LUMBAR INTERVERTEBRAL DISC PROLAPSE
IN TEENAGE TWINS

A CASE REPORT AND REVIEW OF THE LITERATURE

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We report the cases of teenage twin girls presenting within months of each other with severe symptoms from lumbosacral disc prolapses, requiring laminectomy in one and chemonucleolysis in the other. CT scans showed similarities in spinal configuration, including the presence of disc bulges at the L4-5 level. This suggests a strong hereditary factor in prolapse of intervertebral discs, but a review of the literature showed little information on that aspect.

Epidemiological studies of patients with lumbar intervertebral disc prolapse have identified a number of risk factors but surprisingly little is known about possible hereditary influences. We report the cases of identical teenage twins who presented within months of each other with similar symptoms from lumbosacral disc prolapse. We have found no previous report of lumbar disc prolapse in identical twins.

CASE REPORT

Identical twin girls, born in March 1973, both had menarche at the age of 10 years. They were tall and actively involved in sport.

In the middle of 1986, twin A aged 13, developed pain in the right buttock and the posterior thigh with no history of trauma, but was able, at first, to continue sport. A few months later, her sister B, developed backache and left sciatica, which forced her to stop playing sport. By May 1987 her straight leg raise was limited to 20° and her left ankle jerk was absent. Conservative treatment failed and a CT scan showed a small bulge at L4-5 (Fig. 1) and a disc prolapse at L5-S1 with evidence of sequestration (Fig. 2).

At laminectomy in July 1987 a large sequestrated disc fragment was removed. Recovery was excellent and at the last review 18 months after surgery, she had returned to full normal activities including competitive athletics and basketball.

Towards the end of 1987 twin A, who had been the first to experience leg pain, sought medical advice for worsening right leg pain. Straight leg raising was less than 50° but neurological examination was normal. Her CT scan showed a similar L4-5 disc bulge (Fig. 3) and a right-sided L5-S1 disc prolapse which appeared to be contained (Fig. 4). However, her sciatica increased and by April 1988 straight leg raising was restricted to 30°. Chemonucleolysis was performed and two months later she had complete relief of leg pain. Twelve months after the procedure she also had returned to a high level of competitive sport.

DISCUSSION

Lumbar intervertebral disc prolapse is uncommon in children and adolescents (Rugtveit 1966; Bradford and Garcia 1971; Bulos 1973), and disc sequestration is rare (Grobler, Simmons and Barrington 1979; Taylor 1982). Many authors consider that there is a direct causal relationship between disc herniation and injury in the adolescent (Epstein and Lavine 1964; Bulos 1973; Bek and ter Weeme 1975); involvement in competitive sports or athletics is often associated (Grobler et al 1979; Kurihara and Kataoka 1980).

Identical twin studies often provide an indication of
the relative importance of genetic and environmental factors. Our cases show several factors which indicate an hereditary aetiology: neither twin reported an injury, the timing of presentation was similar, and the lesion was at the lumbosacral level in both. In addition there was a striking similarity in the configuration of the spinal canal and posterior elements, including the presence of posterior bulging at L4-5.

Little attention has been given to the influence of heredity on disc failure. Grobler et al (1979) mentioned a positive family history in seven of 29 adolescents with disc herniation, and Wiltse (1971) considered heredity as an important factor. Using survey data obtained by the Arthritis and Rheumatism Council in the United Kingdom, Lawrence (1977) estimated that of the 42 first degree relatives of persons with a suspected disc herniation, five were considered by the physician to have had a herniated lumbar disc compared with the 1.4 expected in the population (p = 0.09). Szypryt et al (1988) showed on MRI scans that 67% of patients with one disc prolapse had disc degeneration at multiple levels, much higher than the general incidence and suggestive of an inborn weakness.

The importance of heredity in the development of disc herniation in dogs has been described by Wiltse (1971) and by Butler (1988), but the small vertebral canal in achondroplasia may be the cause of the increased incidence of clinical signs. Rowe (1965) suggested that an inherent soft-tissue weakness may predispose to herniation: of males with lumbar disc disease 18% had a
history of abdominal hernia, compared with 8% in the general population. This does not prove hereditary predisposition, since occupational or environmental factors may influence both abdominal and disc herniae.

To date there have been no detailed studies focusing on the hereditary aspects of disc failure. Indeed, most epidemiological studies of low back pain and disc herniation (Hrubec and Nashold 1975; Kelsey 1975; Frymoyer et al 1980; Valkenburg and Haanen 1982; Frymoyer et al 1983; Heliövaara 1988) do not mention family history but concentrate on occupational, environmental, demographic and personal factors, be these physical, biological or psychological. However, few of these studies (Hrubec and Nashold 1975; Kelsey 1975) have concentrated on disc prolapse rather than low back pain in general.

We conclude that a more detailed epidemiological study may reveal the importance of heredity in the development of intervertebral disc prolapse.

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REFERENCES


