New disease

A kind of specific osteolytic destruction of the vertebral bodies

Baogan Peng,¹ Jinhong Chen,¹ Xiaodong Pang,¹ Yan Hei²¹Department of Spinal Surgery, General Hospital of Armed Police Force, Beijing, China²Department of Pathology, General Hospital of Armed Police Force, Beijing, China

Correspondence to Professor Baogan Peng, pengbaogan@163.com

Summary

This report describes two young patients with osteolytic destruction in two adjacent vertebral bodies along with the intervertebral disc, and reveals its possible mechanism. A lateral radiograph and CT scan displayed a giant osteolytic cavity in the L4 vertebral body. An MRI or CT scan with a two-dimensional reconstruction displayed the same changes in the L4 vertebral body and lower endplate erosion in the L3 vertebral body. A comprehensive preoperative evaluation did not identify a specific cause of vertebral destruction. Both patients underwent anterior lumbar fusion surgery. The lesions were removed for histological and immunohistochemical examination. Histopathological study of the destructed vertebral bodies in the two patients revealed the disruption or atrophy of bone trabeculae with infiltration of a large amount of B-lymphocytes and macrophages into the marrow cavities. Studies of its pathogenesis reveal that it is likely to be a B-lymphocyte-mediated local immune inflammatory reaction in the lumbar spine.

BACKGROUND

Osteolytic destruction of vertebral bodies, when demonstrated, is commonly associated with the pathological process of spine tumours and infection.¹ Avascular necrosis of the vertebral body is another type of bone destruction that occurs as a result of interruption of the rich blood supply necessary for haematopoietic marrow production.² Avascular necrosis is an uncommon sequela of vertebral collapse that is commonly secondary to osteoporotic compression fracture.^{1 3-8} We report two cases with osteolytic destruction in two adjacent vertebral bodies along with the intervertebral disc. The lesion of osteolytic destruction started in the L4 vertebral body and advanced further from the L3/4 disc to the L3 vertebral body. We ruled out the possibility of spine tumours or tumour-like lesions, metabolic disorders, genetic disorders, infection and avascular necrosis. To the best of our knowledge, no lesion of this type has been reported previously.

CASE PRESENTATION

Two patients visited the authors' hospital because of severe low back pain. Case 1, a 26-year-old man, presented with a 3-year history of low back pain that became increasingly aggravated for 1 year. Unresponsive to conservative therapies, the back pain seriously affected his normal life. Case 2, a 22-year-old woman, presented with symptoms of low back pain for 1 year and 6 months. Low back pain got aggravated after standing and walking for a long time, and did not relieve after various conservative therapies.

Both patients had neither a history of major illness, trauma, systemic steroid treatment or alcohol abuse before illness, nor a history of long-term febrile and night sweat during the course of illness.

INVESTIGATIONS

Physical examination revealed a stiff lumbar spine with a limited range of flexion, extension and lateral bending.

Tenderness and percussion pain were noted over the L3–L4 lumbar spine. Muscle strength, dermatomal sensation and deep tendon reflexes in the lower extremities were all normal. After admission, imaging examinations including lumbar plain radiography, CT, MRI, whole-body bone scan and chest clairvoyance were carried out, together with laboratory examinations including blood cell count, liver enzymes, erythrocyte sedimentation rate, C-reactive protein, HLA-B27, rheumatoid factor, serum tumour markers such as carcinoembryonic antigen and α -fetoprotein, and urine Bence-Jones protein. A tuberculin test was performed on both patients.

No abnormality was detected through chest clairvoyance and abdominal ultrasonic examination in either patient. Laboratory results were either negative or within normal range. Blood cultures were all negative. Tuberculin test results were also negative in both cases.

