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ACQUIRED EPIDERMOID TUMOUR IN THE CERVICAL SPINAL CANAL

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Abstract:

A case of intraspinal epidermoid tumour following a posterior fossa surgery is described. This tumour developped 7 years after a left cerebellar pilocytic astrocytoma resection. We report a 19-year-old male who presented with a progressive 2 years history of cervical cord compression. Magnetic resonance imaging with gadolinium enhancement showed a 13 mm intradural extramedullary mass at the C5-6 level. Due to the unusual localization of this tumour and the absence of clinical and radiological stigmatas of spinal dysraphism, the preoperative diagnosis on this case was arachnoid cyst. This tumour was successfully operated. Pathologic diagnosis revealed an epidermoid tumour. Pathogenesis of this tumour was discussed (skin fragment migration).

Keywords: *Epidermoid tumour; Iatrogenic lesion; Cervical spine; Arachnoid cyst; MRI.*

Abbreviation:

MRI Magnetic resonance imaging.
ADC Apparent diffusion coefficient.

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CASE REPORT:

A 19-year-old male was admitted to the department of neurosurgery, Amiens University Medical Centre, with a 2 years history of aggravating unsteadiness of gait which has been related to his previous surgery and numbness of right lower limb. He also had progressive right sided hemiparesis mostly at right hand. Seven years earlier he had undergone posterior fossa craniectomy for left cerebellar pilocytic astrocytoma. There was neither history of trauma nor previous spinal intervention, including lumbar puncture. General physical examination was unremarkable (no cutaneous lesions on his back). Neurological examination showed right sided weakness with right hand atrophy, and diminution of deep sensation was noted throughout his both legs. Deep tendon reflexes were hyperactive in the lower extremities, and Babinski's sign was present bilaterally. Laboratory examinations were all within normal limits. MRI of the cervicothoracic spine revealed a 13 mm intradural, extramedullary mass at C5-6 level causing anterior displacement and compression of the cervical spine cord (Fig.1).

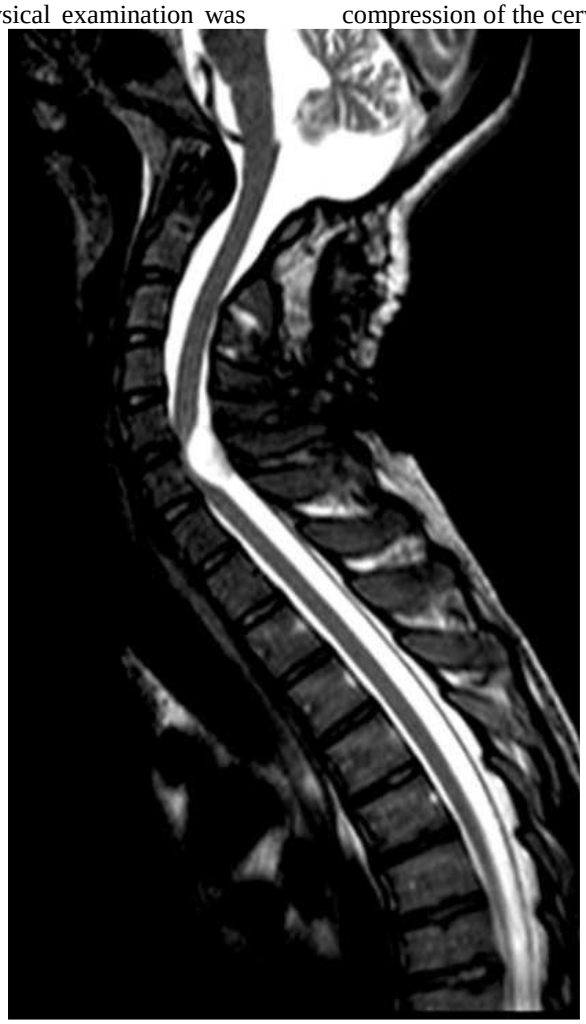

Fig. 1

This mass showed a hypo-intense signal on T1-weighted-MR imaging, hyper-intense on T2, weighted imaging. There was no contrast enhancement after gadolinium injection except a poor peripheral enhancement. This study did not demonstrate spinal dysraphism. MRI of the brain demonstrated no residual or recurrent tumour of the posterior fossa. The patient was operated following day and underwent total removal of the tumour via a C4-C7 laminectomy. At surgery, neither subcutaneous tract nor vertebral anomalies were found. After laminectomy and opening of the dura mater, an encapsulated pearly white tumour was seen in the subarachnoid space (Fig.2).

