


Ethmoid sinus carcinoma metastasizing to the cauda equina: a case report.

Sir: A wide variety of neoplasms metastasise to the spine. The bony elements of the spine are most commonly affected but spread into the extradural space with subsequent compression of the spinal cord or cauda equina may occur. Intradural secondaries are rare. Extradural metastases usually present in subjects known to have malignant disease with constant unremitting low back pain which is particularly troublesome at night.1 A period of up to several months may elapse before neurological symptoms and signs develop in the lower limbs. Later in the course of the disease sphincter problems develop. We report here a patient who developed both extra and intradural metastases in the lumbosacral region from a primary carcinoma of the ethmoid sinus and whose presentation was with low back pain and a neurogenic bladder. Cauda equina compression by metastases from a paranasal sinus carcinoma has not been reported previously and this mode of presentation is seen only infrequently.

A 48-year-old man presented with left proptosis and nasal discharge in 1979. Investigations showed a very extensive tumour arising from the left ethmoid sinus with local extension into the nasal cavity, the orbit, sphenoid sinus and left petrous bone. The histology showed a solid undifferentiated neoplasm. He was treated with radiotherapy and had an excellent response with marked clinical regression of the tumour. In July 1981 he developed pain over the ischial tuberosities which subsequently spread to the posterior thighs. He was first seen in this department in February 1982 when he described the pain as increasingly severe, constant and particularly troublesome at night. On specific questioning he also admitted to urinary frequency and poor flow. Examination revealed reduction in straight leg raising to 45° on both sides. The ankle jerks were depressed but the other reflexes were normal. There was no sensory loss. In the abdomen the bladder was palpable almost up to the umbilicus. There was no clinical evidence of local recurrence of his carcinoma. Investigations showed normal routine haematology. Radiographs of the spine and pelvis and a bone scan revealed no evidence of metastases. On the excretion urogram the bladder was greatly enlarged and did not empty after micturition. Lumbar myelography showed multiple irregular indentations at the level of L5 indicative of tumour. At surgery he was found to have extradural tumour extending from the upper border of the L5 vertebral to below the S2 level and invading sacral bone. In addition there was tumour within the dural sac involving the nerve roots of the cauda equina. Biopsy of intradural and extradural tumour showed undifferentiated carcinoma. Decompressive laminectomy produced marked improvement in his symptoms. In particular micturition returned almost to normal and the bladder could no longer be palpated on abdominal examination.

Carcinoma of the paranasal sinuses almost always presents with local disease, although this is frequently extensive at the time of the diagnosis. Distant metastases are only rarely evident at this stage, but are frequent in the later course of the disease, although again usually overshadowed by the problems of local extension of the original tumour.2 Metastases to the vertebrae have been rarely described with these tumours3 but neither extradural nor intradural secondaries have been reported. In addition the mode of presentation was unusual in that sphincter involvement occurred early as the major neurological problem, the only other neurological abnormality being symmetrically depressed ankle reflexes. Presentation of extradural tumour in this way is uncommon4 but has been described as a feature of primary intradural tumours.3 It therefore seems likely in this case that the intradural metastases were responsible for the bladder symptoms. We would like to draw attention to this hitherto undescribed complication of paranasal sinus carcinoma and also
to emphasise that bladder problems may be an early feature of cauda equina compression.

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References


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Facial nucleus involvement in post-paralytic hemifacial spasm?

Sir: We report two unusual cases in which post-paralytic hemifacial spasm followed the injury of a very peripheral branch of the facial nerve. Such cases are unlikely to be explained by the peripheral hypotheses of the pathogenesis of post-paralytic hemifacial spasm.

A 65-year-old man developed twitches in his right face 12 months after he accidentally cut himself near the right jaw with sharp sheetsteel (Fig. 1A). The second patient, a 52-year-old man, complained of twitches around his right eye spreading over three weeks to the whole face; 10 months previously, his face had been accidentally cut by sharp glass near the right eye (Fig. 1B). In both cases the wound was clean without a haematoma, and there was no loss of consciousness. There was a slight weakness of the right orbicularis oris in the first patient, and of the orbicularis oculi in the second. A mild contracture at rest of the whole right hemiface was noticed in both cases, as well as a paradoxical co-activation of the mimic muscles on blinking or voluntary activation of the frontalis, orbicularis oculi and orbicularis oris. No other neurological signs emerged during a 5-years follow-up.

Electrophysiological examination of the right frontalis, orbicularis oculi and orbicularis oris muscles showed a mild reduction of MUPs in the orbicularis oris in the first patient and in the orbicularis oculi in the second; a subcontinuous activity of low amplitude MUPs at rest and the co-activation of all the muscles recorded during their separate voluntary activation were also noticed in both patients. These findings were consistent with post-paralytic hemifacial spasm.

Lamy1 first proposed the theory of misdirection of regenerating fibres to explain the abnormal associated movements in the facial muscles after incomplete recovery from facial nerve palsy. Another suggested mechanism was the formation of an "artificial synapse", or ephapse, at the site of the nerve injury when transaxonal excitation would give rise to a reverberating short-circuit to provoke mass muscle contraction and twitches.2,3 These hypotheses can explain cases of spasms occurring in a localised sector of the face following a lesion of a peripheral branch of the facial nerve.4 However, both theories are insufficient, in our opinion, to explain the phenomena we observed. Regeneration of a single peripheral branch is unlikely to be so widespread as to reach all the muscles of the face. The formation of an ephapse at the site of the injury does not explain the spasm of the whole face. The occurrence of associated movements of the mimic muscles of the whole face after injury of single
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