Cervical Spondylolysis, Radiologic Pointers of Stability and Acute Traumatic as Opposed to Chronic Spondylolysis

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Study Design: Case report and literature review.

Objectives and Methods: We report a case of subaxial cervical spondylolysis and review 142 reports of this anomaly in the English language literature to determine the cause, the distribution, the stability, and the prognosis of cervical spondylolysis.

Results: Cervical spondylolysis may affect any level of the lower cervical spine with the C6 isthmus being the most commonly affected region (48%). In 40% of cases, there were radiologic features consistent with congenital cervical spondylolysis and in 74% of cases there was a history of acute trauma. The radiographic features of congenital cervical spondylolysis are quite specific and can exclude trauma as a cause for the spondylolysis. Although instability was not commonly assessed, 30% of cases assessed with flexion-extension views were found to be unstable. These included 27 cases of myelopathy of which 26 were attributable to instability. Many treatment modalities have been employed to treat ranging from early mobilization to decompression and fusion. Three patients with radiologic signs of instability and no neurologic deficit were treated conservatively, whereas 1 patient with no neurologic deficit or instability was treated with spinal fusion.

Conclusions: We found a direct relationship between spinal instability and the presence of neurologic deficit ($P < 0.001$). On the basis of this systematic review of case reports of lower cervical spondylolysis, we can recommend that spondylolysis of the lower cervical spine can be treated nonoperatively except in those with radiologically documented cervical instability.

Key Words: cervical vertebrae, spondylolysis, instability, spine

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Unlike lumbar spine spondylolysis cervical spine spondylolysis is most commonly caused by high-energy trauma to the upper cervical spine in the form of a "hangman's" fracture of C2 vertebrae.[1] Despite a large number of case reports of a defect in the subaxial cervical articular masses (spondylolysis), there is a lack of consensus as to the cause and the significance of this anomaly and the appropriate method of treatment.[2–36] The aim of this article is to report a case of such an injury and to review the English language literature looking specifically at radiologic signs of chronic (congenital) as opposed to acute (traumatic) spondylolysis, signs of instability, and the most appropriate mode of treatment of a patient with cervical spondylolysis.

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CASE REPORT

A 54-year-old man presented to our unit after a simple fall from a two feet height hitting the back of his head. He complained of lower back pain and he was found to have an anterior column wedge fracture of the twelfth dorsal vertebra. Two days later, the patient returned to the accident and emergency department complaining of neck pain and paresthesiae in both shoulders. He was treated with prompt spinal immobilization. Clinical investigation revealed no neurologic deficit. Plain radiographs of the cervical spine revealed generalized spondylosis of cervical spine and disruption spinous process of C6 vertebrae and traumatic cervical spondylolysis of C7 vertebrae with the fracture line propagating anteriorly into the superior aspect of the body of C7 vertebrae (Fig. 1). He was further investigated with magnetic resonance imaging (MRI) of the spine and high-resolution computer tomography (Figs. 2, 3). Patient was subjected to controlled flexion-extension views of the cervical spine, which confirmed stability of the segment with less than 10 degrees of anterior angulation and no anterolisthesis (Fig. 4).

He was treated with hard collar immobilization for 8 weeks followed by a repeated lateral cervical spine radiograph, and computed tomography scan of the cervical spine, which showed bony union through both the pars defects in the C7 region and also the disrupted spinous process of C7 vertebrae (Fig. 5). He had full and pain-free range of motion of the cervical spine with no neurologic deficit. His cervical collar was removed. Six months after injury, he was still symptom free with full range of motion of cervical spine and he was discharged from the clinic.

Thirty-five scientific articles in the English language describing 142 separate cases of cervical spondylolysis (excluding hangman-type fractures) were retrieved from the archives using the National Library of Medicine search engine, Pubmed. The date of each case report, the age of the patient, the level of the spondylolysis, the presence of associated congenital

