Case Report

Spinal cord compression in beta-thalassemia: case report and review of the literature

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Study design: A case report of thoracic spinal cord compression in a 34-year-old male with beta-thalassemia is reported.

Objectives: In patients with thalassemia, neurologic complaints should lead to a high index of suspicion for spinal cord compression from marrow expansion, ectopic bone formation and resultant stenosis. Initial presentation, diagnosis, radiographic findings, surgical treatment and follow-up are reviewed.

Setting: This case is reported from Chicago, Illinois.

Method: A chart review is performed for the purposes of this case report.

Results: Patient underwent decompressive laminectomy with good surgical outcome.

Conclusion: Rapid diagnosis and treatment of such a condition is essential to optimize the chances of recovery.

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Introduction

Thalassemia is a hematological disorder in which the red blood cells (RBCs) are formed and destroyed secondary to a defect in the proper synthesis of the globin moiety of hemoglobin. People of Mediterranean descent are at particular risk for such a condition. There is a broad range of symptom complexes in patients with thalassemia. The risk of neurologic compromise in this disease is in part due to the fact that extramedullary hematopoiesis within the spinal canal can result in the compression of neural structures.¹ There have only been a few reports in the literature describing spinal cord compression by such a disease process.^{1–37} We present a case of a patient with thalassemia with thoracic cord compression and progressive myelopathy. The clinical syndrome, radiographic findings, pathophysiology and treatment are reviewed.

Case report

A 34-year-old male of Greek descent presented with a 12-month history of pain in the mid-thoracic region, paresthesia in his feet, progressive difficulty with ambulation and increased frequency of urination with urgency. In the 5 days prior to evaluation, he developed

severe weakness in his lower extremities resulting in the ability to ambulate only with assistance. His difficulty with ambulation marginally improved after a blood transfusion. His hemoglobin count was found to be 7.1 g/dl at the time of his presentation.

Physical examination demonstrated a young male, awake and oriented to person, place and time. His cranial nerve examinations 2–12 were normal. His motor strength in all extremities was 5/5. On sensory testing, he had decreased sensation to pinprick and proprioception in the lower extremities. Reflexes disclosed lower extremity reflex disinhibition, a Babinski response and clonus bilaterally. Rectal tone was normal. His gait was spastic.

MRI of the neuro axis demonstrated severe spinal cord compression from T2 to T11 due to an extramedullary mass, which arose posterior to the cord in the posterior elements of the thoracic vertebrae and epidural space. The signal intensity within the cord also demonstrated early changes consistent with myelomalacia (Figure 1a–c). He also had nerve root compression by the same disease process in the lumbar–sacral junction.

The patient was admitted for medical evaluation and stabilization, and he was started on steroids (dexamethasone). The patient also underwent a transfusion to raise his hemoglobin from 7.1 to 9.8 g/dl to maintain cord perfusion during the anticipated surgery blood loss.

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Figure 1 (a) Sagittal MRI of the thoracic spine showing the spinal cord compression (white arrow). (b) Axial MRI of the mid-thoracic level demonstrating the amount of cord displacement by the extramedullary mass (white arrow). (c) Axial CT of the mid-thoracic spine region. Note the overgrowth of the marrow in response to anemia as a compensatory mechanism (white arrow). (d) Pathology slide of the bone marrow in the lamina of the thoracic spine showing fibroadipose tissue, extramedullary hematopoiesis with erythroid hyperplasia. The inset shows the deposits of foamy histiocytes

This elevation in hemoglobin again resulted in a subjective improvement of his motor strength. The patient had multiple blood antibodies from prior transfusions. This required several days to crossmatch blood for the surgery.

A thoracic laminectomy to decompress the spinal canal was planned. As the decompression was limited to the thoracic spine and spared the cervical and lumbar junctions, stabilization was not felt to be necessary. The patient was placed in a prone position and the mean

arterial blood pressure was maintained at 90 mmHg or greater by volume expansion to maintain spinal cord perfusion. He was also placed on methylprednisolone protocol, normally used for acute spinal cord injury, intraoperatively. The patient did not require any intraoperative vasopressors to keep the mean blood pressure elevated. A standard thoracic laminectomy was performed from T2 to T11 with a high speed drill.

