Case Report

International Journal of Neurology and Neurosurgery
Volume 6 Number 2, July - December 2014

Disappearing Large Calcified Thoracic Disc Herniation in a Patient with Thalessemia

Faiz Uddin Ahmad*, Erica Schallert, Jonathan Dante Nahas B.S.**, Judith Post**, Steven Vanni D.O.***

Abstract

Regression of herniated disc fragments with subsequent improvement in clinical symptoms has been reported in the lumbar and cervical spine. Such regressions in the thoracic spine are extremely rare. We report a 38-year-old thalassemia patient who had regression of a large calcified herniated thoracic disc causing cord compression, with subsequent herniation of a second calcified disc at a different level, and discuss the possible etiopathogenesis. This is the first such case reported in the thalessemia population.

Keywords: Calcification; Iron chelation; Regression; Thalessemia; Thoracic disc herniation.

Background information

It is known that disc degeneration and calcification is more common in thalessemia patients than in the general population.[1] Regression of herniated disc fragments with subsequent improvement in clinical symptoms has also been well reported, usually in the lumbar and cervical spine. Such regressions in the thoracic spine are extremely rare.

Objectives

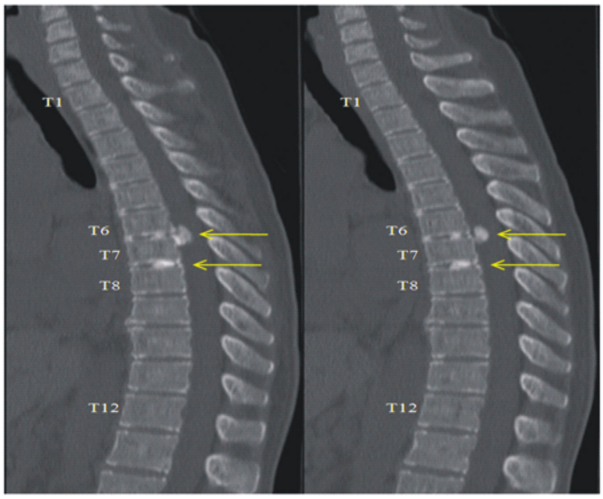
We report an interesting case of regression of a large calcified herniated thoracic disc with cord compression, with subsequent herniation of a second calcified disc at a different level, and discuss the possible etiopathogenesis. This is the first such case reported in the thalessemia population.

Study Design

Case

A 38-year-old female with beta-thalessemia presented to our office in November 2010 with significant upper back pain and a large calcified disc herniation at T6-7 level and a non herniated calcified disc at T7-8. This was

Figure 1a: Initial Presentation of Upper Back Pain. Right Paramidline and Midline Sagittal Noncontrast CT Images of the Thoracic Spine Demonstrate a Calcified Extruded Disc at T6-7 and a Calcified Non Herniated Disc at T7-8



Author's Affiliation: *Department of Neurological Surgery, **Department of Radiology, University of Miami Miller School of Medicine, Miami, Florida U.S.A.

Reprint Request: Steven Vanni, D.O., D.C. Department of Neurological Surgery, University of Miami Miller School of Medicine, 1095 NW 14th Terrace, Lois Pope LIFE Center, D4-6, Miami, FL 33136, U.S.A.

E-mail: SVanni@med.miami.edu

Figure 1b: Initial Presentation of Upper Back Pain. Non Contrast 3 mm Axial Images of the Thoracic Spine Confirm the Large Right Paracentral Calcified Disc Extrusion at T6-7 and the Calcified Non Herniated Disc at T7-8

