PANHYPOPITUITARISM IN A CASE OF NASOPHARYNGEAL CARCINOMA*

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SUMMARY

A case of nasopharyngeal carcinoma exhibiting panhypopituitarism is presented. This unusual clinical phenomenon is evaluated and the spread of the neoplasm is discussed.

Carcinoma of the nasopharynx is one of the most malignant tumours of the upper respiratory tract. In spite of the great interest shown in the medical literature, we remain ignorant of many facets of this singular neoplasm. In Caucasoids these tumours are rare, comprising 5 - 8% of head and neck tumours. In the southern Chinese and related communities in South-East Asia the incidence is remarkably high; deaths from nasopharyngeal carcinoma constitute 15% of deaths from all cases of malignant disease.[1] (This figure is exceeded only by that for carcinoma of the lung.) Environmental factors, such as the burning of incense, and genetic factors have been invoked as being of aetiological significance, but these claims remain unsubstantiated. In the South African Bantu the disease is rare—only about 4 cases are treated annually at Baragwanath Hospital.

The clinical presentation of this neoplasm has been well documented and may be correlated with the spread of the tumour. Nasal symptoms (obstruction or a blood-stained discharge) are the local manifestations of a tumour in the postnasal space. Otological symptoms, i.e. tinnitus, conductive deafness, blocked ears, result from occlusion of the Eustachian tube. Enlarged cervical lymph nodes may be the presenting feature, as lymph spread is a common and early phenomenon. Indeed, 90% of cases have cervical metastases. A variety of cranial nerve lesions indicate direct extension into the base of the skull.[2] The foramen lacerum, the petrous apex, the adjacent cavernous sinus and the internal carotid artery lie above the fossa of Rosenmüller, and are frequently invaded from this site of origin. The 5th nerve is the most commonly invaded, manifesting as numbness of the face, trigeminal neuralgia, or wasting of the muscles of mastication. The 6th nerve may be paralysed with consequent diplopia. Extension into the orbit may involve the 2nd and 3rd cranial nerves. Posterior extension may infiltrate the jugular foramen causing paralysis of the 9th, 10th, 11th and 12th cranial nerves.

The prevertebral muscles and bodies of the cervical vertebrae may be affected, causing severe pain in movements of the head. Lateral spread involves the internal carotid artery, the internal jugular vein and the sympathetic chain, giving rise to Horner's syndrome. Invasion of the floor of the sphenoid sinus was demonstrated in a third of the cases in a radiological study by Matin.[3] Teoh,[4] in a study of 31 necropsies among Chinese dying of nasopharyngeal carcinomas, found that the basi-occiput and posterior part of the body of the sphenoid were invaded in two-thirds of the cases, but gross invasion occurred in only 8 of 31 cases. He found infiltration of the dura mater and basal surface of the brain in 10% of cases. The temporal lobe was invaded in 2 cases, the pons in 1 case. The pituitary gland was invaded in 23% of these cases. In spite of this, pituitary dysfunction has not been widely recognized as a part of the clinical presentation. We have not been able to find a clinical record of panhypopituitarism occurring in a patient with this neoplasm. The following report documents such a case, which was treated at Baragwanath Hospital.

CASE REPORT

A 40-year-old Bantu male presented with headache, poor vision in the left eye, marked loss of weight, numbness on the left side of the face, fainting spells, impotence, and increasing frequency of micturition. A history of heavy smoking and alcoholic intake was elicited. He was emaciated, and his blood pressure was 90/70 mmHg. Small mobile lymph nodes were palpable on both sides of the neck. On the left side, paralysis of the 2nd, 3rd, 5th, 7th and 8th cranial nerves was present. A bilateral 12th-nerve paralysis was present. A large mass was seen in the postnasal space, predominantly on the left side, without ulceration of the superficial mucosa.

Radiological examination of the skull showed gross destruction of the sphenoid, basi-occiput and pituitary

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fossa, and both the anterior and posterior clinoid processes were obliterated (Fig. 1). These findings were confirmed by multiplane tomography. A carotid angiogram showed displacement of the internal carotid artery by an intracranial mass (Fig. 2). A biopsy of the postnasal

