

Prostatic metastases in the nose and paranasal sinuses

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Abstract

Prostatic metastases in the nose and paranasal sinuses are rare. Seven cases have previously been reported in the world literature. We describe the clinical presentation of a patient with prostatic metastases and the use of prostate specific antigen in confirming the diagnosis. We also review the literature about metastases involving the nose and paranasal sinuses.

Key words: Neoplasm metastasis; Prostatic neoplasms; Paranasal sinuses; Nose

Introduction

Prostatic cancer spreads by direct local invasion and through lymphatic and vascular channels. It is commonly manifested by obstructive uropathy, regional lymphatic metastases or haematogenous metastases to the axial skeleton. Intracranial metastases are uncommon. Autopsies from patients with prostatic cancer revealed 4.4 per cent had intracranial metastases (Catane *et al.*, 1976). Clinically advanced prostatic cancers are associated with intracranial metastases in 4.2 per cent of patients (Castaldo *et al.*, 1983).

We report a case where extensive prostatic metastases were found involving all the paranasal sinuses and the nasal cavity. We believe this is the first report of prostatic metastases in the ethmoid sinuses, frontal sinuses and the nasal cavity.

Case report

A 71-year-old Caucasian male with a past medical history of a moderately differentiated adenocarcinoma of the prostate presented to his general practitioner with a two-month history of occipital headaches and dysarthria, followed by diplopia, nausea and subsequently dysphagia and left facial weakness.

He had undergone a transurethral prostatectomy three years previously: 14 g of prostatic tissue was removed at the operation and 95 per cent of this (30 chips) were histologically examined. All were infiltrated with moderately differentiated adenocarcinoma of Gleason grade 7 (3 + 4). Subcapsular orchidectomy was performed two years later. Benign intranasal polyps had been removed 14 and nine years previously. There was no other significant past medical history and in particular no predisposing factors for a cerebrovascular accident.

He was admitted under the physicians with a provisional diagnosis of a cerebrovascular accident. Initial examination revealed dysarthria, decreased sensation on the left side of the face, and bilateral VIth nerve and left VIIth cranial nerve palsies. He had a normal full blood count but a raised alkaline phosphatase of 851 u./l (40–120 u./l). A barium swallow with videofluoroscopy, revealed uncoordinated movements of the tongue and pharynx and aspiration. A CT scan of his brain showed generalized cerebral atrophy and ischaemia with no evidence of metastasis. The paranasal sinuses were not shown on the scan. A diagnosis of multiple strokes was favoured and he was commenced on aspirin: a nasogastric tube was sited and he was discharged home.

A month later he was re-admitted to the hospital because of increasing dysphagia, an episode of haemoptysis and the

development of a right VIIIth nerve palsy. A repeat CT scan showed, in addition to generalized ischaemic changes, extensive soft tissue in the frontal, ethmoid and sphenoid sinuses. In view of these findings he was referred to the Department of Otorhinolaryngology.

Examination revealed an alert but markedly dysarthric man, with bilateral severe exophthalmos and chemosis. Otorhinolaryngological examination showed bilateral middle ear effusions and a left otitis externa. The nasal cavity contained polyps with blood clots and there was blood in the postnasal space. The tongue was markedly atrophied. Neurological examination showed bilateral loss of corneal reflexes, bilateral VIth, VIIth and VIIIth cranial nerve palsies and a right foot drop.

Blood investigations were normal except for haemoglobin of 10.7 g/dl (14.0–17.7 g/dl), erythrocyte sedimentation rate of 45 mm in 1 h (< 20 mm in 1 h), urea of 17.4 mmol/l (2.5–6.7 mmol/l), creatinine of 180 µmol/l (6–120 µmol/l) and alkaline phosphatase of 1904 u./l (40–120 u./l).

The CT scan showed extensive soft tissue filling the maxillary antra and extending into the nasal passages. The soft tissue obliterated the ethmoid air cells, the sphenoid and frontal sinuses. In addition there was alteration in the bony texture of the posterior aspect of the maxillary antra, the ethmoid air cells and the whole of the sphenoid bone and petrous apices, with expansion and remineralization with sclerotic changes. In addition erosion of the body of the first cervical vertebra (Fig. 1) could be seen.

Biopsies from the polypoid masses in the nasal mucosa showed infiltration with adenocarcinoma with positive immunostaining for prostate specific antigen (Fig. 2), an appearance entirely consistent with metastatic adenocarcinoma of the prostate.