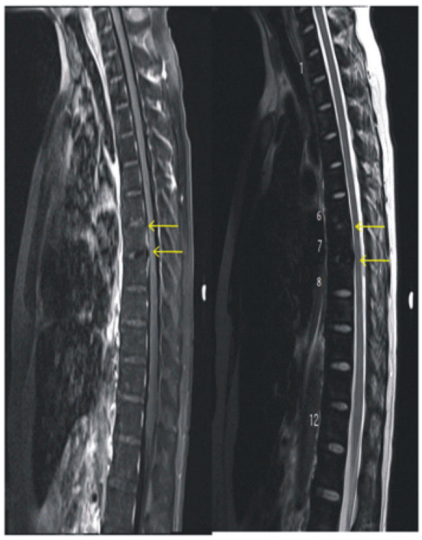


Figure 2a: Almost Two Years Later, Thoracic Back Pain was Aggravated by a Slip and Fall. Sagittal T1 And T2 Non Contrast Magnetic Resonance Images of the Thoracic Spine Demonstrate Resolution of the Previous Herniation and Calcification at the T6-7 Disc with Residual Degenerative Signal (Low T2). There is However, New Herniation of the Previously Calcified Disc at T7-8 which Demonstrates Low T1 and T2 Signal

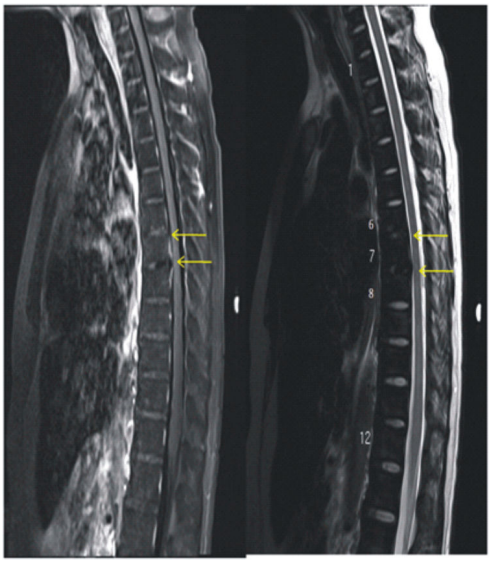
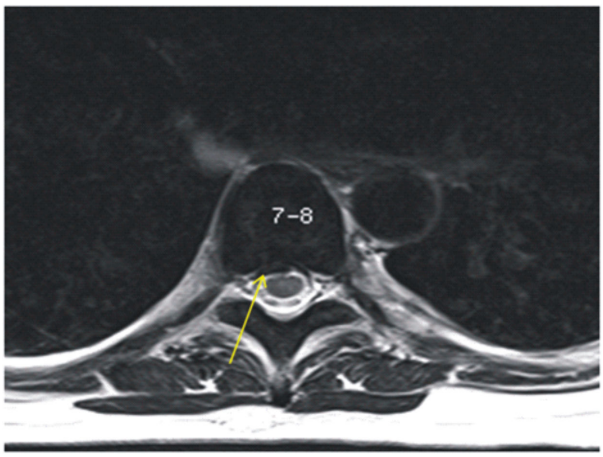


Figure 2b: Almost Two Years Later, Thoracic Back Pain was Aggravated by a Slip and Fall. Axial Non Contrast T2 Weighted Magnetic Resonance Images of the Thoracic Spine at T7-8 Demonstrate a Small Right Paracentral Disc Herniation without Cord Compression



confirmed on computed tomography (CT) scan (Figure 1a, b). She was neurologically intact at that time (normal motor, sensory and reflex exam and no signs of myelopathy), but due to significant cord compression in the thoracic spine, surgical decompression was recommended. While she was waiting to decide on the surgery, her symptoms improved. She was also started on iron chelation therapy for her thalessemia.

We saw her again in clinic in September 2012 when her back pain was aggravated secondary to a slip and fall. She continued to remain neurologically intact. Interestingly, her MRI at this time revealed new herniation of the previously identified non-herniated calcified disc at the T7-8 level while the previously seen calcified disc herniation at T6-7 was no longer visible (Figure 2a, b). Seven weeks later, a follow up CT scan demonstrated progression of the calcified disc herniation at T7-8 which had now extruded and confirmed the disappearance of the calcification of the T6-7 disc (Figure 3). The subsequent MRI correlate scan one week later confirmed these findings. The new central disc herniation at T7-8 demonstrated less cord compression than was seen initially with the

Figure 3: Follow up Non Contrast Sagittal and 2.5 Mm Axial CT Images were Obtained of the Thoracic Spine 7 Weeks Later. the Sagittal Image Confirms Disappearance of the Calcification at the T6-7 Disc and Demonstrates Progression of the Calcified Herniated Disc At T7-8 which is now Extruded. Axial Images at, Above and Below the T7-8 Disc Demonstrate a Moderate Calcified Central Disc Protrusion

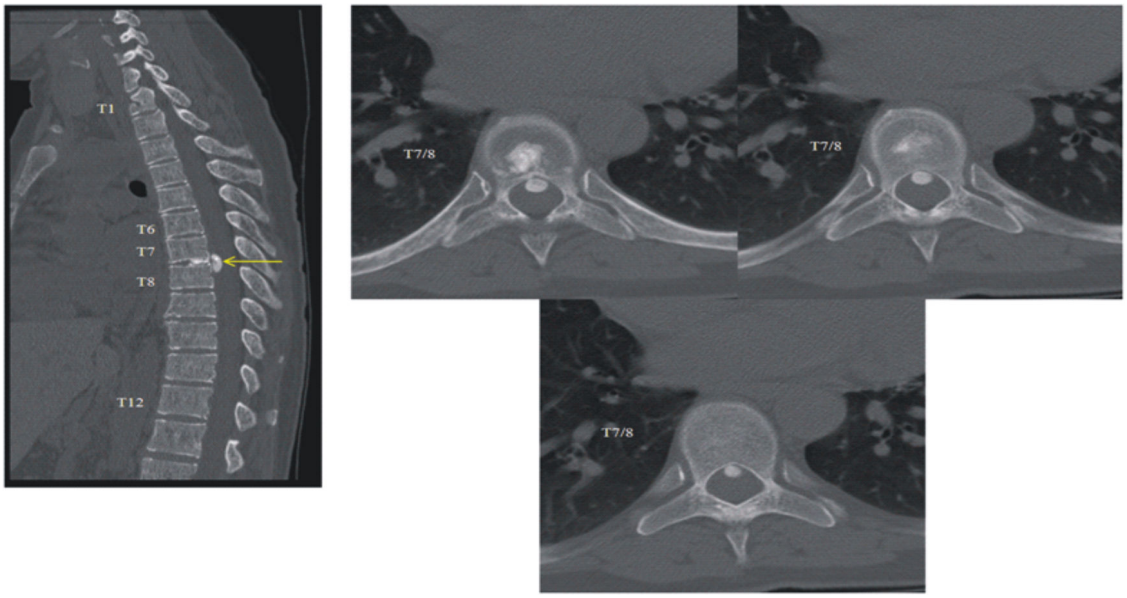
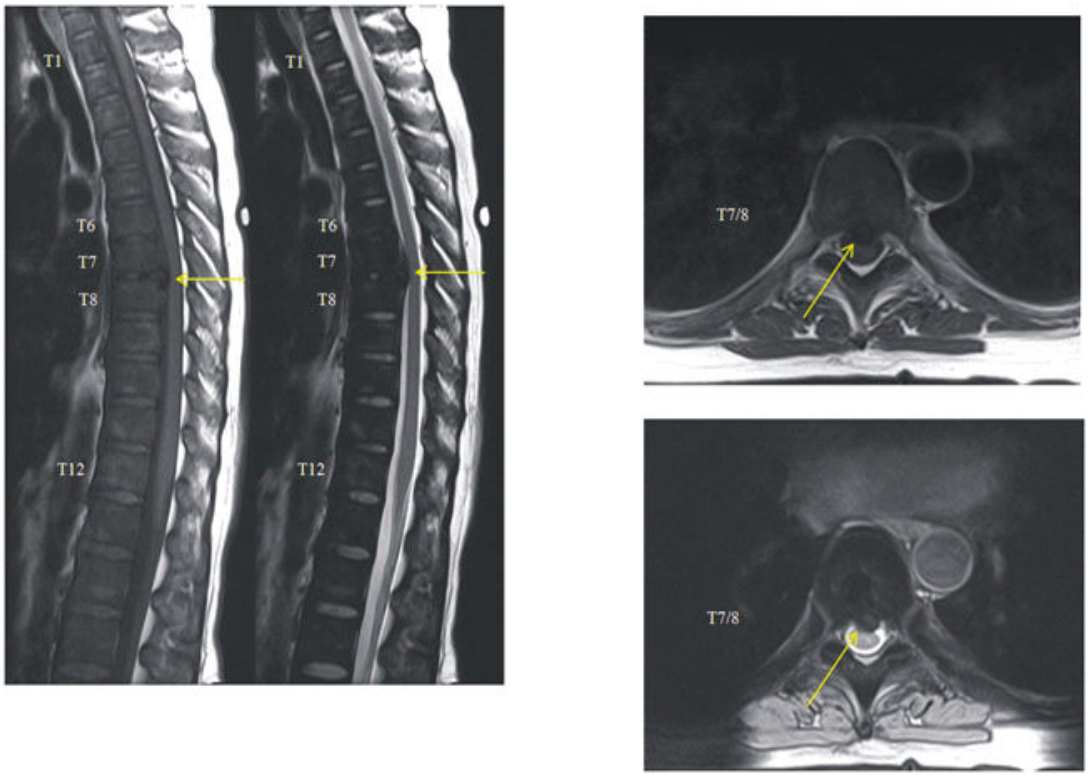


Figure 4: Non Contrast Magnetic Resonance Imaging One Week Later Confirmed the Calcified Extruded Disc at T7-8 not Significantly Changed from the CT. Again, there is Degenerative, Non Calcified Signal at T6-7 Unchanged Since the MRI 8 Weeks Prior. Axial Images Demonstrate the Calcified Central Protrusion which Indents the Ventral Cord without Significant Cord Compression or Abnormal Signal



T6-7 disc herniation (Figure 4) localization of levels was confirmed by counting of the first and last ribs.

Because this new herniation was smaller than before and she remained neurologically intact, we decided in favor of close observation.

Methods and Results

Our initial reaction on seeing the new imaging was that it was a spinal level localization error, but repeated counting of the levels utilizing the first and last ribs as reference confirmed that the level of pathology had indeed changed from T6-7 to T7-8.

Spontaneous regression of disc herniation is well known to neurosurgeons. This, however, happens more commonly with soft disc herniations where the protruding nucleus pulposus either degenerates or is resorbed over time, leading to remission of symptoms. Large calcified disc herniations are very unlikely to resorb, especially in the thoracic spine. In a series of 858 patients, Martinez-Quinones *et al* found that 37 soft disc herniations regressed with time and only one of them was in the thoracic spine.[2]

There is only one well-documented (by CT and MRI) case in English literature of spontaneous regression of a symptomatic calcified thoracic disc herniation.[3] This patient was a 36-year-old female who was initially thought to have a calcified thoracic meningioma but was later presumed to have a herniated disc due to spontaneous regression of most of the lesion and concurrent improvement in symptoms.

Anemia itself is theorized to precipitate degenerative changes in the intervertebral discs. Additionally, the anemia necessitates multiple blood transfusions leading to excessive iron deposition which causes the production of damaging free radicals which accelerate degenerative disc disease. Spinal abnormalities are known to occur in patients with thalessemia, hemochromatosis and other iron excess states, as well as a result of iron chelation therapy.[1,6-8] Aessopos *et al*[6] reported inter-vertebral disc calcification (IDC) in seven of the thirty (23.33%) beta-thalessemia intermedia patients. The patients with IDC had more back pain but otherwise had similar laboratory parameters (of iron excess, extramedullary hematopoesis, etc.) compared to those without IDC. They suggested the possibility of early iron chelation therapy in order to prevent complications from IDC.

Several authors have investigated the causes of back pain in thalessemia. Symptoms have been ascribed to expansion of bone marrow causing pressure on cortical bone, scoliosis, disc degeneration, osteoporotic compression fractures and extramedullary hematopoiesis, which may result in spinal-cord compression, IDC and deferoxamine induced bone dysplasia.[1,9-11] Hartkamp *et al*[7] reported spinal deformities in 22 beta-thalessemia patients treated with desferoxamine.

Various theories have been put forward for spontaneous regression of disc herniations. These include re-accommodation of the herniated nucleus pulposus in the disc space, dehydration and desiccation of the disc, inflammatory resorbtion of the herniated material, pressure from cerebrospinal fluid pulsations and role of epidural venous plexus in its resorbtion.[2,12] In comparison to a rare occurrence in adults, idiopathic IDC in children tends to regress in 61% of cases. This is attributed to the richer vascular supply of the pediatric intervertebral disc and a more active metabolism.

Conclusions

It is possible that the patient in this case, at age 38 years, may still benefit from some of these qualities. Also, in this case, it could be postulated that herniation of the IDC facilitated its resolution in keeping with the above theory of resorbtion. In a period of two years, the herniated IDC at T6-7 disappeared,

while the originally non-herniated IDC at T7-8 persisted and subsequently herniated. This theory could be tested with delayed follow up imaging in approximately 18 months.

References

1. Desigan S, Hall-Craggs MA, Ho CP, *et al*. Degenerative disc disease as a cause of back pain in the thalassaemic population: a case-control study using MRI and plain radiographs. *Skeletal Radiol.* 2006; 35: 95-102.
2. Martínez-Quiñones JV, Aso-Escario J, Consolini F, *et al*. [Spontaneous regression from intervertebral disc herniation. Propos of a series of 37 cases]. *Neurocirugia (Astur).* 2010; 21: 108-117.
3. Piccirilli M, Lapadula G, Caporlingua F, *et al*. Spontaneous regression of a thoracic calcified disc herniation in a young female: a case report and literature review. *Clin Neurol Neurosurg.* 2012; 114: 779-781.
4. Coevoet V, Benoudiba F, Lignières C, *et al*. [Spontaneous and complete regression in MRI of thoracic disk herniation]. *J Radiol.* 1997; 78: 149-151.
5. Morandi X, Crovetto N, Carsin-Nicol B, *et al*. [Spontaneous disappearance of a thoracic disc hernia]. *Neurochirurgie.* 1999; 45: 155-159.
6. Aessopos A, Tsironi M, Polonifi K, *et al*. Intervertebral disc calcification in thalassemia intermedia. *Eur J Haematol*. 2008; 80: 164-167.
7. Hartkamp MJ, Babyn PS, Olivieri F. Spinal deformities in deferoxamine-treated homozygous beta-thalassemia major patients. *Pediatr Radiol.* 1993; 23: 525-528.
8. Olivieri NF, Brittenham GM. Iron-chelating therapy and the treatment of thalassemia. *Blood*. 1997; 89: 739-761.
9. Angastiniotis M, Pavlides N, Aristidou K, *et al*. Bone pain in thalassaemia: assessment of DEXA and MRI findings. *J Pediatr Endocrinol Metab.* 1998; 11: 779-784.
10. Papavasiliou C, Gouliamos A, Vlahos L, *et al*. CT and MRI of symptomatic spinal involvement by extramedullary haemopoiesis. *Clin Radiol.* 1990; 42: 91-92.
11. Aydingöz U, Oto A, Cila A. Spinal cord compression due to epidural extramedullary haematopoiesis in thalassaemia: MRI. *Neuroradiology*. 1997; 39: 870-872.
12. Wood KB, Blair JM, Aepple DM, *et al*. The natural history of asymptomatic thoracic disc herniations. *Spine (Phila Pa 1976).* 1997; 22: 525-529; discussion 529-530.