## Case report

# Symptomatic intravertebral disc herniation (Schmorl's node) in the cervical spine

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SUMMARY A case of a Schmorl's node in the cervical vertebra causing neck pain is reported. An inflammatory focus was found on histological examination of Schmorl's node indicating a possible mechanism of pain production.

The phenomenon of Schmorl's node, an intravertebral disc herniation with subsequent bony response, has been well documented in the thoracic and

Accepted for publication 3 July 1984. Correspondence to Dr Stephen J Lipson, Brigham and Women's Hospital, 75 Francis Street, Boston, Mass 02115, USA. lumbar spine.<sup>1–5</sup> We report a case in which a Schmorl's node formed in a cervical vertebra causing neck pain. No previous reports of cervical Schmorl's nodes are known to us. In addition an inflammatory focus was found in relation to the Schmorl's node, indicating a possible mechanism by which pain and

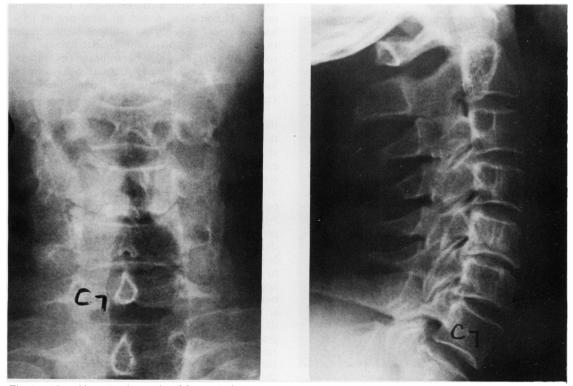


Fig. 1 AP and lateral radiographs of the cervical spine showing disc space narrowing and mild osteophyte formation at C5–6.

the bony response occur to intravertebral disc herniation.

#### **Case report**

A 49-year-old white male labourer presented complaining of neck pain. One and a half years before admission to hospital he had right neck pain of insidious onset which slowly progressed and shifted to the left paracervical area with mild upper arm and parieto-occipital radiation. He denied paraesthesias, numbness, or weakness. The pain worsened with range of motion of the neck. Physical examination revealed tenderness in the upper scapular border and mild restriction of neck extension with pain. No neurological abnormalities were noted.

Laboratory tests showed an erythrocyte sedimentation rate of 11 mm/h. Cervical spine radiographs showed mild narrowing and osteophyte formation at C5–6 (Fig. 1). Chest radiographs with apical lordotic views were normal. A bone scan showed increased activity in the C5 vertebra and C5–6 disc space. Tomography of C5–6 showed degenerative change with a right posterolateral 4 mm lucency in the inferior portion of C5 surrounded



Fig. 2 AP and lateral tomographs at C5-6 revealing a right posterolateral inferior C5 lucent area surrounded by bony sclerosis. There is loss of continuity of the bony endplate.

by bony sclerosis (Fig. 2). There was a localised loss of disc cartilage and localised loss of the endplate. Computerised tomography through the area showed no abnormality. Needle aspiration of C5–6 under fluoroscopy yielded no fluid. Discometrics at C5–6 caused reproduction of the patient's pain. Electromyography of the left upper limit muscles was normal.

An anterior cervical discectomy at C5–6 was done for biopsy. The disc appeared degenerative on gross examination. The endplate of C5 was curetted and found to contain soft tissue in the right posterolateral part. Interbody fusion was performed with an iliac crest graft. Histological examination of the curettings of disc, bone, and endplate showed fragments of hyaline cartilage, fibrocartilage, and nucleus pulposus with acute and chronic inflammatory cells (Fig. 3). Cultures of bone and disc gave no growth of bacteria, fungi, or mycobacteria. The graft became incorporated over three months after fusion, with gradual reduction in pain. The patient remains well at one year follow-up.

#### Discussion

Schmorl's nodes have been noted in 38% of all spines studied, with a slightly higher incidence in males.<sup>1</sup> Hilton *et al.* found an incidence of 75%, with a higher frequency in the thoracolumbar region than in the mid and lower lumbar spines.<sup>2</sup> This regional difference has been noted by others.<sup>4</sup> Studies have been restricted to the thoracic and lumbar spine, and to our knowledge there are no reports of Schmorl's nodes in the cervical spine.

The mechanism of Schmorl's node formation is thought to be discal herniation through a defect in the cartilaginous endplate of the vertebral body at sites where fetal vessels once penetrated the endplates.<sup>1-3</sup> Degenerative disc disease has been correlated with the number of endplate lesions, which may contribute to quicker degeneration, particularly at the thoracolumbar junction.<sup>2</sup> The bony sclerosis observed surrounding Schmorl's nodes has been thought to reflect reactive change due to repeated pressure, leading to cartilage and bone formation.<sup>1</sup> Vertebral body sclerosis adjacent to a degenerative disc has been described<sup>6-8</sup> and appears to be an inflammatory reaction within the bone to disc protrusion.<sup>9</sup> Disc herniation otherwise is not known to have inflammatory infiltrates directed at disc tissue, but inflammatory cells in granulation tissue have been reported to cause the resorption of disc herniation in the epidural space.<sup>10</sup> Inflammatory reaction to disc material is known in the discitis of ankylosing spondylitis, where endplate lesions allow contiguity of disc and the vascular intravertebral space. Disc material may herniate

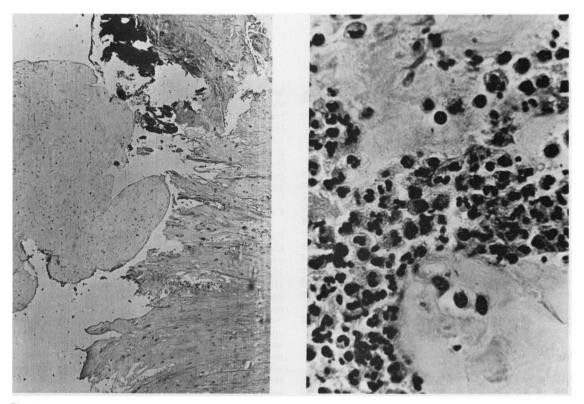


Fig. 3 Curettings of the C5–6 disc space showing fibrocartilage and annulus fibrosis. Haematoxylin and eosin; (Left,  $\times 230$ ). Right: an inflammatory infiltrate is noted about the disc material. ( $\times 560$ ).

through areas of erosion and become involved in the inflammatory process.<sup>9</sup> It would appear that this latter mechanism involves a pre-existing inflammatory process eroding the endplate. Disc tissue may elicit an inflammatory response in tissue exposed to it.<sup>11</sup> It may be that during certain stages of degenerative change exposed disc material may elicit an inflammatory response as seen in the present case.

Schmorl's node formation can occasionally cause pain,<sup>12</sup> <sup>13</sup> though most often Schmorl's nodes are an incidental anatomical finding. Degenerative changes in the disc with intravertebral herniation and vertebral body sclerosis may be accompanied by pain.<sup>6–9</sup> The case presented here represents an unusual and painful intravertebral disc herniation in which inflammation may have been the mechanism by which pain was produced.

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