The laminae were grossly thickened due to the expansion of the cancellous portion of the bone. After the laminectomy, a thinned yellow ligament was removed. The epidural fat was found to be replaced by ectopic hematopoietic tissue resembling cancellous bone. This maintained spinal cord compression post laminectomy and was removed with a Kerrison punch. Hemostasis was achieved with a bipolar cautery. All posterior elements and nerve root exit zones were thoroughly decompressed. Care was taken not to injure the facets or joint capsules. After the decompression, the dura was pulsatile. Gelfoam was placed for hemostasis and two large Jackson–Pratt drains were left in the wound to prevent cord compression secondary to epidural blood accumulation.

The final pathological diagnosis showed markedly hypercellular bone marrow with marked erythroid hyperplasia. This was associated with deposits of sheets of foamy cells without associated bony sclerosis. The epidural mass was composed of soft tissue with extramedullary hematopoiesis featuring the same characteristics mentioned above. The overall histomorphology was that of hyperplastic bone marrow with erythroid hyperplasia associated with deposits of foamy histiocytes, which was also occurring in adjacent soft tissue in the form of extramedullary hematopoiesis (Figure 1d).

Postoperatively, the patient underwent a 7-day steroid taper. He received 2 additional units of packed red blood cells in the postoperative period. He was mobilized 1 day following surgery. His major postoperative complaint related to ileus and incisional pain. He had full motor strength in all his extremities and a remarkably improved gait. No change in bladder function was noted. The postoperative MRI showed complete decompression of the spinal cord (Figure 2a, b).

On 3 months follow-up, the patient improved compared to his preoperative status. He could ambulate independently, had no difficulty with urination, had normal strength and did not require narcotics for pain relief.

Discussion

Gatto³⁷ was the first to describe spinal cord compression from the extramedullary overgrowth of the hematopoietic tissue. The diagnosis at that time was based on clinical examination and myelography. At present, with the addition of CT and MRI, the physician's repertoire for diagnosis is much improved. Characteristic findings

Figure 2 (a) Postoperative MRI of the thoracic spine. Observe the resolution of the cord compression. (b) The axial MRI of the mid-thoracic spine showing the resolution of the cord compression

of thickened lamina, extramedullary marrow tissue and cord compression are easily identified. In the 12 thalassemic patients studied by Issargisil *et al*,¹ the

male-to-female ratio for spinal cord compression by extramedullary mass was 5:1. Thoracic cord compression was seen primarily in the lower thoracic spine.

Extramedullary hematopoiesis occurs in multiple blood disorders. The usual organ involvement includes the liver, spleen, bone marrow and lymph nodes. The onset of neurologic symptoms in a patient with such underlying blood dyscrasias should prompt a high clinical suspicion for cord compression or thecal sac compression by an extramedullary hematopoietic process.

The treatment modalities available to such patients are limited. Intervention options have included multiple blood transfusions to downregulate erythropoietin production, radiation therapy to halt the production of overgrown marrow tissue, surgical decompression or a combination of any of the above.^{1,26} The relative benefit of one treatment over another has not been clearly established due to the infrequency of this disorder.

The risks of surgical treatment include excessive bleeding in a patient with a low hemoglobin and difficulty in transfusion due to antibodies and cardio pulmonary stress. The benefit of surgery includes immediate resolution of compression and its symptoms upon decompression. Surgical decompression also provides a good histologic diagnosis. Lau *et al*²⁶ report the case of a 28-year-old woman with cord compression in the thoracic spine who underwent surgical decompression with immediate postoperative recovery of weakness. A complete resolution of symptoms had occurred at 2 months follow-up. Multiple transfusions were also required to maintain the hemoglobin above 10 g/dl.

The role of transfusion is typically an adjunctive therapy while waiting for a more definitive treatment. It is used in the perioperative period to maintain perfusion.^{1,13} Parsa and Oriezy³⁰ argues that transfusion therapy can be used as a diagnostic tool and the sole treatment modality for patients with thalassemia. They describe the case of a patient who was treated for cord compression with a 4-year follow-up with no need for further therapy. The major risk of transfusion is the inherent risk of infectious disease transfer, iron overload, formation of antibodies and other such problems.

We believe that transfusion is a poor primary treatment as it is increasingly unrealistic and difficult to transfuse patients. As such, it is our opinion that transfusion can be used only as an adjunctive treatment modality.