In case 1, the plain lateral radiograph and CT scan of the lumbar spine displayed a giant osteolytic cavity without a sclerotic margin in the L4 vertebral body (figure 1A). The CT scan with a two-dimensional reconstruction showed osteolytic destruction in the upper half of the L4 vertebral body and inferior endplate erosion in the L3 vertebral body (figure 1B). The lesion was associated with low signal intensity on MR T1-weighted images and high signal intensity on MR T2-weighted images in the L4 vertebral body. A whole-body isotope bone scan demonstrated a high uptake in the L3 and L4 vertebral bodies, especially in the L4 vertebral body. Under suspicion that the huge bone destruction in the L4 vertebral body was caused by the obstruction of L4 lumbar arteries, a transfemoral artery intra-arterial digital subtraction angiography was carried out, which revealed a pair of normal L4 lumbar arteries (figure 1C).

In case 2, the plain lateral radiograph and CT scan of the lumbar spine showed the same change as in case 1 (figure 2A). The mid-sagittal MR T1-weighted and T2-weighted

Figure 1 Case 1: a 26-year-old man. (A) Lateral radiograph of the lumbar spine showing a well-demarcated sharply circumscribed cystic lesion of the L4 vertebral body (arrow). (B) Two-dimensional reconstructive CT scan showing osteolytic bone destruction in the upper half of the L4 vertebral body and lower endplate erosion of the L3 vertebral body (arrow). (C) Intra-arterial digital subtraction angiography demonstrating two pairs of normal L4 and L5 lumbar arteries.

images of the lumbar spine showed marked bone destruction in the upper three-quarters of the L4 vertebral body (figure 2B). The left sagittal MR images of the lumbar spine showed that the inferior endplate of the L3 vertebral body was also involved (figure 2C). A whole-body isotope bone scan demonstrated a high uptake in the L3 and L4 vertebral bodies, especially in the L4 vertebral body.

DIFFERENTIAL DIAGNOSIS

The differential diagnostic considerations for the osteolytic destruction of vertebral bodies in our two cases are spine tumour or tumour-like lesions, metabolic disorders, genetic disorders, infection and avascular necrosis.

TREATMENT

The two patients underwent anterior lumbar interbody fusion with bone graft and internal fixation because of

both severe low back pain unresponsive to various conservative therapies and uncertain diagnosis under routine examination. The lesion was removed in a piece-meal fashion. Case 1 underwent an L3–L4 intervertebral body fusion with an autologous iliac bone graft using an anterior retroperitoneum approach. Case 2 underwent the same surgery as case 1, but an autologous iliac bone graft-filled titanium cage was used for the interbody fusion.

OUTCOME AND FOLLOW-UP

Low back pain symptoms of case 1 disappeared immediately after the operation. Successful bone fusion between the L3 and L4 vertebrae occurred 3 months after the operation. Low back pain did not recur at 4-year follow-up. Low back pain symptoms of case 2 disappeared immediately after the operation, with no recurrence at 2-year follow-up.

Figure 2 Case 2: a 22-year-old woman. (A) Lateral radiograph of the lumbar spine showing a giant, well-demarcated cystic lesion of the L4 vertebral body (arrow). (B) Mid-sagittal MR T2-weighted image showing high signal intensity and bone destruction in the upper three quarters of the L4 vertebral body. (C) Sagittal MR T2-weighted image in the left vertebral margin showing that the lesion is involved with the lower endplate of the L3 vertebral body (arrow).

Figure 3 (A) Histopathological examination showing disruption or atrophy of bone trabeculae with a great deal of inflammatory cell infiltration, fibrosis, disappearance of fat cells and haematopoietic cells within marrow cavities (specimen from case 2, haematoxylin and eosin stain, $\times 100$). (B) Histopathological examination revealing that inflammatory granulation tissue is filled in the osteolytic cavity of the L4 vertebral body (specimen from case 2, haematoxylin and eosin stain, $\times 50$).

