians with this clinical entity as it requires strong clinical suspicion coupled with sagittal reconstruction CT scan for diagnosis and extensive exposure with removal of loose fragments together with discectomy for adequate relief of the symptoms.

Key Words: CT with sagittal reconstruction, HLD, MRI, PRAS

Herniated Lumbar Disc (HLD) is a common spinal problem these days. Changes in lifestyle, nature of work, strenuous physical activity or trauma predisposes to HLD in younger population. HLD, at times, may be associated with separation of the posterior ring apophysis (PRAS) of the adjacent vertebrae. These fragments may sometimes be missed on plain radiographs or MRI scans because of their small size and may even be confused with calcification of PLL or herniated disc or with the posterior degenerative ridge osteophytes.[1,2] This condition is predominantly seen in younger population involved in strenuous physical activities or in young adults with a history of traumatic episode.[1,5] Clinicians have difficulty in diagnosing the condition partly because of the small size of the fragment and partly due to unfamiliarity with this clinical entity.

Case Report

A 32-years-old gentleman presented with the history of severe low back pain since 1 week. Initially the pain was mild with gradual onset since 3-4 months which radiated

Figure 1: Pre-operative plain radiograph lateral view

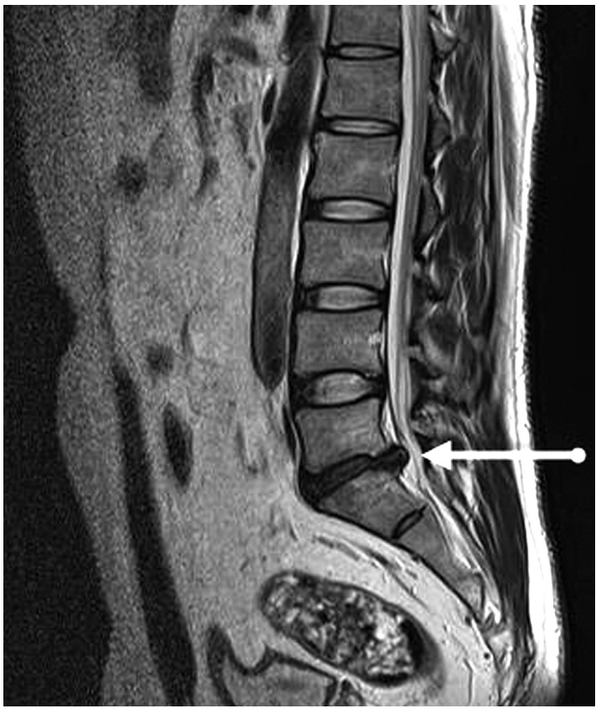

Figure 2: T2W sagittal image showing HLD at L5-S1 level

to the left lower limb. The pain was worsened by lifting heavy object and was associated with dysesthetic pain over the left leg since 1 week. The pain was aggravated on coughing and sneezing. The patient could not stand straight and had limping gait. Further neurological examination revealed diminished sensation over the left S1 dermatome and bilateral straight leg raising test (SLRT) was positive (i.e. cross sciatica positive). Magnetic Resonance Imaging (MRI) of lumbosacral (LS) spine revealed left lateral disc herniation at L5-S1 level with mild compression of the exiting nerve root. The patient was initially managed conservatively with steroids, analgesics, methylcobalamine, pregabalin and rest. However, there was no significant improvement

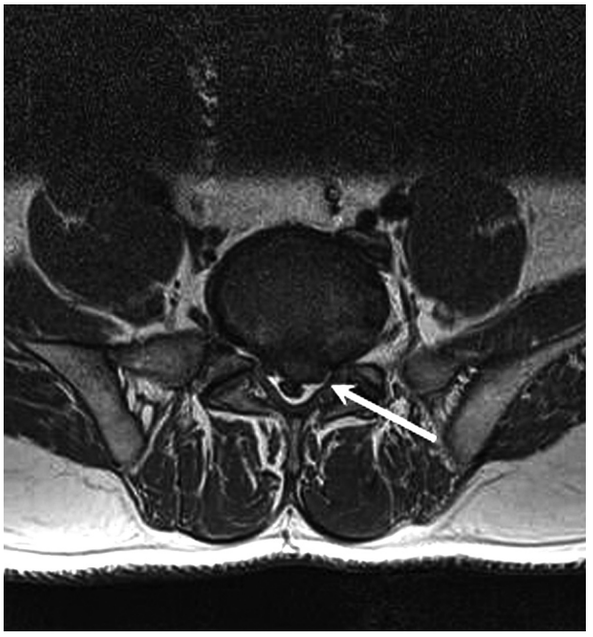

Figure 3: T2W axial image at L5-S1 level showing left sided HLD and compressing the exiting nerve root

Figure 4: Pre-operative CT scan (sagittal section) showing PRAS of upper lip of S1 vertebra

Figure 5: Pre-operative CT scan (axial section) at L5-S1 level showing PRAS of upper lip of S1 vertebra causing root canal stenosis.