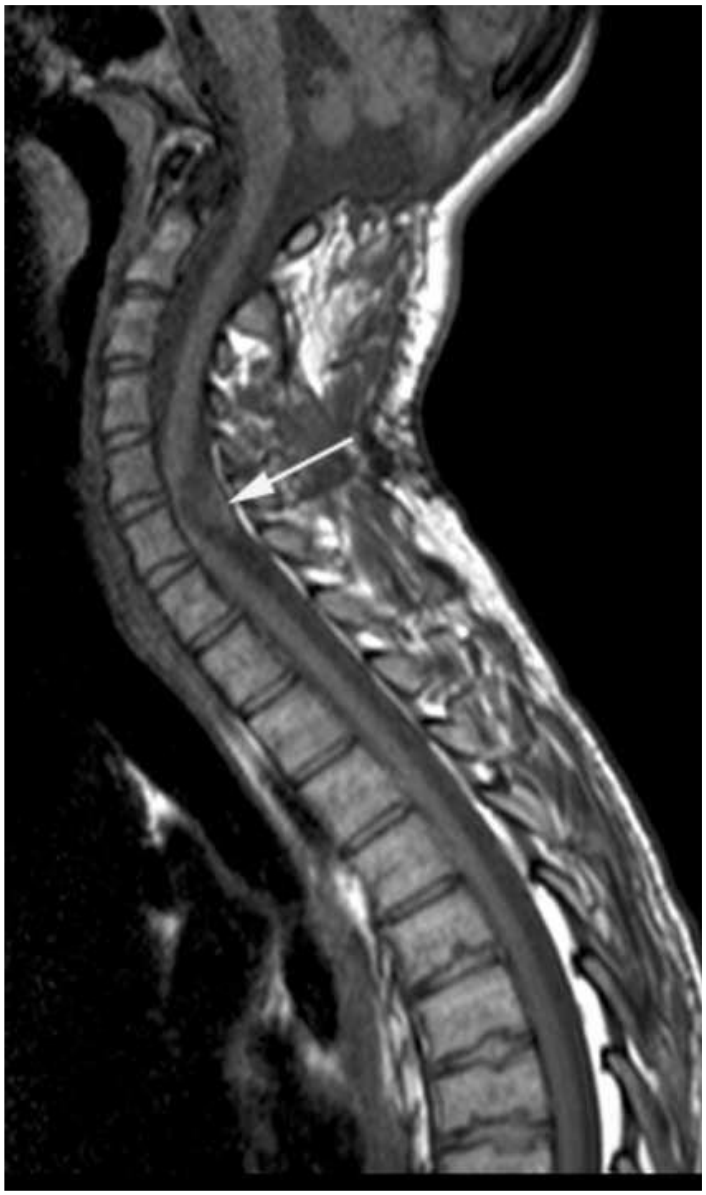

Fig.2
A monobloc excision of the tumour was performed without any visible cord injury or tumour remnant. A well –demarcated plane was noted between the tumour and the spinal cord after removal. Pathological diagnosis revealed
an epidermoid tumour without signs in favour of malignancy. The patient showed rapid neurological improvement following surgery. Postoperative MRI performed 4 months later revealed neither residual nor recurrent tumour. He was asymptomatic at the last follow –up.

Figure A: Spinal sagittal T2-weighted MR imaging demonstrating a 13 mm seized hypersignal intradural extramedullary mass located at the level of the fifth and sixth cervical vertebrae. B T1-weighted sagittal imaging highlighting a low-signal intensity mass (white arrow). Figure 3 without contrast enhancement following gadolinium injection except faint peripheral enhancement (white arrowheads).

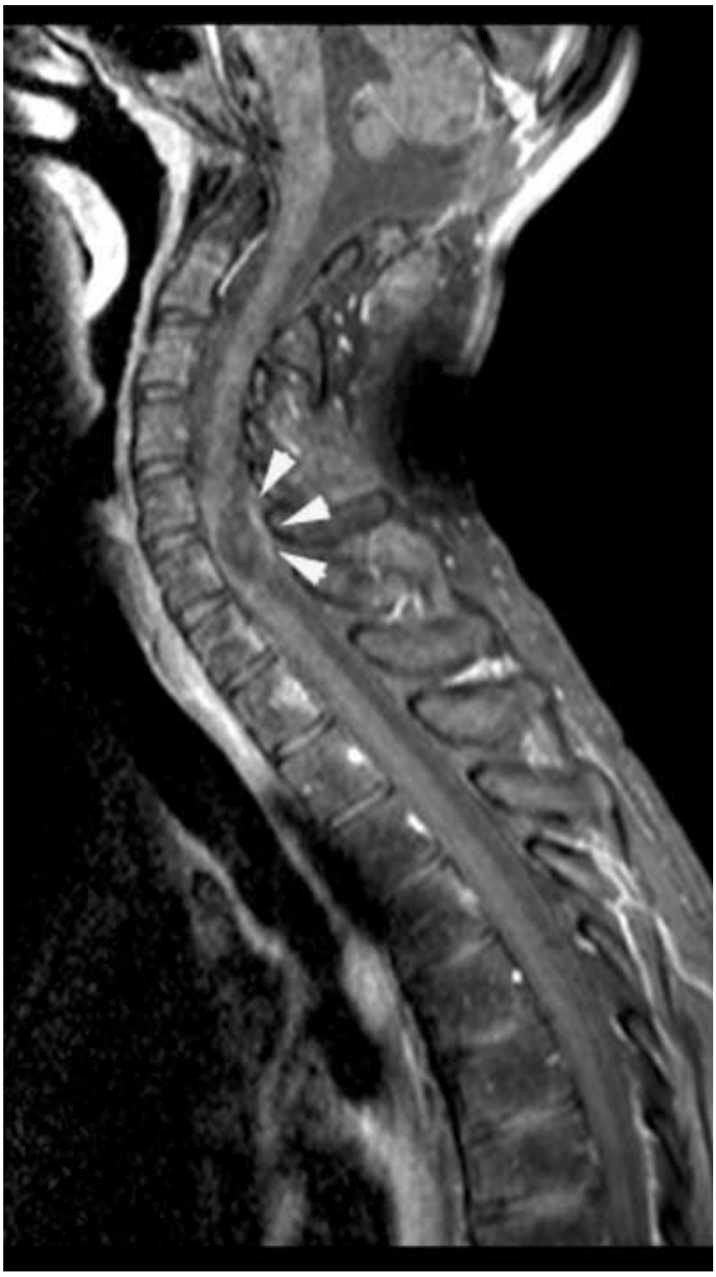

Fig. 3

Figure 4: Intraoperative photograph showing a wellencapsulated white pearly mass at C5-6 causing anterior displacement and compression of the spinal cord.