During surgery, the lesions were removed for histopathological examination with routine haematoxylin and eosin stain. Immunohistochemical examination was performed to identify the type of infiltrated cell in the specimens of the two contiguous vertebrae and the intervertebral disc. The following primary antibodies were used: mouse anti-macrophage antigens (CD68 and CD163, respectively), T-lymphocyte antigens (CD3 and CD4, respectively) and B-lymphocyte antigen (CD20) (Santa Cruz Bio. Inc., Santa Cruz, California, USA). All these antibodies were diluted to 1:100. Dewaxed sections were treated with pepsin (1.25 g/l) to repair antigens. Immunohistochemical staining was performed using UltraSensitive S-P staining kits (Maxin Company, Sunnyvale, California, USA) according to the recommendations of the producers.

Histopathological study showed identical changes in destructive vertebral bodies and adjacent discs in both patients. The vertebral body in the lesion adjacent to the normal bone showed a great deal of inflammatory cell infiltration, fibrosis, disappearance of fat cells and haematopoietic cells within marrow cavities, disappearance or death of osteocytes within bone trabeculae, and disruption or atrophy of bone trabeculae (figure 3A). The inflammatory granulation tissue was filled in the osteolytic cavity of the L4 vertebral body (figure 3B). In the adjacent

degenerated discs, vascular ingrowth and inflammatory cell infiltration were found. Further immunohistochemical staining demonstrated that large amounts of CD20⁺ B-lymphocytes and CD163⁺ and CD68⁺ macrophages infiltrated into the marrow cavities (figure 4A,B) and the degenerated disc, mainly nucleus pulposus. No CD3⁺ and CD4⁺ T-lymphocytes were found in the marrow cavities and discs.

No microorganisms and malignant cells were found under microscope; besides, Gram, fungal and acid-fast strains and cultures were all negative.

DISCUSSION

The major differential diagnostic consideration for the osteolytic destruction of vertebral bodies in our two cases is spine tumour or tumour-like lesions, including spine metastases, lymphoma, leukaemia, polyostotic fibrous dysplasia, generalised fibromatosis, multiple focal fibrocartilaginous dysplasia, non-ossifying fibromata including Jaffe-Campanacci syndrome, cystic angiomas, and multiple haemangiomas including Gorham's disease.

Spine metastases are most often expansile and commonly associated with soft tissue.⁹ MRI shows homogeneous and diffuse vertebral signal abnormality, a convex vertebral border, pedicle involvement and sparing of the

Figure 4 (A) Immunohistochemical examination showing marked CD68⁺ macrophages infiltrated into marrow cavities (specimen from case 2, S-P stain, original magnification, $\times 200$). (B) Immunohistochemical examination showing marked CD20⁺ B-lymphocytes infiltrated into marrow cavities (specimen from case 2, S-P stain, original magnification, $\times 200$).