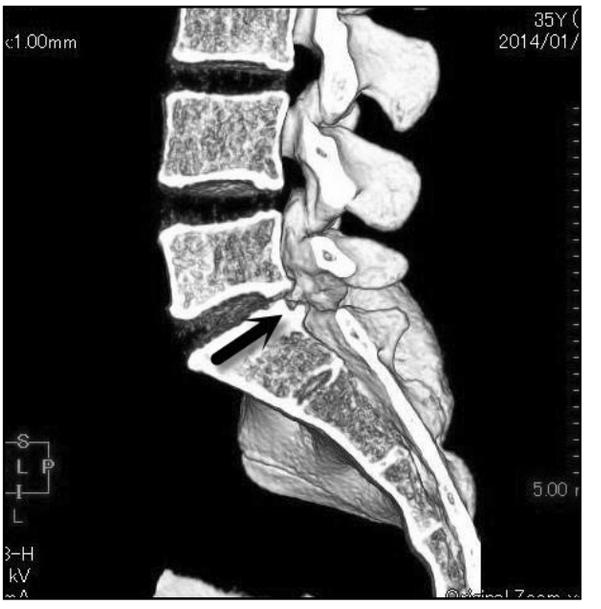

Figure 6: Pre-operative CT sagittal reconstruction

with conservative treatment. A repeat MRI scan of LS spine after 6 weeks revealed increase in disc herniation with significant compression of the left S1 nerve root **(Figure 1, 2, 3)**. CT scan with sagittal reconstruction of LS spine revealed PRAS of upper lip of S1 vertebra with severe stenosis of the neural foramen **(Figure 4, 5, 6)**. The patient then underwent L5 partial laminectomy and microdiscectomy with removal of the loose bony fragment after determining its mobility intra-operatively. Post-operative CT imaging of LS spine demonstrated adequate decompression of the spinal canal and the neural foramen with release of the nerve impingement **(Figure 7, 8)**. There was significant improvement in the neurological condition of the patient following surgery with no additional neurological deficits.

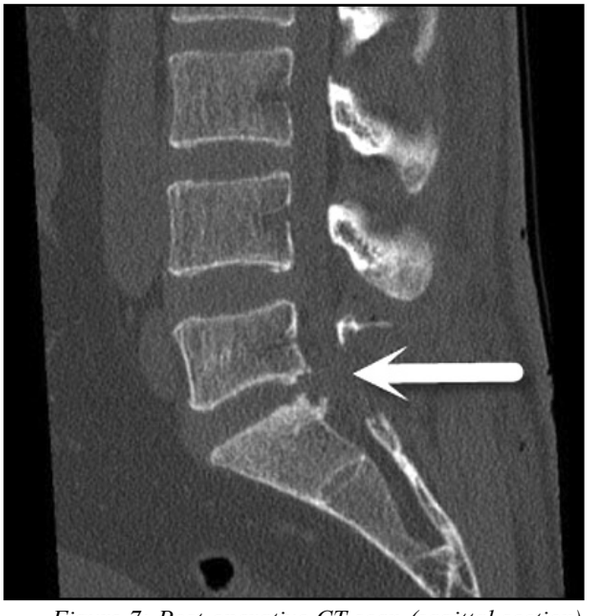

Figure 7: Post-operative CT scan (sagittal section) showing decompression of spinal canal following removal of mobile bony fragment.

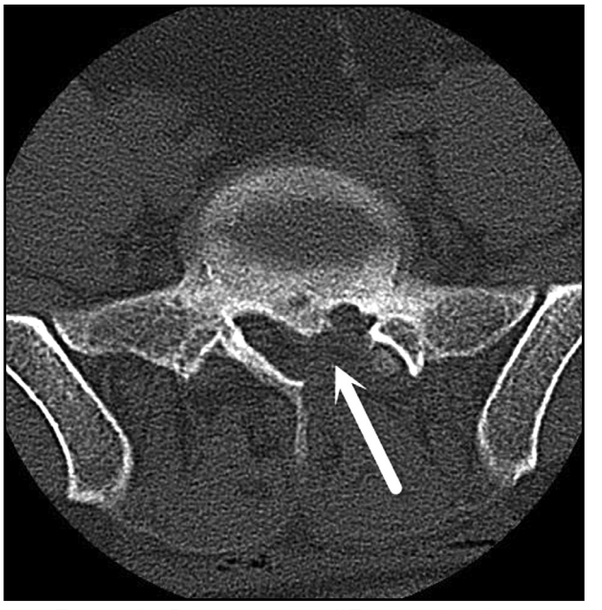

Figure 8: Post-operative CT scan (axial section) showing decompression of neural foramen.