The risks of radiotherapy in the treatment of cord compression in such patients include the lack of any tissue for histologic diagnosis and the risks involved with radiation exposure. The benefits include ready availability, effectiveness in the resolution of symptoms in a short period of time and reduction of local recurrence.¹⁷ Abassioun and Amir-Jamshidi⁸ in 1982 reported the case of a 15-year-old female who was paraparetic with long tract signs. The patient's symptoms resolved after 1500 cGy of radiation in five treatments with only residual right sustained clonus. Kaufman *et al*¹⁷ also reported two patients with thalassemia who underwent radiation therapy with resolution of symptoms in 3–7 days. Singhal *et al*²¹ in a review of the literature argue that radiation therapy should be the primary modality for treatment, and surgical intervention and transfusion should be reserved for the recurrent cases postradiation. The radiation dosage used mostly in different treatment protocols included a range between 1000 and 3000 cGy.

Cross *et al*,³⁶ Luyendijk *et al*⁵ and Shin *et al*,²⁷ each report patients with cord compression who underwent combined surgical decompression and radiation therapy with good results. In each of these studies, it has been argued that local recurrence may be decreased using radiation therapy to accompany spinal decompression.

Our patient demonstrated only partial response to transfusion. The spinal cord had evidence of injury and myelomalacia on MRI. Given advanced and progressive findings and active progression, we do not believe that transfusion alone was a realistic treatment option. Radiation exposes an already damaged cord to further potential injury. As such, we feel that such cases are best treated by a surgical decompression. Radiation can be saved as a salvage procedure after a wound is healed if the patient has residual or recurrent disease.

A thorough medical search of the Medline describing different hemoglobinopathies and their spinal cord consequences is summarized in Table 1.

Table 1 Different hemoglobinopathies and their spinal cord consequences

Ref.	Year	Study	Age (years)	Sex	Diagnosis	Presentation	Treatment	Outcome
5	1975	Luyendijk <i>et al</i>	42	M	Thalassemia	Paraparesis	Surgery/XRT	Partial recovery
2	1978	Cromwell and Kerber	36	M	Angiogenic myeloid metaplasia	Paraparesis	XRT/steroids exchange	Partial recovery
3	1979	Stahl <i>et al</i>	54	M	Myeloid metaplasia	Paraparesis	Transfusions	Death
4	1980	Rice <i>et al</i>	68	M	Polycythemia vera	Paraplegia	Surgery/XRT/steroids	No recovery
5	1980	Issargisil <i>et al</i>	24	M	Thalassemia	Paraparesis	Transfusions	Partial recovery
	1980	Issargisil <i>et al</i>	22	M	Thalassemia	Paraparesis	XRT	Partial recovery
	1980	Issargisil <i>et al</i>	34	M	Thalassemia	Paraparesis	XRT/steroids	Partial recovery
	1980	Issargisil <i>et al</i>	37	M	Thalassemia	Paraparesis	Transfusions	Partial recovery

Table 1 (continued)