intervertebral disc. These clinical and MRI findings are different from those of our patients. Lymphoma may present as a systemic disease with skeletal manifestations or as an isolated bony tumour. When lymphoma involves the spine, it has a destructive nature and can cause collapse of the vertebral body with extraosseous extension canal, which is not evident in our patients. The tumour on radiographs is a lytic lesion with an ill-defined cortex. Percutaneous or open biopsy is desirable to clarify the diagnosis.¹⁰ Leukaemia occurs in children. Lethargy, anaemia and fever occur commonly and often in combination with each other. The peripheral leukocyte count is elevated in 60% of patients, and the erythrocyte sedimentation rate is also elevated. Radiographs may show focal lytic lesions, occasional sclerotic lesions or isolated periosteal reactions. A whole-body isotope bone scan shows no uptake in areas of obvious bony destruction in some cases.¹¹ Fibrous dysplasia is a bone disorder of unknown origin characterised by slow, progressive replacement of normal cancellous bone in the medullary canal with immature fibro-osseous tissue. According to the involvement of one or more bones, it is classified into two forms: monostotic and polyostotic. Fibrous dysplasia rarely occurs in axial bones and is accidentally discovered on radiographic films. Besides, its clinical presentation is usually painless and asymptomatic. Radiographic findings include a matrix 'ground-glass appearance', areas of marginal sclerosis adjacent to lytic regions with a narrow transitional zone, and pathological fractures. A better needle biopsy of the lesion would have facilitated diagnosis.¹² Generalised fibromatosis is part of the spectrum of fibromatoses in infancy and childhood. The lesions are usually multiple and fibrous in nature. Generalised fibromatosis can be limited to the skeleton and rarely manifests as a solitary bone lesion. Multiple osseous lesions often regress without treatment.^{13 14} First described in 1985, focal fibrocartilaginous dysplasia is an uncommon lesion that typically affects the tibia of children younger than 2 years. Histology shows dense fibrous tissue mixed with cartilage. The lesion is self-limiting and the deformity often corrects itself by the age of 6 years.¹⁵ Non-ossifying fibroma is referred to as a fibrous cortical defect when smaller than 3 cm in size, which is the most common type of benign fibrous bone lesion. Sometimes considered to be a developmental defect arising in the trabeculae of tubular bones, non-ossifying fibroma consists of spindle-shaped fibroblasts, multinucleated giant cells and foamy histiocytes. It is most commonly present in skeletally immature patients and its natural course is involution with progressive ossification over several years.¹⁶ The Jaffe-Campanacci syndrome usually occurs in the peripuberty years and its diagnostic stigmata include café au lait macules, skeletal deformities and multiple histologically confirmed non-ossifying fibromas of the long bones.^{17 18} Cystic angiomas involve the axial and proximal appendicular skeleton and presents multiple lytic bone lesions with a honeycomb appearance. The presence of soft-tissue involvement further supports the diagnosis. Histologically, the skeletal lesions of cystic angiomas consist of multiple dilated vascular canals lined with flat endothelial cells.¹⁹ Skeletal haemangiomas are extremely vascular bone tumours, usually involving the spine. MRI is extremely sensitive in detecting spinal

haemangiomas. On both T1-weighted and T2-weighted images, these lesions tend to show increased signal intensity, reflecting the adipose tissue rather than the haemorrhagic component. The hyperintense stroma surrounds foci of very low signal intensity, which represent the thickened vertical trabeculae. Focal fatty marrow replacement is the major differential diagnosis on MRI.²⁰ Gorham's disease is a very rare disorder characterised by uncontrolled, destructive proliferation of vascular or lymphatic capillaries within the bone and surrounding soft tissue. A definitive diagnosis must be established by performing a biopsy of the offending lesion.^{21 22}

The differential diagnosis also includes metabolic disorders, such as hyperparathyroidism with brown tumours and tumour-induced osteomalacia, and rare genetic disorders, such as juvenile Paget's disease and familial expansile osteolysis.

A vertebral brown tumour with a lytic vertebral lesion, a non-neoplastic process resulting from excess osteoclast activity, is found in primary or secondary hyperparathyroidism. Vertebral brown tumours can be easily distinguished from our cases depending on the presence or absence of hyperparathyroidism. A fludeoxyglucose (FDG) positron emission tomography/CT may demonstrate diffuse and multiple foci of increased FDG uptake and a focal uptake in the thyroid region. Study of parathyroid function can reveal high parathyroid hormone levels. Histologically, collections of osteoclasts are intermixed with fibrous tissue and poorly mineralised bone.^{23 24} When tumour-induced osteomalacia involves the spine, vertebral bodies soften and become biconcave with loss of medial height. But, biochemically, patients display significant hypophosphataemia, abnormally low renal tubular maximum resorption of phosphate (hyperphosphaturia) and low or inappropriately calcitriol levels, which make it easy to differentiate from our cases.²⁵ Juvenile Paget's disease is a rare genetic disorder characterised by markedly accelerated bone turnover secondary to enhanced osteoclastic activity, and presents in early childhood. A markedly elevated level of alkaline phosphatase and radiographs revealing characteristic mosaic pattern (both osteolysis and osteosclerosis) are diagnostic of Paget's disease.²⁶ Familial expansile osteolysis is a well-documented autosomal dominant genetic syndrome clinically characterised by early hearing loss, bone pain preceding skeletal abnormalities and dental involvement.²⁷ The lytic lesion shows loss of trabecular pattern and gradual increase in size, and causes cortical thinning. These early changes are later followed by bone expansion, deformity and fractures. Serum alkaline phosphatases and hydroxyproline are elevated.²⁸