The patient died a month later and a postmortem request was refused.

Discussion

Bernstein *et al.* (1966) reviewed 72 cases of metastases to the nose and paranasal sinuses from the world literature in the years 1905 to 1966 and added 10 of his own. He found that the kidney was the commonest site of primary tumour (50 per cent) followed by the lungs, breasts, testis, gastrointestinal tract, uterus, thyroid, adrenal, skin melanoma and pancreas. The frequency of sinus involvement was maxillary (50 per cent), ethmoid (19 per cent), frontal (16 per cent), nasal cavity (10 per cent) and the sphenoid sinus (5 per cent).

Kent and Majumdar (1985) reviewed the literature on met-

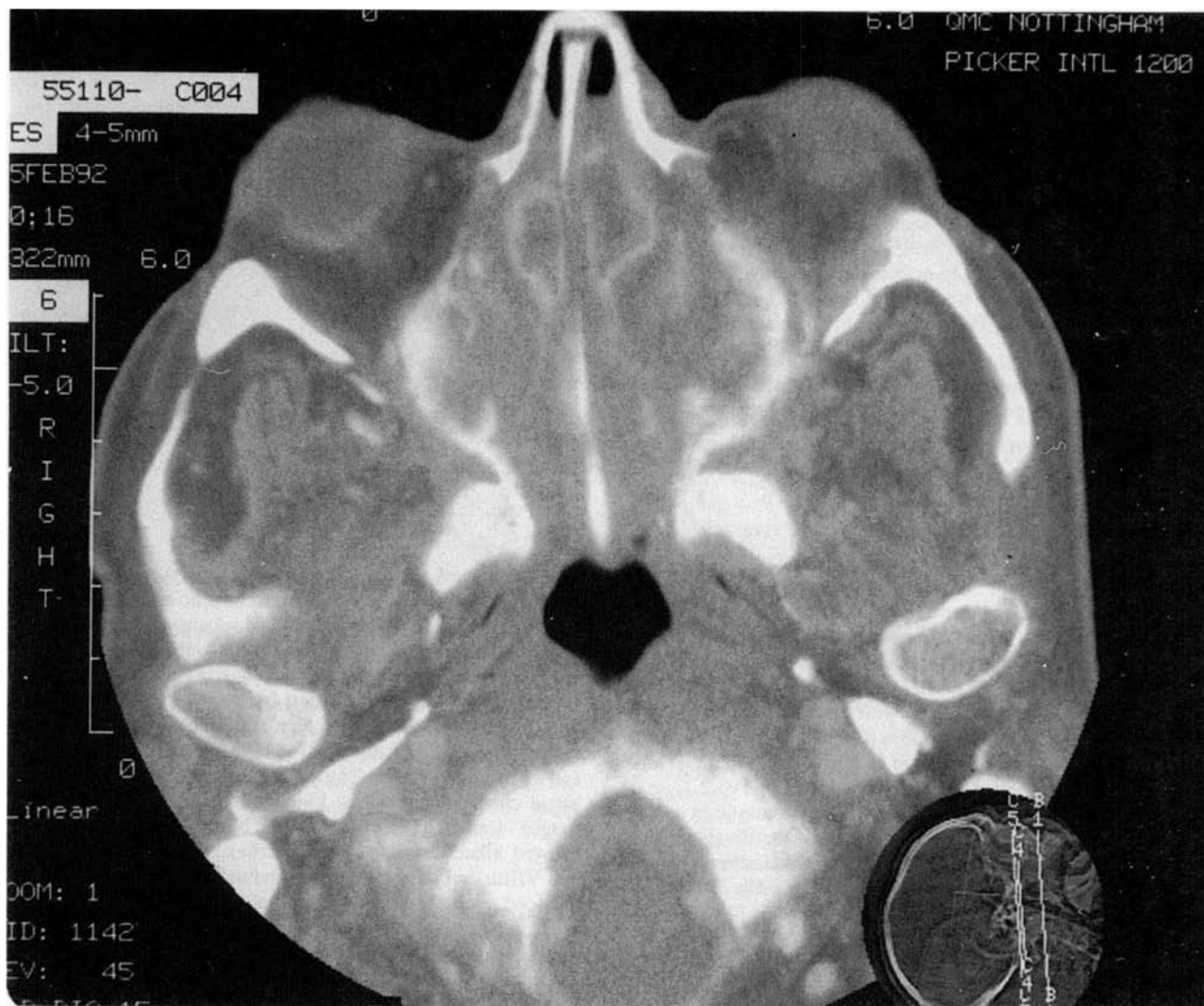


FIG. 1

A CT scan showing the soft tissue filling and expanding the paranasal sinuses with sclerosis of the bony architecture.

astases in the maxillary sinus from the year 1900 to 1985 and presented 55 cases. Their results supported those of Bernstein,

the kidney being the commonest primary site. In both series none of the cases originated in the prostate.