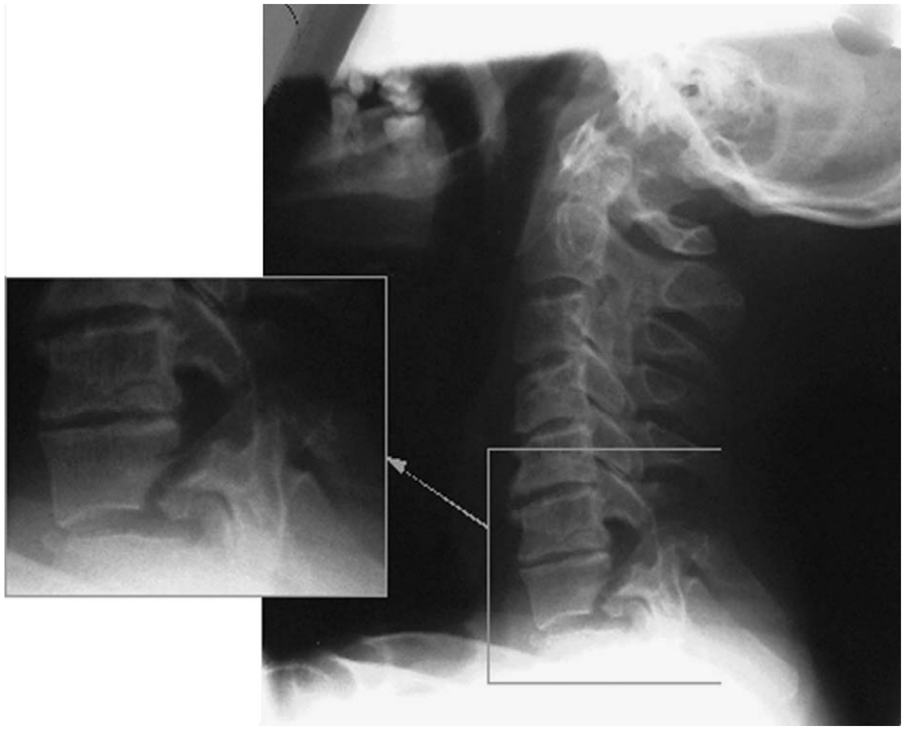


FIGURE 1. Plain lateral radiograph of cervical spine showing an oblique defect in the pars interarticularis of C7 vertebrae propagating anteriorly and inferiorly in to the superior aspect of the body of C7 vertebrae and C7-T1 disc and posteriorly and superiorly in to the C6 spinous process.

anomalies of the cervical spine as described by Forsberg et al,[5] the presence of trauma preceding the diagnosis, the interval between injury and the presentation, the mechanism of trauma when present, the presence of signs or symptoms of neurologic abnormality, the assessment of instability with flexion/extension views and the subsequent treatment method and outcome were recorded (Table 1). We divided the assessment of cervical spine stability into 3 groups: group-1 included patients who had signs of cervical instability on plain or dynamic radiographs of the cervical spine as described by White et al.[37] The cervical spine was considered to be unstable when "all anterior or posterior elements were destroyed or unable to function; more than 3.5 mm of horizontal displacement in 1 vertebrae in relation to subjacent vertebrae or more than 11 degrees of anterior tilt as

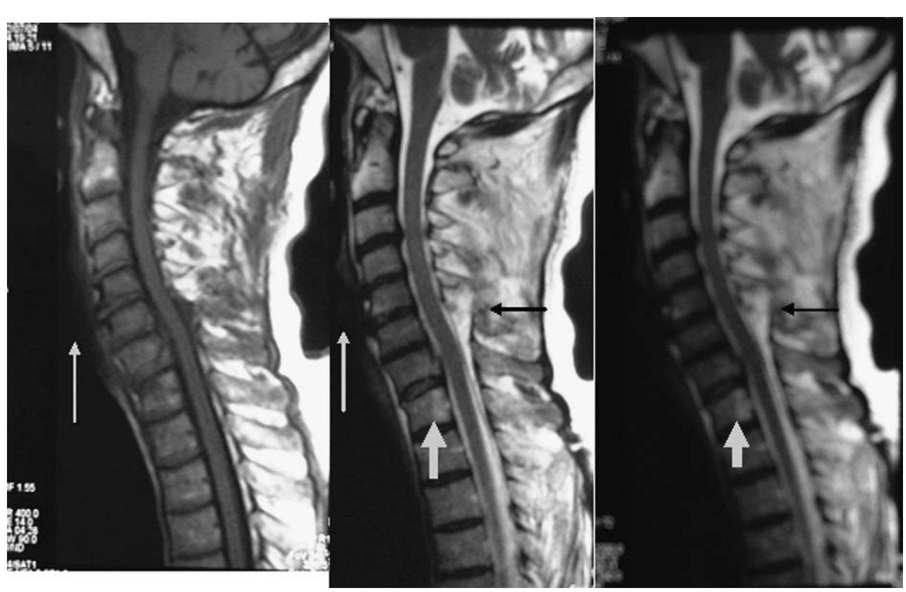


FIGURE 2. MRI of cervical spine showing preserved alignment and soft tissue reaction (thin arrows) and bruising the vertebral body (thick arrows) of C7 vertebrae suggestive of an acute injury to cervical spine.