Ref.	Year	Study	Age (years)	Sex	Diagnosis	Presentation	Treatment	Outcome
	1980	Issargisil <i>et al</i>	19	F	Thalassemia	Paraparesis	XRT	Partial recovery
	1980	Issargisil <i>et al</i>	32	M	Thalassemia	Paraparesis	XRT	Partial recovery
	1980	Issargisil <i>et al</i>	25	F	Thalassemia	Paraparesis	XRT	Partial recovery
	1980	Issargisil <i>et al</i>	26	M	Thalassemia	Paraparesis	XRT	Partial recovery
	1980	Issargisil <i>et al</i>	40	M	Thalassemia	Paraparesis	XRT	Partial recovery
	1980	Issargisil <i>et al</i>	17	M	Thalassemia	Paraparesis	XRT/transfusions	Partial recovery
	1980	Issargisil <i>et al</i>	29	M	Thalassemia	Paraparesis	Surgery/XRT	Partial recovery
	1980	Issargisil <i>et al</i>	23	M	Thalassemia	Paraparesis	XRT/transfusions	Partial recovery
6	1980	Oustwani <i>et al</i>	70	M	Polycythemia	Paraplegia	Surgery	No recovery
7	1981	Ahmed <i>et al</i>	17	M	Thalassemia	Paraparesis	Surgery	Partial recovery
8	1982	Abassioun and Amir-jamshidi	19	M	Thalassemia	Paraparesis	Surgery	Full recovery
	1982	Abassioun and Amir-jamshidi	19	M	Thalassemia	Paraparesis	Surgery	Partial recovery
9	1982	Luitjes <i>et al</i>	55	M	Thalassemia	Paraparesis	Surgery/XRT	Partial recovery
10	1982	Heffez <i>et al</i>	72	M	Thalassemia	Paraparesis	Surgery/XRT	Partial recovery – unrelated death.
11	1984	Lewkow and Shah	43	M	Sickle cell anemia	Paraplegia	Surgery/XRT / transfusions	No recovery
12	1985	Price and Bell	74	F	Myelofibrosis	Back pain	Surgery/XRT	Full recovery
13	1987	Mann <i>et al</i>	21	M	Thalassemia	Paraparesis	Surgery	Full recovery
14	1988	Jackson <i>et al</i>	29	M	Thalassemia	Paraparesis	XRT	Full recovery
15	1989	Jackson and Burton	52	M	Polycythemia vera	Paraplegia	XRT	Partial recovery
16	1990	Papavasiliou <i>et al</i>	22	M	Thalassemia	Paraparesis	XRT	Partial recovery
	1990	Papavasiliou <i>et al</i>	26	M	Thalassemia	Paraparesis	XRT	Partial recovery
	1990	Papavasiliou <i>et al</i>	34	M	Sickle cell anemia	Back pain	XRT	Full recovery
	1990	Papavasiliou <i>et al</i>	24	M	Thalassemia	Paraparesis	XRT	Full recovery
	1990	Papavasiliou <i>et al</i>	32	F	Thalassemia/ pregnancy	Paraparesis	No therapy	No outcome listed
17	1991	Kaufman <i>et al</i>	41	F	Thalassemia	Paraparesis	XRT	Partial recovery
		Kaufman <i>et al</i>	40	F	Thalassemia	Paraparesis	XRT/steroids	Partial recovery
18	1991	Singounas <i>et al</i>	32	F	Thalassemia	Paraparesis	Transfusions	Full recovery
19	1991	Hassoun <i>et al</i>	22	M	Thalassemia	Paraparesis	XRT	Full recovery
20	1991	Amir-jamshidi <i>et al</i>	16	M	Thalassemia	Paraparesis	Surgery	Full recovery
	1991	Amir-jamshidi <i>et al</i>	14	M	Thalassemia	Paraparesis	Surgery	Full recovery
21	1991	Singhal <i>et al</i>	21	M	Thalassemia	Paraparesis	Surgery/XRT	Partial recovery
	1991	Singhal <i>et al</i>	33	M	Thalassemia	Paraparesis	XRT	Partial recovery
22	1992	Pantongrag-Brown and Suwanwela	27	M	Thalassemia	Paraparesis	XRT	Partial recovery
23	1993	McDonald <i>et al</i>	67	M	Myelofibrosis	Paraparesis	XRT	Partial recovery
24	1993	De Klippel <i>et al</i>	61	M	Myelofibrosis	Paraparesis	Surgery/XRT/ steroids	Full recovery– unrelated death
25	1993	Dore <i>et al</i>	30	M	Thalassemia	Paraparesis	XRT	Partial recovery
	1993	Dore <i>et al</i>	39	M	Thalassemia	Paraparesis	XRT	Partial recovery
26	1994	Lau <i>et al</i>	28	F	Cooley's anemia	Paraparesis	Surgery	Full recovery
27	1994	Shin <i>et al</i>	22	M	Thalassemia	Myelopathy	Surgery/XRT	Full recovery
28	1994	Cook and Sharp	67	M	Myelofibrosis	Paraparesis	Surgery/XRT	Partial recovery
29	1995	Gouliamos <i>et al</i>	20	M	Thalassemia	Back pain	XRT	Full recovery
30	1995	Parsa and Oriezy	16	F	Thalassemia	Paraparesis	XRT/transfusions	Partial recovery
	1995	Parsa and Oriezy	39	M	Thalassemia	Paraparesis	Transfusions	Partial recovery
31	1996	de Morais <i>et al</i>	46	M	Polycythemia vera	Paraplegia	Surgery/ phlebotomy	No recovery– unrelated death
32	1997	Guerhazi <i>et al</i>	36	M	Myelofibrosis	Paraparesis	XRT/steroids	Partial recovery
33	1997	Aydingoz <i>et al</i>	27	M	Thalassemia	Paraparesis	XRT	Partial recovery
34	1998	Munn <i>et al</i>	35	M	Thalassemia	Paraparesis	XRT	Partial recovery
35	2001	Haran and Ni	68	M	Polycythemia vera	Paraparesis	XRT	Partial recovery