Other likely considerations in the differential diagnosis include spine infection and avascular necrosis. According to the clinical presentations, imaging studies and laboratory examinations, infection is easily diagnosed. Vertebral osteomyelitis has a more precipitous presentation with severe pain, high fever and profound clinical symptoms. The organism can be isolated either by a blood culture or from needle aspiration material.^{9 29}

Avascular necrosis has been associated with various risk factors and is classified as secondary or idiopathic. Renal diseases, exposure to steroids and alcohol abuse account for more than 90% of all known associated risk

factors.^{30 31} The aetiology and pathogenesis of non-traumatic avascular necrosis have not been fully elucidated. Alteration of arteriolar and capillary blood flow can be postulated as the final common pathway resulting in ischaemia and ultimately idiopathic avascular necrosis. Avascular necrosis of the vertebral body is thought to be the result of an injury, which is caused by either traumatic or non-traumatic mechanisms, at the anterior segment of the vertebral body in the area of a vascular watershed.³² In compression fractures of an osteoporotic vertebral body with minor trauma, interfragmentary movement in the injured vertebral body bears the risk of non-union and can result in later collapse.³³ One clinical feature of avascular necrosis of the vertebral body is a delayed post-traumatic vertebral collapse, which was initially described by Kummell in 1895.⁴

Neither of our two patients had any significant risk factors for vertebral avascular necrosis. They fell ill early and had no history of trauma. Intra-arterial digital subtraction angiography in case 1 revealed a pair of normal L4 lumbar arteries, which indicates that the aetiology of the lesion in the L4 vertebral body is not avascular necrosis. Our cases are substantially different from those of some previous reports that presented with either idiopathic or secondary avascular necrosis of the vertebral body.^{32–34}

Histopathological study of the destructed vertebral bodies in these patients revealed substantial differences from the changes of avascular necrosis. The main characteristic changes in our patients were the disruption or atrophy of bone trabeculae with infiltration of a large amount of B-lymphocytes and macrophages into the marrow cavities. Chou and Knight³² and Lee *et al*³⁴ showed no histological evidence of inflammation in the avascular necrosis of the vertebral body.

Many studies have demonstrated that the pathological feature of the herniated disc, mainly the sequestration and transligamentous extrusion specimens, is the formation of granulation tissue in the margin of herniation specimens containing many macrophages and a mild degree of T-lymphocyte infiltration.^{35–37} Surrounded by the annulus fibrosus, the nucleus pulposus is normally shielded from the immune system. Once exposed to the circulation system, it will incur immunological reaction.^{38 39} Theoretically, disc herniation into the epidural space and disc exposure to the bone marrow through endplate destruction should share the same pathological mechanism to disc fate immunologically. Marked B-lymphocyte infiltration into the herniation disc has never been reported.

Another marked difference of vertebral destruction on imaging in our patients is involved with two contiguous vertebral bodies and an intervertebral disc. MRI 1 year ago in case 2 did not show erosion of the inferior endplate in the L3 vertebral body. But it occurred on MRI after admission, which indicated that the lesion advanced further from the disc to the superior vertebral body. The extent of destruction in the inferior vertebral body is obviously more severe than that in the superior vertebral body, which also indicates that the inflammatory reaction is attenuated gradually.

On the basis of imaging, histopathological and immunohistochemical studies, we consider that the pathogenesis of the lesion is a B-lymphocyte-mediated local immune inflammatory reaction in the lumbar spine. The

pathogenic process derives from the B-lymphocyte-mediated immune inflammatory reaction in the inferior vertebral body before it spreads to the adjacent disc and the superior vertebral body. Recognition of this pathogenic process is very important so that confusion with spine tumours or tumour-like lesions, metabolic disorders, genetic disorders, infection and avascular necrosis may be avoided. Further studies are needed to clarify its exact aetiology and pathogenesis.