TABLE I
PROSTATIC METASTASES IN THE NOSE AND PARANASAL SINUSES: REVIEW OF LITERATURE

Author	Age of the patient	Site of metastasis	Presenting symptoms and signs	Course of disease
Barrs <i>et al.</i> (1979)	57	Sphenoid sinus	Diplopia, decreased acuity, ptosis, numbness of left face. Left IIrd, IIIrd, IVth, Vth, VIIth, and VIIIth cranial nerve palsies.	Patient died two years after presentation
Barrs <i>et al.</i> (1979)	61	Sphenoid sinus	Diplopia	Patient died two years after presentation
McClatchey <i>et al.</i> , (1985)	55	Sphenoid sinus	Frontal headaches, blurring of right eye, unsteady gait, nasal stuffiness, decreased sense of smell and taste	Patient alive, with disease, after one year
Matsumoto <i>et al.</i> , (1986)	79	Sphenoid sinus	Headache and diplopia, right VIth cranial nerve palsy	Unknown
Leduc <i>et al.</i> , (1986)	75	Sphenoid sinus and right cavernous sinus	Diplopia, right exophthalmos, right IIIrd cranial nerve palsy	Patient well 17 months after presentation
Har-El <i>et al.</i> , (1987)	77	Right maxillary sinus	Right exophthalmos	Unknown
Mickel and Zimmerman (1990)	67	Sphenoid sinus	Diplopia, numbness right side of the nose. Right VIth cranial nerve palsy	Patient died 2.5 months after presentation
Saleh <i>et al.</i> , (1993)	71	All paranasal sinuses and nasal cavity	Occipital headaches, diplopia, exophthalmos, dysarthria, dysphagia, deafness. Bilateral Vth, VIth, VIIth and VIIIth cranial nerve palsies	Patient died one month after presentation