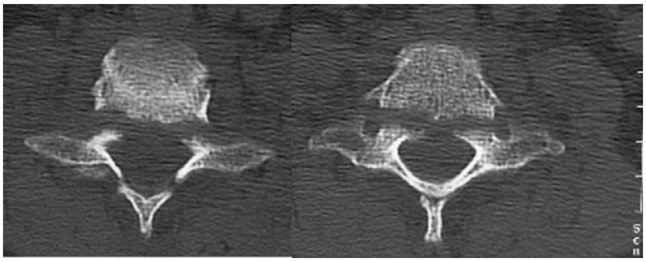


FIGURE 3. High-resolution computed tomography of the cervical spine showing disruption of the neural arch of the C6 vertebrae in addition to the deformities seen in the plain film and suggesting acute trauma as a cause of the anomalies by virtue of the sharp edges of the fracture and disrupted trabeculae.

compared with subjacent vertebrae in lateral cervical radiograph or dynamic radiographs."[37] The group-2 patients were assessed by flexion/extension views and were found to have a stable spine. Group-3 included the patients who did not have any radiographic assessment of instability with flexion/extension views.

To calculate any association between neurologic insult and cervical instability, the presence of neurologic deficit in patients who demonstrated instability in static cervical spine radiographs and flexion/extension views was analyzed statistically using χ^2 test.

RESULTS

There were a total of 142 patients with an average age of 33.5 ± 12 years (range 8 to 81 y). Fifty-seven patients (40%) had associated congenital anomalies involving cervical vertebrae with spondylolysis. Sixty-seven patients (48%) had spondylolysis of the C6 vertebrae and 5 patients (4%) had multilevel spondylolysis (Graph 1). One hundred and five patients had suffered acute spinal trauma before radiology of the spine.[5–7,9,20,29–36] Of these, 29 were flexion-type injuries, 3 were compression-type injuries, and 57 were extension-type injuries. The mechanism of injury was not specified in the other 16 cases.[5–7,9,20,29–36] Of those who had preceding trauma, 96 patients presented immediately after their injury, 6 patients presented within days but after 24 hours of the injury and 3 patients presented several months after the episode of trauma (Table 1).[5–7,9,20,29–36] Twenty-seven patients presented with symptoms of gross neurologic deficit and myelopathy one of which was transient.[7,8,26,30,34–36] In 26 of these cases, the neurologic deficit was attributable to neural compression caused by instability.[7,8,26,32,35,36] Thirty-seven other patients had mild signs and symptoms of nerve root irritation and 28 patients had no neurologic symptoms at presentation.[2–6,9,10–20,23,24,31,33,35,36] Sixty-eight patients had gross instability on plain radiography of the spine. The stability was assessed using flexion/extension views in another 25 patients of whom seven were found to be unstable and 18 patients were found to be stable.[2–28]

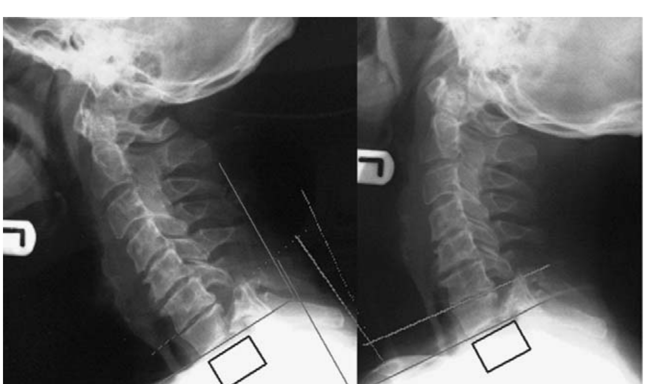


FIGURE 4. Controlled flexion/extension views confirming inherent stability with less than 10 degrees of anterior tilt in C6-C7 level and virtually no movement at C7-T1 level. Note the box outlines the T1 vertebrae.

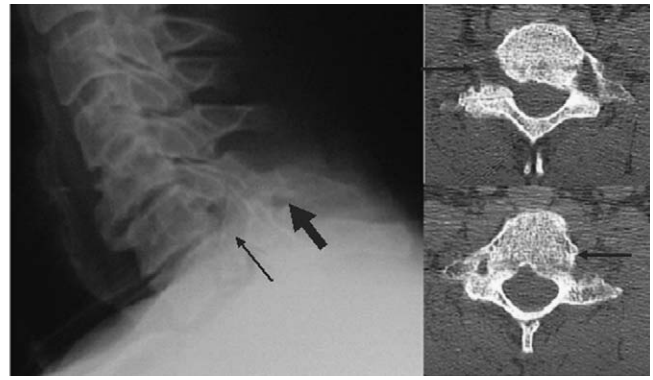


FIGURE 5. Repeated lateral radiograph and high-resolution computed tomography, showing union through the spondylolysis and anterior part of the injury (thin arrows) and also spinous process disruption (thick arrow).