Learning points

- Osteolytic destruction of the vertebral body is commonly related to the pathogenic process of spine tumours or tumour-like lesions, metabolic disorders, genetic disorders, infection and avascular necrosis.
- Two cases with the osteolytic destruction in two adjacent vertebral bodies along with the intervertebral disc were presented. The lesion of osteolytic destruction started in the L4 vertebral body and advanced further from the L3/4 disc to the L3 vertebral body.
- The pathogenic process derives from the B-lymphocyte-mediated immune inflammatory reaction in the inferior vertebral body before it spreads to the adjacent disc and the superior vertebral body.
- Recognition of this pathogenic process is very important so that confusion with spine tumours or tumour-like lesions, metabolic disorders, genetic disorders, infection and avascular necrosis may be avoided.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. Naul LG, Peet GJ, Maupin WB. Avascular necrosis of the vertebral body: MR imaging. *Radiology* 1989;**172**:219–22.
2. Dupuy DE, Palmer WE, Rosenthal DI. Vertebral fluid collection associated with vertebral collapse. *AJR* 1996;**167**:1535–8.
3. Theodorou DJ. The intravertebral vacuum cleft sign. *Radiology* 2001;**221**:787–8.
4. Ma R, Chow R, Shen FH. Kummell's disease: delayed post-traumatic osteonecrosis of the vertebral body. *Eur Spine J* 2010;1065–70.
5. McKiernan F, Faciszewski T. Intravertebral clefts in osteoporotic vertebral compression fractures. *Arthritis Rheum* 2003;**48**:1414–19.
6. Maldague BE, Noel HM, Malghem J. The intravertebral vacuum cleft: a sign of ischemic vertebral collapse. *Radiology* 1978;**129**:23–9.
7. Yu CW, Hsu CY, Shih TF, *et al*. Vertebral osteonecrosis: MR imaging findings and related changes on adjacent levels. *Am J Neuroradiol* 2007;**28**:42–7.
8. Mirovsky Y, Anekstein Y, Shalmon E, *et al*. Vacuum clefts of the vertebral bodies. *Am J Neuroradiol* 2005;**26**:1634–40.
9. Zimmerli W. Vertebral osteomyelitis. *New Engl J Med* 2010;**362**:1022–9.
10. Aprin H. Primary malignant tumors of the spine. In: Akbarnia BA, ed. *Spine: state of the art reviews*. Philadelphia: Hanley & Belfus, 1988:289–99.
11. Rogalsky RJ, Black GB, Reed MH. Orthopaedic manifestation of leukemia in children. *J Bone Joint Surg Am* 1986;**68**:494–501.
12. Chen G, Yang H, Gan M, *et al*. Polyostotic fibrous dysplasia of the thoracic spine. Case report and review of the literature. *Spine* 2011;**36**:E1485–8.
13. Aguirre Vila-Coro A, Dominguez R, Calk JB, *et al*. Congenital generalized fibromatosis. *Ann Ophthalmol* 1990;**22**:217–21.
14. Bolano LE, Yngve DA, Altshuler G. Solitary fibromatosis of bone. A rare variant of congenital generalized fibromatosis. *Clin Orthop Relat Res* 1991;**263**:238–41.