Type	Description
Type I	Arcuate fragment in spinal canal with no osseous defect in vertebral body
Type II	Separation of cortical and cancellous bony rim from vertebral body
Type III	Localised tear-drop fracture of the end-plate
Type IV (elaborated by Epstein et al.)[6, 7]	Fracture along the full axial length of the posterior margin of the vertebral body

Table 1: Takata et al. classification

Type	Description
Type I	Partial separation of bony fragment from posterior edge of vertebral body
Type II	Complete separation of bony fragment from posterior edge of vertebral body
Based on relationship of the bony fragment with the displacement of disc material into the canal	
Stage A	Disc material displaced upto the margin of bony fragment
Stage B	Disc material displaced beyond the margin of bony fragment

Table 2: Akhaddar et al. classification

Discussion

Herniated Lumbar Disc is one of the common spinal problems in the youngsters in recent days. Less commonly, HLD may be associated with separation of posterior ring apophysis of the adjacent vertebrae. PRAS is mainly considered to be the problem of the adolescents. As the apophysis is still not fused with the vertebral body till the end of skeletal growth i.e. around 18-20 years,[8] it acts as the weak point between the apophysis and the vertebral body and is prone to separation with trauma or strenuous physical activities.[3,8,11,14] The pathological mechanism of the PRAS is still controversial, however, it is thought that hyperextension of the lumbar spine or rapid flexion coupled with axial compression of the vertebral column leads to PRAS.[1,9] Some authors also emphasize the additional role of degenerative process of the inter-vertebral disc and the vertebral cartilage in the pathogenesis of PRAS.[1,10,11] while others suggest PRAS to be the result of avascular bone necrosis of the posterior end plate or a slipped vertebral apophysis syndrome analogous to the slipped femoral epiphysis.[1,5] The incidence is reported to be around 5.7-7.5% of all patients with HLD and with higher incidence among adolescents.[1,8,12]

PRAS does not occur in isolation.[1] So it is characterized clinically by the features of HLD. In literature, children and adolescents with PRAS presenting with reactive scoliosis has been well documented.[1,10] Reactive scoliosis occurs to compensate for the stenosed foramen and offers to widen the foramen and hence protect the impinged nerve root. HLD with PARS most commonly involves the L5-S1 level, as in our case, as opposed to HLD alone which most commonly involves the L4-L5 level. Involvement of more than one level is very rare in PRAS with the superior endplates of the lower vertebra at the involved level being most commonly affected.[1,5,8,11] MRI has limitations in diagnosing PRAS as the small bone fragments appear as areas of low signal intensity protruding into the spinal canal along with the disc forming a Y- or 7-shaped configuration elevating the PLL. This may be confused with the calcified PLL or disc or with the posterior ridge osteophytes.[1,2,4,8,11] CT-Scan with sagittal reconstruction provides a very good view with accurate delineation of the posterior margin of the vertebrae with the bony defect and the fragments near the edge of the endplate.[1,8]

Classification

Based on the radiological feature of the ring apophysis separation, many authors have classified them in Various Ways **(Table 1, 2)**.

Based upon these classifications, our case was classified as Type II. [13] or Type IB.[1]

Treatment

Conservative treatment has been shown to be less effective in the management of most cases of PRAS and surgery is recommended for adequate relief of the symptoms.[3] The extent of exposure required to relieve compression on the nerve root depends on the type of the fracture.[1,3,6,8,15] Based

on their classification, Akhaddar et al.[1] suggested the following approach, Type I (with immobile fragments): discectomy with posterior decompression, Type II (with mobile fragments): discectomy with excision of the fragment. They also suggested adequate exposure of duramater, root sleeves and bone fragments to achieve wide posterior decompression as the bone fragment is less pliable than the disc material. In cases of high level lesion or bilateral involvement, Jung-Sik et al.[8] have proposed a wide posterior decompression and posterior fixation if need be. In cases with no clear plane of dissection among the duramater, nerve root and bone fragment, discectomy with foraminotomy and wide posterior decompression is advisable. [1,8,14,15] As in our case, partial laminectomy with discectomy and removal of mobile fragment suffice the relief of impinged nerve root.

Conclusion

PRAS, though uncommon, must be suspected in young, physically active adolescents and adults who present with persistent sciatic scoliosis with radiological features of disc calcification. MR imaging is not very effective in detecting the fractures of posterior fragments and may be confused with calcified PLL or disc or with posterior ridge osteophytes. Computed tomography scanning with sagittal reconstruction is the investigation of choice for diagnosis and should always be performed when the possibility of PRAS is suspected clinically and by MR imaging. Treatment includes extensive exposure with removal of disc and excision of the mobile fracture fragments and liberal decompression to relieve the nerve root compression.

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