Sixty-seven patients had surgical intervention. This comprised of 1 patient with posterior decompression and 26 patients with fusion (1 patient with posterior decompression and anterior fusion, 49 anterior inter-body fusions, 16 posterior fusions, and 1 patient with combined anterior and posterior instrumentation and fusion).[6–11,13,17,18,23,30,31] Of the patients who had fusion, 51 were found to be unstable preoperatively, 1 was found to be stable but was fused after decompression and 14 were not assessed for instability preoperatively using flexion-extension views.[6,9–13,17,18,23] In the group of patients who were not treated with fusion, 8 patients had closed reduction of the associated listhesis and maintenance of reduction with halo frame. In the conservatively treated group, 17 patients had flexion/extension views of which 16 were found to be stable and interestingly one was found to be unstable.[2,4,20] Eighteen patients had no assessment of instability before the decision for conservative treatment.[8,12,15,19,20,23]

We found a direct statistical association between the presence of instability diagnosed by the lateral C-spine radiograph or after flexion/extension views and the presence of neurologic anomaly ($\chi^2 = 34$, $P < 0.001$, DF = 1). In the patients with congenital anomalies, C6

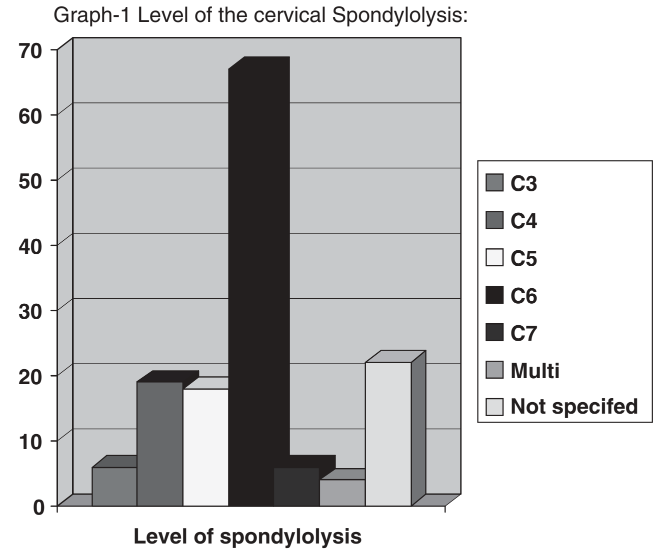
TABLE 1. List of Case Reports of Cervical Spondylolisthesis Including the Age of the Patient, Level of Pathology, Presence of Associated Congenital Anomalies or Preceding trauma, Mode of Presentation, Presence of Instability or Distal Neurovascular Deficit and Mode of Treatment in English Literature[1,2,4–9,11–34]

Authors	Year	Age	Level	Trauma	Associated Pathology	Mechanism	Presentation	Flexion/Extension	Neurology	Treatment
Our case	2004	54	C7	Fall	Trauma	Flexion	> 24 h	Stable	None	Conservative
Shah and Rajshekhar[30]	2004	40	C7	Fall	Trauma	Flexion	> 24 h	Unstable*	Gross	Ant fusion
Kalayci et al[35]	2004	33	C7	RTA	Trauma	Flexion	Immediate	Stable	None	Conservative
Kalayci et al[35]	2004	34	C7	RTA	Trauma	Flexion	Immediate	Unstable	Mild	Ant fusion
Menku et al[31]	2004	35	C6	RTA	RTA	Flexion	Immediate	Unstable*	None	Ant and postfusion
Tannoury et al[32]	2004	20	Multi	RTA	RTA	Compression	Immediate	Unstable*	Gross	Conservative
Ido et al[9]	2002	56	C6	RTA	Trauma	Extension	> 24 h	Unstable†	None	Ant fusion
Schwartz[29]	2001	34	C6	RTA	Congenital	Extension	Immediate	None	None	Conservative
Lifeso and Colucci[36] (50 patients)	2000	37	90% C4-C6	RTA 38 Fall 5 Other 7	Trauma	Compression/Extension	Immediate	Unstable	8 None 16 Gross 26 Mild	Conservative 21 Ant fusion 18 Postfusion 11
Redla et al[27]	1999	29	C6	RTA	Congenital	Extension	> 24 h	None	None	Ant fusion
Redla et al[27]	1999	53	C6	None	Congenital	None	Chronic	None	None	Conservative
Redla et al[27]	1999	59	C6	Fall	Congenital	Extension	> 24 h	None	None	Conservative
Hinton et al[33]	1993	8	C3	Fall	Congenital	Extension	Immediate	Stable	None	Conservative
Poggi et al[25]	1992	20 to 81	6 C6 4 C4	8 Trauma (5 RTA) 2 None	10 Congenital	3 Flexion 5 Not Spec 2 none	5 Immediate 5 Chronic	Stable	None	Conservative
Bhojraj and Shahane[8]	1992	8	C6	None	Congenital	None	Chronic	Unstable*	Gross	Vertebrectomy and ant fusion
Jones and Sage[28]	1992	18	C6	RTA	Congenital	Not spec	Immediate	Stable	None	Conservative
Rovin et al[6]	1992	26	C6	RTA	Trauma	Compression	Immediate	None	None	Ant fusion
Lee and Woodring[34] (22 patients)	1991	Not spec	75% C4-C6	Not spec	Trauma	Flexion	Immediate	1/3 None 2/3 Unstable	12 None 6 Gross 4 Mild	Posterior fusion
Faure et al[16]	1990	24	C6	RTA	Congenital	Extension	Immediate	None	None	Postfusion
Forsberg et al[5] (12 patients)	1990	20 to 80	C6	9 Trauma (8 Major) 3 None	Congenital	Not specified	3 Chronic 9 Immediate	Not spec	1 Mild 11 None	Variable but not specified
Hirota et al[7]	1988	38	C6	RTA	Congenital	Extension	Chronic	Stable‡	Gross	Postdecompression and ant fusion
Morvan et al[26]	1984	19 to 56 Average 29	5 C6 1 C3 1 Multi	Not spec	6 Congenital 1None	Not Spec	Not Spec	2 Unstable 1 Stable 4 None	1 Gross 6 None	1 Decompression 6 Conservative
Schwartz et al[23]	1982	14	C6	None	Congenital	None	Screening	None	None	Postfusion
Schwartz et al[23]	1982	8	Multi	None	Congenital	None	Screening	Stable	None	Conservative
Schwartz et al[23]	1982	15	C7	None	Congenital	None	Chronic	None	None	Conservative
Schwartz et al[23]	1982	20	C5	Not spec	Congenital	Not spec	> 24 h	None	None	Conservative
Kosnik et al[13]	1979	42	C6	None	None	None	Chronic	Unstable	Mild	Ant fusion
Charlton et al[3]	1978	60	C6	None	Congenital	None	Chronic	None	None	Conservative
Charlton et al[3]	1978	50	C6	None	Congenital	None	Chronic	None	None	Conservative
Charlton et al[3]	1978	37	C4	RTA	Congenital	Not Spec	Immediate	None	None	Conservative
Sheikholeslamzadeh et al[19]	1977	37	C4	None	None	None	Chronic	None	None	Conservative
Mosley et al[20]	1976	14	Multi	Fall	Congenital	Not spec	> 24 h	Stable	None	Conservative
Guillaume et al[14]	1976	50	C6	None	Congenital	None	Chronic	None	Mild	Not spec
Guillaume et al[14]	1976	42	C6	None	Congenital	None	Chronic	None	Mild	Not spec

TABLE 1. (*continued*)

Authors	Year	Age	Level	Trauma	Associated Pathology	Mechanism	Presentation	Flexion/Extension	Neurology	Treatment
Proleau and Wilson	1975	46	Multi	None	Congenital	None	Chronic	Unstable	Mild	Ant fusion (C5-6) only
Bellamy et al[10]	1974	16	C5	None	Congenital	None	Chronic	Unstable	Mild	Postfusion
Azouz et al[24]	1974	34	C6	None	Congenital	None	Chronic	None	None	Not spec
Azouz et al[24]	1974	45	C6	None	Congenital	None	Chronic	None	None	Not spec
Azouz et al[24]	1974	38	C4	None	Congenital	None	Chronic	None	None	Not spec
Cautilli et al[15]	1972	15	C6	None	Congenital	None	Chronic	None	None	Conservative
Cautilli et al[15]	1972	15	C6	None	Congenital	None	None	None	None	Conservative
Hanai et al[22]	1971	12	C6	None	None	None	Chronic	None	None	Conservative
Dawley[17]	1971	11	C6	None	Congenital	None	Chronic	None	None	Ant Fusion
Niemeyer and Penning	1963	43	C6	None	None	None	Chronic	Stable	None	Conservative
den Orth et al[12]	1969	41	C6	Fall	None	Compression	> 24 h	None	None	Conservative
Durbin[11]	1956	25	C4	None	Congenital	None	Chronic	Unstable	None	Postfusion
Perlman and Hawes[2]	1951	19	C6	None	Congenital	None	Chronic	None	None	Conservative

*Grossly unstable Flexion extension view not attempted.
†Flexion in halo-brace reduced the fracture.
‡Dynamic myelogram performed.
Ant fusion indicates anterior fusion; gross, myelopathy; mild, radiculopathy; multi, multilevel spondylolysis/spondylolisthesis; not spec, not specified; postfusion, posterior fusion; RTA, road traffic accident.



GRAPH 1. Level of the cervical spondylolysis.

vertebral spondylolysis was 74%, more common as compared with spondylolysis caused by acute trauma (30%) ($\chi^2 = 27$, $P < 0.001$, DF = 1).

There was one complications of conservative treatment in form of progression of the neurologic deficit, which was treated with subsequent anterior fusion.[36]

DISCUSSION

Hangman's fracture is a common and well-described cause of spondylolysis of the second cervical vertebrae.[1] This injury is usually caused by high-energy trauma often with devastating results.[1] Isthmic defects of the subaxial cervical spine have been previously reported in the literature.[2–36] They may be unilateral or bilateral.[2–36]

Unlike spondylolysis of the lumbar spine, this anomaly is often discovered during the investigation of neck injury (Table 1).[5–9,12,16,20,29–36] This has lead to the impression that all spondylolysis is caused by trauma, that it is unstable and requires stabilization with spinal instrumentation and fusion.[5,6,9,23] There is often no clearly uniform mechanism of injury or the force causing "the injury" is often not severe enough to explain all of the observed cervical anomalies (Table 1).[5–9,12,16,23]

An observational radiologic study of the cervical spine by Forsberg et al[5] concluded that cervical spondylolysis in the presence of a well-marginated cleft between the facets, triangular configuration of the pillar fragments on either side of the spondylolytic defect, posterior displacement of dorsal triangular pillar fragment, hypoplasia, or hyperplasia of the ipsilateral articular pillars at the level above or below the defect or spina bifida of the involved vertebrae suggest congenital cervical spondylolysis.[5] Other authors have also supported these findings (Table 1).[2,7,10,11,14–18,20–25,27–29,34–36] Congenital spondylolysis is associated with Rubinstein-Tabies syndrome or may be seen as an isolated sporadic anomaly or be inherited through a X-linked recessive or an autosomal dominant genetic condition.[5,21] The findings of Forsberg et al[5] may be disputed as a previous histologic study by Eisenstein et al[38] has shown that despite the traumatic nature of lumbar spondylolysis, the subsequent pseudoarthrosis is not a scar tissue and is a ligamentous structure with a discrete nerve supply and may resemble a developmental defect. There is a cohort of patients in whom the anomalies that are suggestive of trauma are present often in absence of a clear past history of significant cervical injury (Table 1).[4,6,8,9,12,13,19,26,29–31,34,39] In fact, Smith et al[39] has described some of Forsberg's congenital anomalies as being caused by trauma and signifying malunion of spondylolysis. Is a C6 vertebra, most vulnerable to nonunion leading to chronic spondylolysis being most common in this region even though acute trauma is not as common in this level? No matter the cause, changes described by Forsberg signify chronic spondylolysis as oppose to an acute fracture.[5,39]

This begs the question, was the pars interarticularis defect caused by neck injury or was it present before the injury and would it lead to a potential weakness in the spine and subsequent catastrophic spinal injury in this level after a relatively minor trauma?[7–9,12,16,23]

Rovin et al[6] identified 2 different patterns of injury resulting in traumatic subaxial cervical spondylolysis.

1. A posteriorly unstable injury: the simple flexion type, which is associated with posterior element disruption and is stabilised by posterior fusion.
2. An anteriorly unstable injury: the extension type resulting in anterior instability or the axial compression type, which is associated with axial loading in which case there is a 3-column instability, both of which require anterior stabilization and fusion.

Rovin et al[6] has based his findings on clinical observation and previous case reports in the literature. We feel that our patient suffered a type-1 injury leading to the disruption of the posterior column structures namely the interspinous ligaments and both facet joints. What makes our case unique is the persisting stability despite injury to the posterior longitudinal ligament and the posterior aspect of the vertebral body of the C7 vertebra.

Advocates of cervical fusion for the treatment of cervical spondylolysis, whatever the cause, cite radiologic instability (abnormal motion of the involved segment) and risk of displacement or future weakness in the cervical spine as a reason for spinal fusion. This has led to more patients undergoing fusion in the more recent case reports as compared with the earlier reports of this anomaly.[2–30,33] Despite the presumed instability of this lesion and the often-delayed presentation in cases with a previous history of trauma, there have been only 4 cases of spondyloptosis and 27 cases of gross neurologic abnormality seen in the patients with this anomaly.[7,8] Two of the 4 cases with spondyloptosis were caused by high-energy trauma, causing flexion-distraction of the cervical spine at the injured level.[30,32] Finite element analysis of cervical spine after facetectomy and removal of spinal ligaments and facets have shown the spine to be significantly unstable after laminectomy and facetectomy (congenital spondylolysis) or facet excision and removal of the posterior ligaments (traumatic spondylolysis).[37,40,41]

As this review of the literature and our case demonstrate, not all acute traumatic spondylolysis are radiologically unstable. Instability, if present, is more likely to be associated with neurologic symptoms.[2–36] The chronic instability and worsening of neurologic symptoms is exclusive to acutely unstable spine. We feel that if the original injury has not led to the disruption of the anterior structures and spondylolisthesis or neurologic compromise, the injury is stable enough to be treated with immobilization and will lead to union and stability with time. The effectiveness of the external bracing (as opposed to fusion) has been shown in the treatment of the multilevel spinal fracture-dislocation after initial reduction.[32]

The usefulness of MRI has been suggested in the literature and MRI findings have been reported previously.[5,16,25,27,29–31] We found that MRI is poor in delineating the bony architecture of the cervical spine unless there is a gross spondylolisthesis.[25,29–31] We found it useful in assessing the acuteness of the injury by the presence of tissue and bony edema around the involved segment and the cord and the presence or the absence of associated disc prolapse in the unstable segment, which may complicate closed reduction of spondylolisthesis (Fig. 3).[8,23,27,29,30,33,42]

CONCLUSIONS

The commonest level for cervical subaxial spondylolysis is the sixth cervical vertebra.

Radiographic features of congenital cervical (chronic) spondylolysis are quite specific and can be used to exclude recent trauma as a cause for the spondylolysis.

Two different mechanisms of acute traumatic spondylolysis have been described in the literature. The extension/axial compression type, which is stabilised by anterior fusion, and the flexion type, which is best treated with posterior fusion.

We feel that in the absence of radiologic signs of cervical spinal instability on plain cervical spine radiographs and in the absence of neurologic deficit and in the presence of active head control, controlled dynamic views can be performed to further assess the stability of cervical subaxial spondylolysis.

Findings of this study should not be used to rationalize early mobilization of cervical spine after traumatic spondylolysis.

REFERENCES

1. Marar BC. Fracture of the axis arch. "Hangman's fracture" of the cervical spine. *Clin Orthop*. 1975;106:155–165.
2. Perlman R, Hawes LE. Cervical spondylolisthesis. *J Bone Joint Surg (A)*. 1951;33:1012–1013.
3. Charlton OP, Gehweiler JA, Morgan CL, et al. Spondylolysis and spondylolysthesis of the cervical spine. *Skeletal Radiol*. 1978;3:79–84.
4. Niemeyer TH, Penning L. Functional reontgengraphic examination in a case of cervical spondylolisthesis. *J Bone Joint Surg (A)*. 1963;45:1671–1678.