15. **Bakman M**, Monu JU. Focal fibrocartilaginous dysplasia (FFCD). *Pediatr Radiol* 2007;**37**:107.
16. **Hetts SW**, Hilchey SD, Wilson R, *et al*. Nonossifying fibroma. *Radiology* 2007;**243**:288–92.
17. **Al-Rikabi AC**, Ramaswamy JC, Bhat VV. Jaffe-Campanacci syndrome. *Saudi Med J* 2005;**26**:104–6.
18. **Hau MA**, Fox EJ, Cates JM, *et al*. Jaffe-Campanacci syndrome. A case report and review of the literature. *J Bone Joint Surg Am* 2002;**84**:634–8.
19. **Boyse TD**, Jacobson JA. Case 45: cystic angiomatosis. *Radiology* 2002;**223**:164–7.
20. **Sainani NI**, Pungavkar SA, Patkar DP *et al*. Multiple hemangiomas involving the vertebral column. *Acta Radiologica* 2005;**5**:510–13.
21. **Green HD**, Mollica AJ, Karuza AS. Gorham's disease: a literature review and case reports. *J Foot Ankle Surg* 1995;**34**:435–41.
22. **Vigorita VJ**, Magitsky S, Bryk E. Gorham's disease. An autopsy report. *Clin Orthop Relat Res* 2006;**451**:267–73.
23. **Fargen KM**, Lin CS, Jeung JA, *et al*. Vertebral brown tumors causing neurologic compromise. *World Neurosurg*. Published Online First: 17 Nov 2011. doi:10.1016/j.wneu.2010.02.022
24. **Araujo SM**, Bruin VM, Nunes AS, *et al*. Multiple brown tumors causing spinal cord compression in association with secondary hyperparathyroidism. *Int Urol Nephrol*. Published Online First: 17 Jan 2012. doi:10.1007/s11255-012-0123-2
25. **Maricic M**. Osetomalacia. *Curr Osteoporosis Reports* 2008;**6**:130–3.
26. **Indumathi CK**, Dinakar C, Roshan R. Juvenile Paget's disease. *Indian Pediatrics* 2009;**46**:354–6.
27. **Dinulos MB**, Stern DL, Graham CB, *et al*. Expansile bone lesions in a three-generation family. *Am J Med Genet* 1999;**82**:1–5.
28. **Marik I**, Marikova Hyankova E, Kozlowski K. Familial expansile osteolysis-not exclusively an adult disorder. *Skeletal Radiol* 2006;**35**:872–5.
29. **Lurie JD**, Gerber PD, Sox HC. A pain in the back. *New Engl J Med* 2000;**343**:723–6.
30. **Mont MA**, Joes LC, Einhorn TA, *et al*. Osteonecrosis of the femoral head: potential treatment with growth and differentiation factors. *Clin Orthop Relat Res* 1998;**355S**:S314–35.
31. **Urbaniak JR**, Harvey EJ. Revascularization of the femoral head in osteonecrosis. *J Am Acad Orthop Surg* 1998;**6**:44–54.
32. **Chou LH**, Knight RQ. Idiopathic avascular necrosis of a vertebral body: case report and literature review. *Spine* 1997;**22**:1928–32.
33. **Maheshwari PR**, Nagar AM, Prasad SS, *et al*. Avascular necrosis of spine: a rare appearance. *Spine* 2004;**29**:E119–22.
34. **Lee JH**, Kim KT, Suk KS, *et al*. Avascular necrosis of the spine in solid posterior fusion segments. *Spine* 2009;**34**:E158–61.
35. **Haro H**, Shinomiya K, Komori H, *et al*. Upregulated expression of chemokines in herniated nucleus pulposus resorption. *Spine* 1996;**21**:1647–52.
36. **Kawaguchi S**, Yamashita T, Yokoguchi K, *et al*. Immunophenotypic analysis of the inflammatory infiltrates in herniated intervertebral discs. *Spine* 2001;**26**:1209–14.
37. **Woertgen C**, Rothoerl RD, Brawanski A. Influence of macrophage infiltration of herniated lumbar disc tissue on outcome after lumbar disc surgery. *Spine* 2000;**25**:871–5.
38. **Kanerva A**, Kommonen B, Gronblad M, *et al*. Inflammatory cells in experimental disc injury. *Spine* 1997;**22**:2711–15.
39. **Satoh K**, Konno S, Nishiyama K, *et al*. Presence and distribution of antigen-antibody complexes in the herniated nucleus pulposus. *Spine* 1999;**24**:1980–4.

This pdf has been created automatically from the final edited text and images.

Copyright 2012 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Peng B, Chen J, Pang X, Hei Y. A kind of specific osteolytic destruction of the vertebral bodies. *BMJ Case Reports* 2012;10.1136/bcr-03-2012-6121, Published XXX

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow