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REVIEW

X. Phélip

Why the back of the child?

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X. Phélip (✉)
Department of Rheumatology,
University Hospital of Grenoble, BP 217,
F-38043 Grenoble Cedex, France
e-mail: xavier.phelip@ujf-grenoble.fr,
Fax: +33-4 76 76 56 02

Abstract An international congress about "the back of children and teenagers and the prevention of backache" was held in March 1999 in Grenoble (France). Beside specific low back pain following progressive and growth diseases, special attention was paid to non-specific low back pain (LBP). Some epidemiological data show a high incidence of LBP during and after the rapid growth phase, with the concomitant possibility of continued or recurrent evolution. MRI studies reveal frequent signs of disc degeneration: they start after the growth phase, spread during adolescence and are often correlated with backache. An immunohistological study seems to confirm the presence of degenerative-type alterations and changes in collagen in the vertebral plates and nucleus of juvenile spine. These data must be confirmed, and their relation to natural history and prognosis of juvenile LBP have to be clarified by longitudinal studies.

Key words Low back pain · Growth phase · Discal degeneration · Epidemiology · MRI · Histology

Introduction

Rheumatologic literature is rather poor concerning child and adolescent non-specific back pain. It essentially considers specific low back pain (LBP). Some cases are due to progressive diseases, such as infections, benign and malignant tumors, or inflammatory rheumatisms. Others are secondary to specific growth diseases: spondylolysis and spondylolisthesis, kyphosis and Scheuermann's disease, scoliosis.

Concerning non-specific LBP, the first studies date back about 20 years. Clinical and epidemiological data, analysis of risk factors, MRI and immunohistological findings draw attention to the early degenerative changes of the spine and to the usefulness of precocious prevention.

Epidemiological data

During adulthood

Biering-Sorensen [4] found that cumulated prevalence in adults increases with age, but annual incidence (6% every year for those aged between 30 and 60), is maximum in the age group 20 to 30 (11% every year). In occupational LBP, Abenhaim et al. [1] showed that the first requests for compensation are three times more numerous in patients aged between 20 and 24 than in those aged 55–64. In the Deyo and Tsuyi-Wu study [6], the LBP story was found usually to have begun between 20 and 29 years of age, and 11% of LBP had started before the age of 20.

These results suggest that in some adults, LBP may be prepared by asymptomatic lumbar changes in youth. Moreover, the decrease of incidence linked with associated increase of cumulated prevalence with aging shows the natural trend of LBP towards recurrent evolution.

During youth

Inquiries using questionnaires administered through schools have been conducted by some authors: Mierau et al. [10], Balague et al. [3], Troussier et al. [17], Salminen et al. [14], Burton et al. [5], Leboeuf-Yde and Ohm Kyrik [9]. They have been summarized by Troussier et al. [16].

The earliest cases of non-specific LBP occur from 11 to 12 years of age, at puberty, and are more frequent in girls. Later, their frequency increases with age: the prevalence increases by 10% every year, reaching about 50% of adolescents aged 18. Usually, pain is mild and not very disturbing. However, cases of chronic and recurrent LBP are not rare (5–15%), and disability results in 2–12.4% of the cases.

The results of the different inquiries are not exactly the same because of the diversity of the questionnaires. However, they all find common epidemiological and evolutive characteristics of non-specific juvenile LBP: precocious onset after puberty, increasing prevalence and incidence among teenagers, usual but not constant benignity. Yet no clinical prognosis criterion can determine beforehand the long-term evolution.

Risk factors of LBP have been studied by Salminen [12], Balague and Nordin [2], and Troussier et al. [15–17]. Age, spinal injuries, familial context of LBP, asymmetry of the trunk, rapid increase in height, nicotinism, time spent watching television, female sex, competitive sports and intense sporting activity, depression, stress and emotional conditions are significantly associated with LBP in the young. But most of the studies are cross-sectional and can not distinguish between causal and prognostic factors.

Radioclinical data

Standard radiographs were studied by Harreby et al. [8] in 640 subjects followed from 14 to 38 years of age. Among adults, prevalence, number of admissions to hospital and disabilities are all higher among those who suffered LBP during youth. But radiologic changes at age 14 are not statistically predictive of LBP at age 38.

MRI brought about an important advance in the knowledge of the juvenile spine during the growth period. The studies of Salminen [13] and Erkintalo [7] in schoolchildren show signs of disc degeneration (DD) in certain adolescent spines. At 15, DD is found in 26% of asymptomatic and 38% of symptomatic subjects, disc protrusions in respectively 2.5% and 20%, and Scheuermann-type changes in 8% and 23%. The radiographic presence of disc protrusion or Scheuermann-type changes is closely correlated with DD in symptomatic subjects. From 15 to 18 years of age, it appears that the number of DD and disc protrusions increases similarly in symptomatic and non-symptomatic subjects, and these two abnormalities remain strongly correlated.

Juvenile DD discovered by MRI is so frequent that it can be considered as a common change of the intervertebral disc. But its association with LBP, disc protrusion and later progressive degeneration is not rare. The concordance of these knowledges and epidemiological data has to be underlined.

Immunohistological data

Nerlich et al. [11] studied the anatomy, histology and histochemistry of 229 discs from 47 lumbar spines in subjects of all ages (fetus to 84 years).

Macroscopic signs of DD are present in some discs, occasionally as early as adolescence, and they increase with age. Histologically, the first signs of degeneration are noted sometimes in the group of subjects from 8 to 13 years of age: initially alterations of the endplates (irregularities, mucoid substance depositions). Among older subjects, the changes reach vessels, cells, chondrocytes and tissular structure, and they extend to nucleus pulposus and anulus fibrosus. Limited fibrocartilage clefts are seen in some adolescents aged 16 to 18 years, and extensive fibrocartilage fibrillation is noted in young adults aged between 20 and 25.

Immunochemical studies of the nucleus pulposus, the anulus fibrosus and the endplate show diverse modifications.

1. The deposition in some infantile and juvenile endplate chondrocytes and their nucleus of N-(carboxylmethyl) lysine (CML), which is regarded as a biomarker for oxidative stress. This deposition increases with age.

2. The change in composition and distribution of interstitial collagen types, especially the occurrence of collagen types IV and X, expressing phenotypic changes in the nucleus of chondrocytes.

These early modifications are in accordance with MRI results and strengthen the notion that disc degeneration can start as early as the second decade of life.

Conclusion

All the reported clinical, epidemiological, imaging and biological data are in concordance. They show the precocity of degenerative changes in the different tissues of the intervertebral discs.

However, the precise significance of these facts is unknown. The nature of their relation with LBP in youth and their prognostic value as regards the occurrence of LBP in adulthood need clinical, MRI and biological follow-up studies. Many risk factors may contribute to clinical symptoms, beside anatomic lesions of the discs. Yet general knowledge about these alterations has focused attention on non-specific LBP of the young.

Non-specific LBP has to be studied, with the patient's history as a fundamental concern. Some vertebral pain can originate in youth, so early prevention needs to be improved.

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