Are the metabolic characteristics of congenital intraspinal lipoma cells identical to, or different from normal adipocytes?*

Y. Giudicelli 1, A. Pierre-Kahn 2, A. M. Bourdeaux 1, P. de Mazancourt 1, D. Lacasa 1, and J. F. Hirsch 2

1 Department of Biochemistry, Faculté de Médecine Paris-Ouest, Centre Hospitalier de Poissy, F-78303 Poissy, France
2 Department of Neurosurgery, Hôpital Necker-Enfants Malades, 149, rue de Sèvres, F-75743 Paris Cédex 15, France

Abstract. Congenital intraspinal lipomas are frequently responsible for progressive neurological deficits caused by distortion or compression of the nervous system. Since fat metabolism in these lesions has not been previously studied, the aim of this study was to determine whether intraspinal lipoma cells behave like lipomas or like normal adipocytes. In 11 patients, intraspinal lipoma cells were compared with normal adipocytes isolated from adjacent subcutaneous adipose tissue for the following parameters: lipoprotein lipase (LPL), lipogenesis from U14C glucose, β-receptor number, adenylate cyclase activity, cyclic AMP production, and lipolysis in response to β- and α2-adrenergic agonists. No significant difference between these two cell populations was found, suggesting that intraspinal lipomas are not lipomatous tumors, but hamartomatous lesions capable of growth and regression along with the changes in the rest of the fatty pool. This emphasizes the danger of an abnormal weight gain, as well as the possible usefulness of an hypocaloric diet in patients who worsen in spite of previous surgery.

Key words: Spina bifida – Congenital intraspinal lipoma – Lipid metabolism.

The vast majority of congenital intraspinal lipomas are situated in the lumbosacral spine and occur concurrently with a spina bifida at the same level. The lipoma is usually subcutaneous and intraspinal, the two masses being joined by a fibrolipomatous stalk that penetrates the defect of the spina bifida. The subcutaneous portion of the lipoma may be responsible for a disfiguring deformation of the lumbosacral region. The deep portion of the lesion extends subdurally and terminates inside a low-lying conus terminalis. Although they are congenital malformations, these lesions are frequently the cause of progressive neurological deficits. These aggravations are usually explained by pulling (tethering) and/or compression of the nervous tissue. Tethering of the spinal cord, which results from fixation of the conus terminalis to the lipoma and to the dural sheet, is now a well-accepted concept [1, 9, 25, 31]. Conversely, the compression of the spinal cord and/or of the nerve roots is still controversial, and observations demonstrating this phenomenon, or the possibility of an increase in volume of such lesions, are rare [3, 21, 35]. The aim of this work was to study the metabolism of adipocytes in these intraspinal lipomas to evaluate their growth and possible subsequent compression of the nervous system.

Patients and methods

Case studies

Sixty-one patients with lumbosacral spina bifida and intraspinal lipoma have been treated surgically (50 cases) or followed as outpatients (11 cases) in the Neurosurgical Department of the Hôpital des Enfants Malades in Paris between 1971 and 1984. Of these, 6 exhibited evidence of a growing lipoma, as shown by the progressively increasing volume of their subcutaneous mass or the enlargement of the intraspinal mass, as seen on successive myelograms. To these 6 patients, 4 others are added whose spinal cord and roots were proved radiologically (Fig. 1) and/or operatively to be dramatically compressed. Two particular observations are described.

Patient 1. A boy born at term was first examined at the age of 3 weeks because of typical dysraphic cutaneous abnormalities on the midline of his lumbar region. These were associated with a spina bifida at L4–5 and of the sacrum. Because of the young age and normal neurological status of the child, it was decided with the parents that the lesion would be operated upon, but not before the age of 6 months. Less than 4 months later, the infant was readmitted as an emergency patient because of the recent onset of rapidly progressive paraplegia, which was predominantly distal and on the left side. The deep tendon reflexes of the lower limbs, present at the time of the first referral, were then absent. Sensation was globally diminished, with no saddle anesthesia. Apparently there were no sphincter disturbances. In the lumbar region, the already described cutaneous abnormalities were now accompanied by a fatty subcutaneous mass that was not present.

* Research supported by INSERM (CRL 824005 and CRE 854010) and the D.G.R.
Offprint requests to: A. Pierre-Kahn
Fig. 1. Myelogram with metrizamide demonstrating the considerable compression of an abnormally low-lying spinal cord by a massive intraspinal lipoma.

Fig. 2. Iopamidol myelogram: mass lesion occupying the entire lumbosacral canal up to L2.

Fig. 3 A, B. Metrizamide myelograms demonstrating the obvious increase in volume of a congenital intraspinal lipoma at an interval of 4 years. In 1978 (A), the mass extended from the middle height of L3 to L5–S1; in 1982 (B), it filled the entire lumbosacral canal up to the level of the upper margin of L3. Note: in 1982 the presence of a considerably enlarged lumbosacral canal with scalloping of the posterior aspects of L3 and L4.