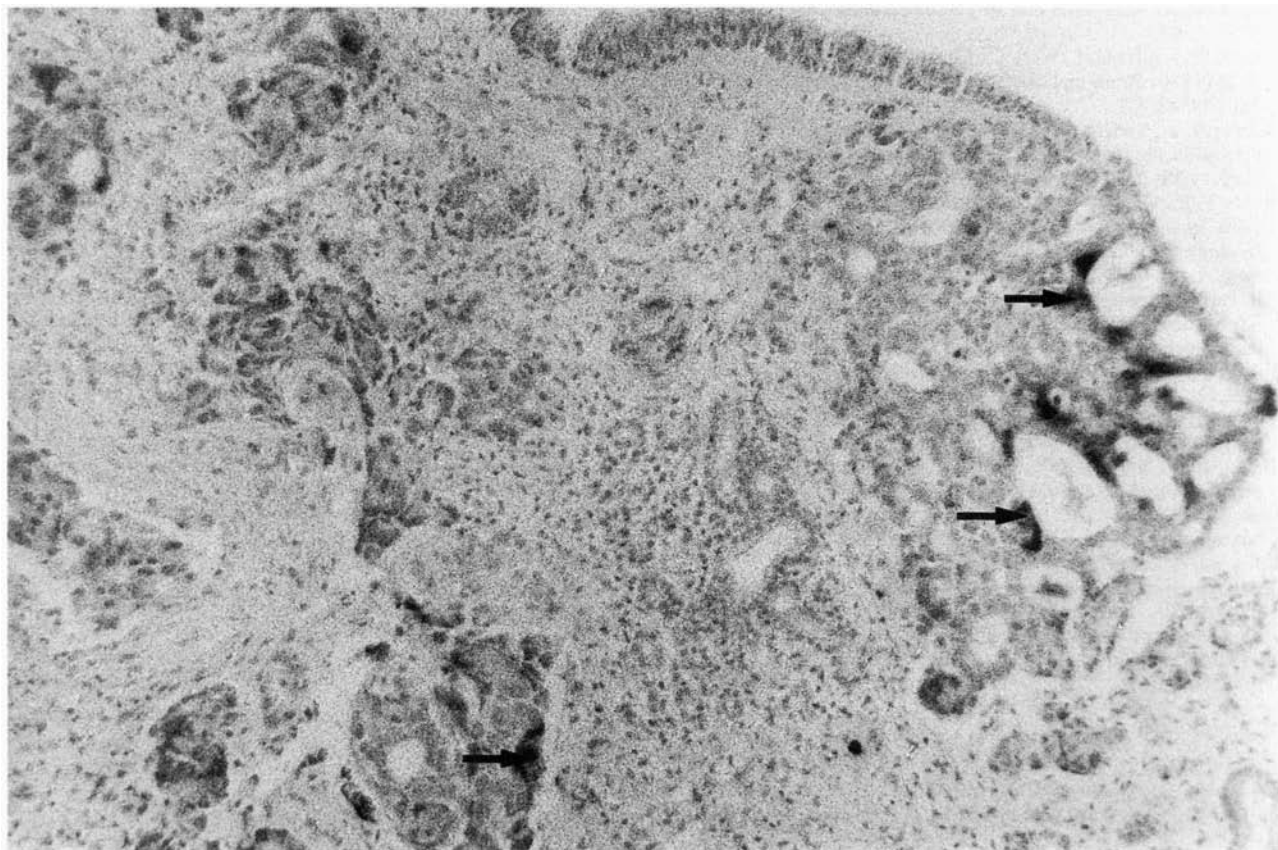


FIG. 2

Nasal mucosa infiltrated by tumour showing a glandular architecture. The dark stain 'arrows' show the positive immunostaining for prostate specific antigen.

Prostatic metastases have been reported in the sphenoid (Table I). McClatchey *et al.* (1985), then Mickel and Zimmerman (1990) and Andaz *et al.* (1991) have presented cases where prostatic metastases were the commonest, followed by the lung and large bowel. This leads to the conclusion that the distribution of metastases in the sphenoid sinuses is different from the distribution in the other sinuses.

The mode of presentation of metastases in the sinuses appears to be similar to the presentation of primary tumours in the same site, the symptoms and sign include headache, facial pain, diplopia, visual loss, exophthalmos, hearing loss and variable cranial nerve lesions (IInd to VIIIth) (Har-El *et al.*, 1987; Mickel and Zimmerman, 1990). The exceptions are the metastases from renal carcinoma, in which 70 per cent of cases present with epistaxis (Bernstein *et al.*, 1966; Patel *et al.*, 1980) and metastatic thyroid carcinoma (Cinberg and Terrife, 1980; Renner *et al.*, 1984) which can also present with epistaxis.

There is still controversy concerning the route by which prostatic carcinoma spreads to the axial skeleton. Baston (1940) proposed the presence of valveless vertebral veins which allow spread to the axial skeleton bypassing the vena cava and lungs. Castaldo *et al.* (1983) found evidence to support this theory at autopsy examinations. However, Dodds *et al.* (1981) after a study of patients using technetium 99m bone scans concluded that spread of tumour from the prostate followed the conventional venous channels and were distributed with the arterial system. The sites of secondary deposits, he suggested, were related to adhesive interactions of the circulating tumour cells and target organs.

Prostate specific antigen (PSA) is present in prostatic acini, ducts and secretions; it can be used as a specific marker of tumours of prostatic origin. Nadji *et al.* (1981) reported 100 per cent positive immunostaining for PSA in surgical and autopsy specimens of 122 known cases of prostatic carcinoma. Prostatic

acid phosphatase is normal in up to 15 per cent of patients with known bony metastases of prostatic origin (Bruce *et al.*, 1981).

For the past 50 years diethylstilboestrol or bilateral orchidectomy have been the main therapies for advanced or metastatic prostatic cancer. The recent development of new therapeutic agents has altered the options in the treatment of this disease (Huben and Perrapato, 1991). New alternatives include antiandrogens (e.g. flutamide) and gonadotrophin releasing hormone analogues (e.g. goserelin), either alone or in combination. They are superior to diethylstilboestrol in achieving only tumour response without causing serious complications (Emtage *et al.*, 1988) and are alternatives to orchidectomy in lowering testosterone to castrate level with the resulting tumour regression (Ryan and Peeling, 1988).

The prognosis of metastatic disease in the nose and paranasal sinuses is uniformly poor (Barrs *et al.*, 1979). Death within a few months of diagnosis is typical though long-term survival has been reported (Mickel and Zimmerman, 1990).

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