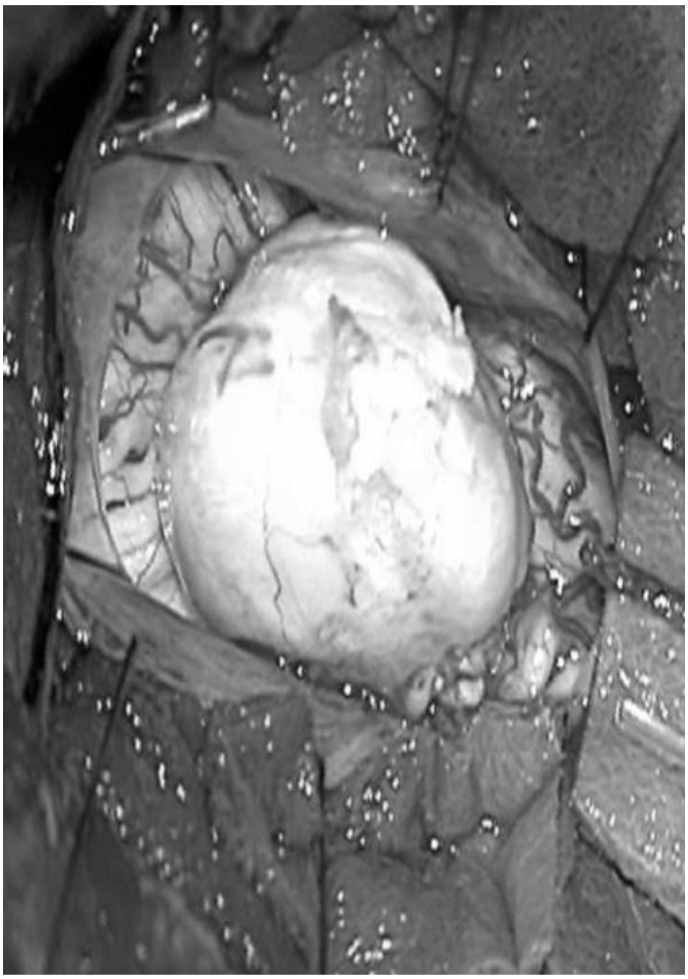

Fig. 4

DISCUSSION:

Epidermoid tumours were described by Cruveilhier in 1835, [6,13] who called them "tumours perlees" (pearly tumours).They represent less than 1% of all intraspinal tumours. These lesions are found most commonly in the subdural extramedullary space of the lumbosacral region, [4,6,16,17] while thoracic spine epidermoids are very rare and extremely rare in the cervical spine [10,14]. When epidermoid tumours occur in intradural extramedullary space, they are frequently associated with spinal dysraphism1[6,10] or as a consequence of repeated lumbar punctures, [11-13,15,20] while a congenital intradural extramedullary spine epidermoid tumours, without soft tissue or bony defect, have been reported [3,4,17].

The second favourite site for epidermoid tumours is the thoracic spine, usually intramedullary [1,14,16]. Some reports of acquired thoracic spine epidermoid tumours by CSF drop migration of skin fragment to sites more rostral have also been reported [11]. From a review of the literature, these tumours are extremely rare in the cervical spinal canal. Three large series of epidermoid tumours were reviewed by Lunardi et al [10] who found 8 epidermoids among 680 primaries spinal tumours. In 1989 Roux et al [16] reported 78 epidermoid tumours, in1973 Guidetti et al [7] found 3 epidermoid tumours of spinal cord. None of these series had epidermoid tumours in the cervical spinal canal. Nevertheless, it has been reported few cases of cervical epidermoid tumours either intramedullary, [14,18] or extramedullary but only associated with spinal dysraphism [8]. The genesis of congenital spinal epidermoids is explained by an anomalous implantation of ectodermal cells during closure of the neural tube between the third and fifth week of life, [12,16,18] and acquired lesions have been found several years after lumbar spinal punctures, trauma [11-13,15,20]. Histologically, they consist of lined stratified squamous epithelium supported by an outer layer of collagenous tissue. Progressive desquamation and rupture of keratin from the

epithelial lining into the interior of the cyst produce the characteristic content [4,7,16].

An iatrogenic origin was first reported by Choremis et al in 1956, [2] who cited implantation of skin fragment during lumbar puncture as the cause. In 1958, Gibson et al[5] proved experimentally that skin fragments can adhere to the needle during the puncture. Manno et al [12] reviewed series of 90 intraspinal epidermoid cysts from the literature and confirmed that 44 % of them were acquired.

The growing of epidermoids is slow, linear contrary to the most of the other tumours with their exponential growth. Therefore, there is a significant delay from the time of trauma e.x lumbar puncture until the onset of symptoms [3,10,12,16]. Most authors have reported an interval of 2 to 10 years [10,12,16]. Symptoms and signs of epidermoid tumours depend on both size and location of the tumour, and usually range from back pain, motor weakness,
sensory loss, and sphincter disturbance [4,7,8,10,16].