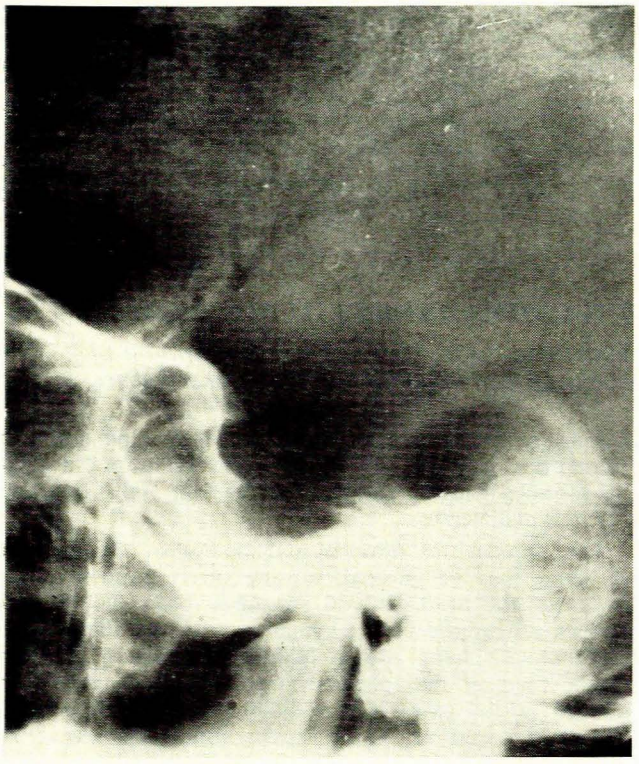

Fig. 1. Lateral view of skull showing erosion of pituitary fossa by carcinomatous invasion from the nasopharynx.

tumour showed the features of a well-differentiated squamous carcinoma.

Investigation of the blood showed that the erythrocyte sedimentation rate was 110 mm/hour, and a normochromic and normocytic anaemia was present with a haemoglobin level of 8·7 g/100 ml. The serum electrolytes were: urea 37 mEq/litre; potassium 4·3 mEq/litre; sodium 160 mEq/litre; CO_2 19 mEq/litre; chloride 170 mEq/litre. A random estimate of blood glucose was 67 mg/100 ml. The protein-bound iodine was 2·2 μg/100 ml and, the serum cholesterol was 105 mg/100 ml.

Examination of the urine showed a SG of 1·002. The patient developed a marked polyuria, passing 5 litres daily. The urinary electrolytes were: urea 470 mg/100 ml; potassium 13 mg/100 ml; sodium 18 mg/100 ml; and chloride 11 mg/100 ml. Microscopic examination showed 1 - 2 polymorphonuclears per high-power field. No albumin was present. The urinary 17-ketosteroids were 0·2 mg/100 ml (normal 6 - 25 mg/100 ml), and the 17-hydroxycorticosteroids were 1.5 mg/100 ml (normal 5 - 23 mg/100 ml). The CSF protein was 97 mg/100 ml and the VDRL was 2 units positive.

Administration of pitressin tannate 5 IU reduced the urinary output to 2 litres in 24 hours. Hydrocortisone was given by intramuscular injection but the patient's condition continued to deteriorate and he died.

Postmortem examination showed a huge squamous carcinoma of the postnasal space, invading the body and greater wings of the sphenoid beyond the foramen ovale on both sides. The basi-occiput was destroyed and replaced with tumour except for a small (2·5 mm) margin around the foramen magnum. The left carotid sheath was invaded and the pituitary fossa was replaced by tumour.

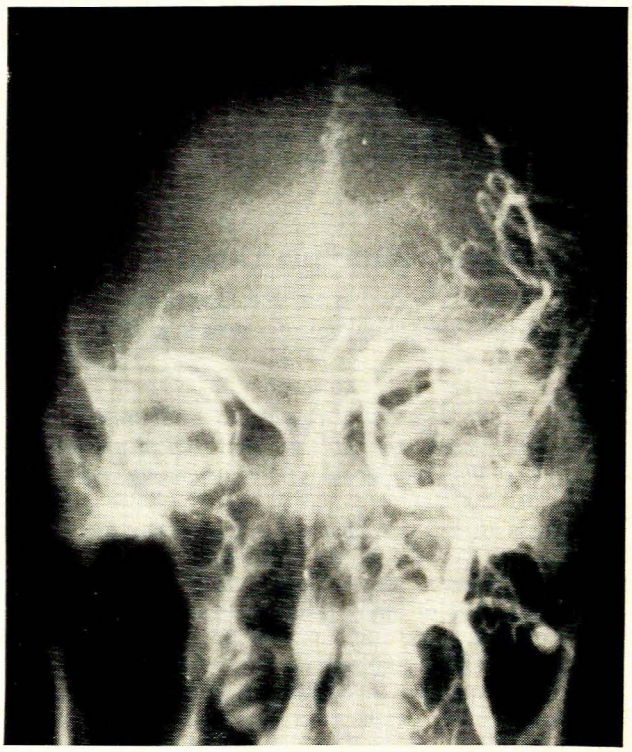

Fig. 2. Carotid angiogram showing displacement of internal carotid artery by carcinomatous invasion of the base of the skull.

DISCUSSION

This case shows the development and progression of panhypopituitarism as a result of carcinomatous infiltration. Wasting and weakness resulted from malignant cachexia and loss of all pituitary hormones. Episodes of fainting were due to postural hypotension. Moderate hypoglycaemia was present due to a decrease in growth hormone and cortisol. Reduced 17-hydroxycorticosteroids and 17-ketosteroids were indicative of ACTH deficiency. A low protein-bound iodine level demonstrated pituitary hypothyroidism. The absence of ADH resulted in a hypertonic dehydration with excessive diuresis and low urinary specific gravity.

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