5. Forsberg DA, Martinez S, Volger JB, et al. Cervical spondylolysis: imaging findings in 12 patients. *Am J Reontgenol*. 1990;154:751–755.
6. Rovin RA, Brown JT, Meyer PR. Traumatic spondylolisthesis of the lower cervical spine. *Neurosurgery*. 1992;30:124–127.
7. Hirota S, Amano K, Taketoshi M, et al. A case of cervical spondylolysis causing tetraplagia. *Spine*. 1988;13:113–116.
8. Bhojraj SY, Shahane SM. Posttraumatic cervical spondyloptosis at C6-7 with late onset cord compression: a new clinical entity. *J Neurosurg*. 1992;77:792–794.
9. Ido K, Murakami H, Kawaguchi H, et al. An unusual reduction technique prior to surgical treatment for traumatic spondylilisthesis in the lower cervical spine. *J Clin Neurosci*. 2002;9:664–666.
10. Bellamy R, Lieber A, Smith SD. Congenital spondylolisthesis of sixth cervical vertebrae. *J Bone Joint Surg (A)*. 1974;56:405–407.
11. Durbin FC. Spondylolisthesis of the cervical spine. *J Bone Joint Surg (A)*. 1956;38:734–735.
12. den Orth JO, Penning L, Kluft O. Unilateral spondylolysis of the sixth cervical vertebra. *J Bone Joint Surg (A)*. 1969;51: 1379–1382.
13. Kosnik EJ, Johnson JC, Scoles PV, et al. Cervical spondylolisthesis. *Spine*. 1979;4:203–205.
14. Guillaume J, Rouleau J, Fardou H, et al. Congenital spondylolisthesis and frontal narrowing of the spinal canal. Report of two cases. *Neuroradiology*. 1976;11:159–163.
15. Cautilli RA, Joyce MF, Lin PM. Congenital elongation of the sixth cervical vertebrae in identical twins. *J Bone Joint Surg (A)*. 1972;54:653–656.
16. Faure B, Taylor W, Greenberg BG. Cervical spondylolysis. *Orthopaedics*. 1990;13:250–252.
17. Dawley JA. Spondylolisthesis of the cervical spine: case report. *J Neurosurg*. 1971;34:99–101.
18. Proleau GR, Wilson CB. Cervical spondylolysis with spondylolisthesis. *J Neurosurg*. 1975;43:553–570.
19. Sheikholeslamzadeh S, Alami-Harandi B, Fateh H. Spondylolisthesis of cervical spine. *J Bone Joint Surg (Br)*. 1972;68:704–706.
20. Mosely I. Neural arch dysplasia of the sixth cervical vertebrae. *Br J Radiol*. 1976;49:81–83.
21. Robson MJ, Brown LM, Sharrad WJW. Cervical spondylolisthesis and other skeletal abnormalities of Rubinstein-Taybi syndrome. *J Bone Joint Surg (Br)*. 1980;62:297–299.
22. Hanai K, Miyashita T, Saburi H. Cervical spondylolisthesis. *Acta Orthop Scand*. 1976;47:63–66.
23. Schwartz AM, Wechsler RJ, Landy MD, et al. Posterior arch defects of cervical spine. *Skeletal Radiol*. 1982;8:135–139.
24. Azouz EM, Chan JD, Wee R. Spondylolysis of the cervical vertebrae. Report of three cases with review of the English and French literature. *Radiology*. 1974;111:315–318.
25. Poggi JJ, Martinez S, Hardaker WT, et al. Cervical Spondylolysis. *J of Spinal Disord*. 1992;5:349–356.
26. Morvan G, Busson J, Frot B, et al. Cervical Spondylolysis. 7 cases review of literature. *J Radiol*. 1984;65:259–266.
27. Redla S, Sikdar T, Saifuddin A, et al. Imaging of cervical Spondylolysis with emphasis on MR appearances. *Clin Radiol*. 1999; 54:815–820.
28. Jones DN, Sage MR. Bilateral spondylolysis and associated dysplasia of C6 vertebrae. *Australas Radiol*. 1992;36: 260–261.
29. Schwartz JM. Bilateral Cervical Spondylolysis of C6. *Radiology*. 2001;220:191–194.
30. Shah KC, Rajshekhar V. Successful management of post-traumatic C7-T1 spondyloptosis with uninstrumented ventral surgery. *Surg Neurol*. 2004;62:431–434.
31. Menku A, Kurtsoy A, Tucer B, et al. The surgical management of traumatic C6-C7 spondyloptosis in a patient without neurological deficits. *Minim Invas Neurosurg*. 2004;47:242–244.
32. Tannoury TY, Zmurko MG, Tannoury CA, et al. Multiple unstable cervical fractures with cord compromise treated nonoperatively. A case report. *Spine*. 2004;29:E234–E238.
33. Hinton Ma, Harris Mb, King AG. Cervical spondylolysis. Report of two cases. *Spine*. 1993;18:1369–1372.
34. Lee C, Woodring JH. Sagittally oriented fractures of the lateral masses of the cervical vertebrae. *J Trauma*. 1991;31:1638–1643.
35. Kalayci M, Cagavi F, Acikgoz B. Unilateral cervical facet fracture: presentation of two cases and literature review. *Spinal Cord*. 2004;42:466–472.
36. Lifeso RM, Colucci MA. Anterior fusion for rotationally unstable cervical spine fractures. *Spine*. 2000;25:2028–2034.
37. White AA III, Johnson RM, Panjabi MM, et al. Biomechanical analysis of clinical stability in the cervical spine. *Clin Orthop*. 1975;109:85–96.
38. Eisenstein SM, Ashton IK, Roberts S, et al. Innervation of spondylolysis "ligament". *Spine*. 1994;19:912–916.
39. Smith GR, Beckly DE, Abel MS. Articular mass fracture: a neglected cause of post-traumatic neck pain? *Clin Radiol*. 1976; 27:335–340.
40. Kumaresan S, Yoganandan N, Pintar FA, et al. Finite element modeling of cervical laminectomy with graded facetectomy. *J Spinal Disord*. 1997;10:40–46.
41. Ng H-W, Teo EC, Lee KK, et al. Finite element analysis of cervical instability under physiological loading. *J Spinal Disord*. 2003;16: 55–65.
42. Goldberg AL, Rothfus WE, Deeb ZL, et al. The impact of magnetic resonance on the diagnostic evaluation of acute cervicothoracic spinal trauma. *Skeletal Radiol*. 1988;17:89–95.