Intraspinal epidermoids can be difficult to detect radiographically. MRI is the investigation of choice [3,6]. The lesions are commonly hypo-intense on T1-weighted imaging and hyper-intense on T2-weighted imaging without contrast enhancement or minimal peripheral enhancement following intravenous gadolinium administration [4,21]. However, the signal characteristics of epidermoid tumours vary widely. This variation makes preoperative diagnosis very difficult [11,17,20,21]. In our case, the preoperative diagnosis was an arachnoid cyst, having most of the characteristics of epidermoid tumours. In rare difficult cases, we can need diffusion-weighted-imaging MRI and measurement of (ADC) values in order to differentiate epidermoid tumours from arachnoid cysts [9,19].

The treatment of epidermoid tumour is surgical; if possible, complete removal is the goal and is generally curative [3,17]. However, the adhesions of the capsule to the surrounding neural structures may sometimes limit the extent of excision [3,10,14,17].

In other point of view, it is possible that the epidermoid tumour was already present at birth. By reviewing the patient's medical file, the neuroradiologic proof of absence of tumour at the time or immediately after the first operation is lacking. Fortunately, we have found only one of five brain MRIs was done for follow-up the excised brain tumour . This MRI was performed 4 years after brain tumour excision (3 years prior spinal epidermoid tumour
excision). It has included cervical spine. This sagittal FLAIR MRI already showed 1×1 cm seized hypointense lesion at the upper level of C6 (not noticed by the neuroradiologist at that time), displacing the spinal cord anteriorly Figure 5: Spinal FLAIR MR imaging performed 4 years after brain tumour resection in posterior fossa, revealing a 1×1 cm low signal intradural extramedullary mass at the level of C6.

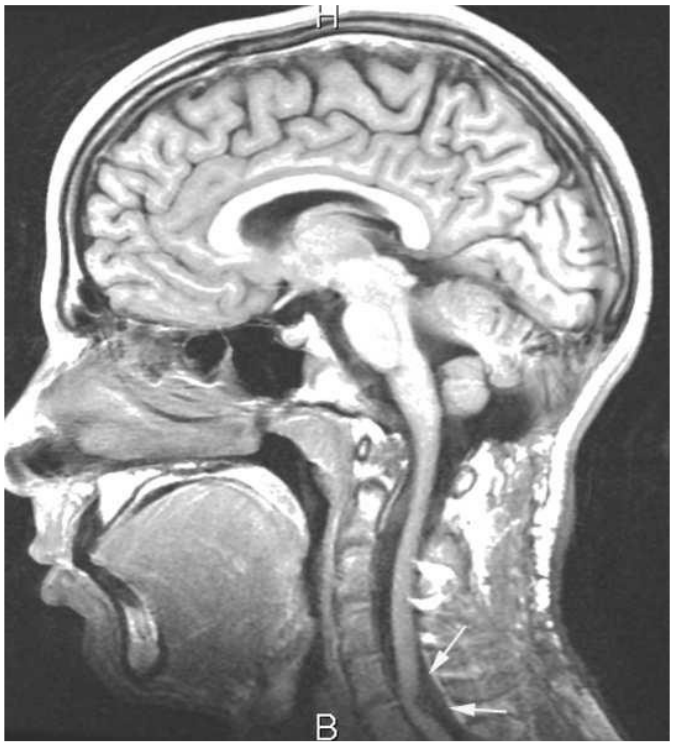

Fig. 5

In our case, the unusual localization of intradural extramedullary tumour, the absence of clinical and radiological signs in favour of spinal dysraphism, and the previous posterior fossa surgery with opening of the dura mater, argue for an acquired tumour. Moreover, we believe that the rate of growing of these tumours is very slow and linear. Indeed, the tumour had already been measured of 1×1 cm at the age of 17 years and it had become 1.3×2.6 cm at the time of resection.

In the present case, the site of the tumour is different from that of the operation: it seems that the skin fragment had probably migrated before developing into a tumour. As far as we know this is the first report of an acquired epidermoid tumour in the cervical spinal canal in which not only the possible relation between a previous posterior fossa surgery and epidermoid tumour, but also the presence of this tumour (intradural extramedullary) in the cervical spine with absence of spinal dysraphism.

CONCLUSION:

Though rare, epidermoid tumours must be a consideration for intradural lesions of the cervical spinal cord. Diffusion-weighted imaging with calculation of ADC images is considered to be potentially useful tool for differential diagnosis of spinal cystic tumours. The primary treatment should be complete emptying of the cyst material.

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