Case Report

Early Recurrence of an Intra-spinal Dermoid with Rupture and Intra-cranial Spread

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INTRODUCTION

Spinal dermoid is a rare, benign, slow-growing, dysontogenetic tumour arising from the inclusion of ectopic embryonic rests of the ectoderm and mesoderm within the spinal canal at the time of neural tube closure between the 3rd and the 5th week of embryonic development. They may also rarely occur secondary to implantation of dermal fragments into the spinal sub-arachnoid space by accidental trauma, lumbar puncture or surgery [1]. Although referred to as tumours, they are embryological malformations with a capacity to expand rather than true neoplasms [2]. Their rupture and spread is a rare event, which may be asymptomatic or may present acutely as chemical meningitis. Although spinal dermoids may recur after surgery, the symptom-free interval after initial surgery is generally long [3].

CASE REPORT

A 29-year-old man, whose lumbar intramedullary dermoid had been resected 2 years previously, presented with sudden onset pain and stiffness of back, with altered behaviour. There was low-grade fever with headache. There was no history of convulsions. Patient had apparently been asymptomatic after the previous surgery and was not followed-up. Magnetic resonance imaging (MRI) of the dorsolumbar spine was performed with screening of the cervical spine. Fast spin-echo T2-weighted images revealed a 5 cm × 3 cm, heterogeneous lesion expanding the lower cord at the level of first and second lumbar vertebrae. On T1-weighted images, the lesion showed hyperintense signals, which were suppressed on fat-saturated images indicating its lipid content (Fig. 1). A screening T1-weighted sequence of the cervicodorsal spine was performed, which showed multiple hyperintense foci in the subarachnoid space suggestive of rupture. Axial images of the cord revealed an expanded central canal filled with fat (Fig. 2), which showed as hyperintense on T1-weighted images. Based on the imaging findings and past history, a diagnosis of chemical meningitis due to rupture of the dermoid was made. He subsequently had a further resection of the recurrent dermoid.

DISCUSSION

Intra-spinal dermoids have been described as unusual but not rare lesions. They comprise 1–2% of intra-spinal tumours and can be intramedullary, intra-dural-extra-medullary or extra-dural. They occur predominantly in the lumbosacral region (60%), involving the cauda equina and the conus medullaris, but are quite rare in the thoracic (10%) and cervical (5%) regions [1]. About 40% of spinal dermoids are intramedullary [4]. There is a slight male predominance and most spinal dermoids present during the second or the third decades [1]. Twenty percent of dermoids are associated with a dermal sinus [4]. Other associations include myelomeningocele, hypertrichosis and bony malformations [4].

Dermoid cysts are generally slow growing and tend to spread in the subarachnoid space, and are, therefore, relatively asymptomatic [1]. These often become acutely symptomatic after rupture or infection, precipitating an inflammatory meningeal reaction, which frequently precedes signs and symptoms due to the space-occupying lesion itself. Leakage of fat into the cerebrospinal fluid may be clinically silent [1]. The rupture can be spontaneous, iatrogenic or traumatic. The exact incidence of rupture of spinal dermoids is not known. The contents of a spinal dermoid include sebaceous secretions, desquamated epithelial cells and hair, which are very irritating. Cholesterol is the most irritating element in the dermoid, which causes a granulomatous meningeal response in the event of rupture of the dermoid cyst [5].

Spinal dermoids appear as variegated, space-occupying lesions showing a heterogeneous signal on T2-weighted MRI due to the different components within the cyst. These have high protein-containing fluid and thereby appear brighter than cerebrospinal fluid on T1-weighted images [4]. The high lipid content also gives a characteristic hyperintense signal on T1-weighted spin-echo images. Keratin and collagen probably

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contribute non-specifically as solutes, causing slight shortening of the water protons relaxation time. The role of cholesterol in these cysts is not well understood. They may cause high signal on T1-weighted sections, if concentration of short T1 lipid protons is sufficiently high [4].

The differential diagnosis of a spinal dermoid includes lesions with high lipid content such as teratomas and lipomas [1]. Treatment of intramedullary dermoids is surgical. Evacuation of the cyst contents, usually via laminectomy, with appropriate care not to spill the contents, is the treatment of choice. A conservative approach is taken regarding the cyst wall [2]. Typically the capsule is in close proximity to the adjacent neural tissue and total excision is hazardous and unnecessarily risks neural damage. Incomplete removal of the capsule may result in cyst recurrence, which is not likely to occur for years after the initial procedure and does not justify the risk involved in complete capsule removal [2]. Although the follow-up is often relatively short for these slowly growing tumours, the number of recurrences reported is nonetheless small [3].

Fig. 1 – Sagittal T1-weighted images reveal a heterogeneous mass expanding the lower spinal cord at the level of first and second lumbar vertebrae. Note the characteristic hyperintense signal within it and scattered hyperintense foci in the upper spine suggestive of fat. Also note the laminectomy.

Fig. 2 – Axial T1-weighted image showing the hyperintense signal filling the central canal.

Fig. 3 – Axial T1-weighted image of brain showing fat droplets, seen as hyperintense foci in the peri-mesencephalic cistern.
In conclusion, it is important that whenever a diagnosis of intra-spinal dermoid is suggested, it is essential to screen the entire spine, as well as the brain, to look for a possible rupture and spread. A recurrence of the tumour after incomplete removal can occasionally occur early and a regular follow-up is necessary.

REFERENCES