Conclusion

Extramedullary erythropoiesis is a natural tissue response to a low hemoglobin count in patients with thalassemia. This may cause neurologic conditions such as spinal cord compression. Rapid diagnosis and treatment is important to prevent further neurological injury and improve functional outcome in such patients. Surgical decompression and radiation therapy are both effective in treating the compression. Transfusion can also transiently ameliorate the symptoms.

Given the infrequency of the disease and rare symptomatic cord compression, it is unlikely that a randomization of radiation versus surgical treatment could be carried out in an individual institution. This requires a multi-center randomized study. By reviewing the literature, we conclude that in patients with early spinal cord compression, radiation or surgery can be equally effective. However, we prefer immediate decompression with radiation as a salvage in younger patients with signal changes within their cord to reduce the long-term effects of radiation on the neural tissues.

References

- 1 Issargisil S, Piankijagum A, Prawase W. Spinal cord compression in thalassemia. Report of 12 cases and recommendations for treatment. *Arch Intern Med* 1981; **141**: 1033–1036.
- 2 Cromwell LD, Kerber C. Spinal cord compression by extramedullary hematopoiesis in myeloid metaplasia. *Radiology* 1978; **128**: 118.
- 3 Stahl SM, Ellinger G, Baringer JR. Progressive myelopathy due to extramedullary hematopoiesis: case report and review of the literature. *Ann Neurol* 1979; **5**: 485–489.
- 4 Rice GP, Assis LJ, Barr RM, Ebers GC. Extramedullary hematopoiesis and spinal cord compression complicating polycythemia rubra vera. *Ann Neurol* 1980; **7**: 81–84.
- 5 Luyendijk W, Went L, Schaad HD. Spinal cord compression due to extramedullary hematopoiesis in homozygous thalassemia. Case report. *J Neurosurg* 1975; **42**: 212–216.
- 6 Oustwani MB, Kurtides ES, Christ M, Ciric I. Spinal cord compression with paraplegia in myelofibrosis. *Arch Neurol* 1980; **37**: 81–84.
- 7 Ahmed F, Tobin MS, Cohen DF, Gomez-Leon G. Beta Thalassemia. Spinal cord compression. *NY State J Med* 1981; **81**: 1505–1508.
- 8 Abassioun K, Amir-Jamshidi A. Curable paraplegia due to extradural hematopoietic tissue in thalassemia. *Neurosurgery* 1982; **11**: 804–807.
- 9 Luitjes WF, Braakman R, Abels J. Spinal cord compression in a new homozygous variant of beta-thalassemia. *J Neurosurg* 1982; **57**: 846–848.
- 10 Heffez DS, Sawaya R, Udvarhelyi GB, Mann R. Spinal epidural extramedullary hematopoiesis with cord compression in a patient with refractory sideroblastic anemia. Case report. *J Neurosurg* 1982; **57**: 399–406.
- 11 Lewkow LM, Shah I. Sick cell anemia and epidural extramedullary hematopoiesis. *Am J Med* 1984; **76**: 748–751.
- 12 Price F, Bell H. Spinal cord compression due to extramedullary hematopoiesis. Successful treatment in a patient with longstanding myelofibrosis. *JAMA* 1985; **253**: 2876–2877.
- 13 Mann KS et al. Paraplegia due to extramedullary hematopoiesis in thalassemia. Case report. *J Neurosurg* 1987; **66**: 938–940.
- 14 Jackson Jr DV, Randall ME, Richards F. Spinal cord compression due to extramedullary hematopoiesis in thalassemia: long term follow-up after radiotherapy. *Surg Neurol* 1988; **29**: 389–392.
- 15 Jackson A, Burton IE. Retroperitoneal mass and spinal cord compression due to extramedullary haemopoiesis in polycythemia rubra vera. *Br J Radiol* 1990; **42**: 91–92.
- 16 Papavasiliou C et al. CT and MRI of symptomatic spinal involvement by extramedullary hematopoiesis. *Clin Radiol* 1990; **42**: 91–92.
- 17 Kaufman T, Coleman M, Giardina P, Nisce LZ. The role of radiation therapy in management of hematopoietic neurologic complications in thalassemia. *Acta Haematol* 1991; **85**: 156–159.
- 18 Singounas EG et al. Paraplegia in a pregnant thalassemic woman due to extramedullary hematopoiesis: successful management with transfusions. *Surg Neurol* 1991; **36**: 210–215.
- 19 Hassoun H et al. Spinal cord compression secondary to extramedullary hematopoiesis: a noninvasive management based on MRI. *Am J Hematol* 1991; **37**: 201–203.
- 20 Amir-jamshidi A, Abbassioun K, Ketabchi SE. Spinal extradural hematopoiesis in adolescents with thalassemia. Report of two cases and a review of the literature. *Childs Nerv Syst* 1991; **7**: 223–225.
- 21 Singhal S et al. The role of radiation therapy in the management of spinal cord compression due to extramedullary hematopoiesis in thalassemia. *J Neurol Neurosurg Psychiatry* 1992; **55**: 310–312.
- 22 Pantongrag-Brown L, Suwanwela N. Case report: chronic spinal cord compression from extramedullary haematopoiesis in thalassemia – MRI findings. *Clin Radiol* 1992; **46**: 281–283.
- 23 McDonald AC, Cook G, Sharp RA, Bissett D. Spinal cord compression in myelofibrosis – a case report. *Acta Oncol* 1993; **32**: 692–693.
- 24 De Klippel N et al. Progressive paraparesis due to thoracic extramedullary hematopoiesis in myelofibrosis. Case report. *J Neurosurg* 1993; **79**: 125–127.
- 25 Dore F et al. Recurrence of spinal cord compression from extramedullary hematopoiesis in thalassemia intermedia treated with low doses of radiotherapy. *Am J Hematol* 1993; **44**: 148.
- 26 Lau SK, Chan CK, Chow YY. Cord compression due to extramedullary haemopoiesis in a patient with thalassemia. *Spine* 1994; **19**: 2467–2470.
- 27 Shin KH et al. Combined radiotherapeutic and surgical management of a spinal cord compression by extramedullary hematopoiesis in a patient with hemoglobin E beta-thalassemia. *Acta Haematol* 1994; **91**: 154–157.
- 28 Cook G, Sharp RA. Spinal cord compression due to extramedullary haemopoiesis in myelofibrosis. *J Clin Pathol* 1994; **47**: 464–465.
- 29 Gouliamos AD et al. Case report: magnetic resonance imaging of spinal cord compression in thalassemia before and after radiation treatment. *Clin Radiol* 1995; **50**: 504–505.
- 30 Parsa K, Oriezy A. Nonsurgical approach to paraparesis due to extramedullary hematopoiesis. Report of two cases. *J Neurosurg* 1995; **82**: 657–660.

- 31 de Morais JC *et al.* Spinal cord compression due to extramedullary hematopoiesis in the proliferative phase of polycythemia vera. *Acta Haematol* 1996; **96**: 242–244.
- 32 Guemarazi A, Miaux Y, Chiras J. Imaging of spinal cord compression due to thoracic extramedullary hematopoiesis in myelofibrosis. *Neuroradiology* 1997; **39**: 733–736.
- 33 Aydingoz U, Oto A, Cila A. Spinal cord compression due to epidural extramedullary haematopoiesis in thalassemia: MRI. *Neuroradiology* 1997; **39**: 870–872.
- 34 Munn RK, Kramer CA, Arnold SM. Spinal cord compression due to extramedullary hematopoiesis in beta-thalassemia intermedia. *Int J Radiat Oncol Biol Phys* 1998; **42**: 607–609.
- 35 Haran M, Ni S. Recurrent reversible paraplegia. *Lancet* 2001; **357**: 1092.
- 36 Cross JN, Morgan OS, Gibbs WN, Cheruvanky I. Spinal cord compression in thalassemia. *J Neurol Neurosurg Psychiatry* 1997; **40**: 1120–1122.
- 37 Gatto I, Terrana V, Biondi L. Comprssione sul midollo spinale da proliferazione di midollo osseo nello spazio epidurale in soggetto affetto da malattia di Colley splenectomizzato. *Haematologica* 1954; **38**: 61–75.