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Radiation-induced cranial nerve palsy: hypoglossal nerve and vocal cord palsies

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Abstract

Cranial nerve palsies are an unexpected complication of radiotherapy for head and neck tumours. We present a case of this radiation-induced cranial palsy.

An 18-year-old female with nasopharyngeal carcinoma developed a right hypoglossal nerve palsy 42 months after cancericidal doses of radiotherapy. In addition, she developed a bilateral vocal cord palsy 62 months after therapy. Follow-up over four years has demonstrated no evidence of tumour recurrence and no sign of neurological improvement.

Introduction

The appearance of cranial nerve palsy associated with nasopharyngeal carcinoma (NPC) after radiotherapy usually signifies recurrence, since peripheral and cranial nerves are radioresistant structures and direct damage to cranial nerves has not been implicated in late radiation injury (Berger and Bataini, 1977). For the past 15 years, during follow-up visits of approximately 400 patients with head and cancer tumours (including 52 NPC patients) following radiotherapy, we have had only one patient with a radiation-induced cranial nerve palsy. This manifested as an unusual unilateral atrophy of the tongue and vocal cord palsy after a course of curative radiotherapy for NPC. To our knowledge, this late complication of radiotherapy for head and neck tumours is extremely rare (Ballantyne, 1975; Cheng and Schulz, 1975; Berger and Bataini, 1977; Saunders and Hodgson, 1979). We present the case history of this patient and describe the cranial nerve damage after radiotherapy.

Case report

An 18-year-old Japanese female presented with non-keratinizing carcinoma with lymphoid infiltration (lymphoepithelioma) of the nasopharynx with metastases to the bilateral upper cervical nodes ($T_3N_3M_0$) on 9 September 1980 (Fig. 1). There was no cranial palsy. The field arrangement for radiotherapy was lateral opposing fields for the nasopharynx and upper cervical lymph nodes and anterior field for the lower neck and supraclavicular fossae. Radiotherapy was administered from 20 September 1980 to 7 November 1980, utilizing cobalt 60, with radiation delivered to the upper and lower neck, and anterior and lateral nasopharyngeal fields. The tumour dose to the nasopharynx was 62 Gy. The involved lymph nodes of the upper neck 62 Gy and the uninvolved lower neck 42 Gy. Both the primary lesion and metastatic lymph nodes regressed following radiation therapy. Immediately following radiotherapy, she had severe xerostomia and loss of taste, but after two years these symptoms showed gradual improvement. There was no recurrence of the tumour during follow-up visits.

In July 1984, she was first noted to have atrophy of the right

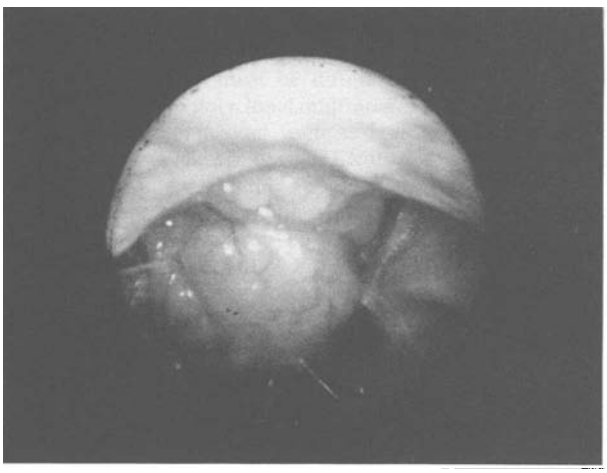

Fig. 1
Photograph showing tumour of the nasopharynx.

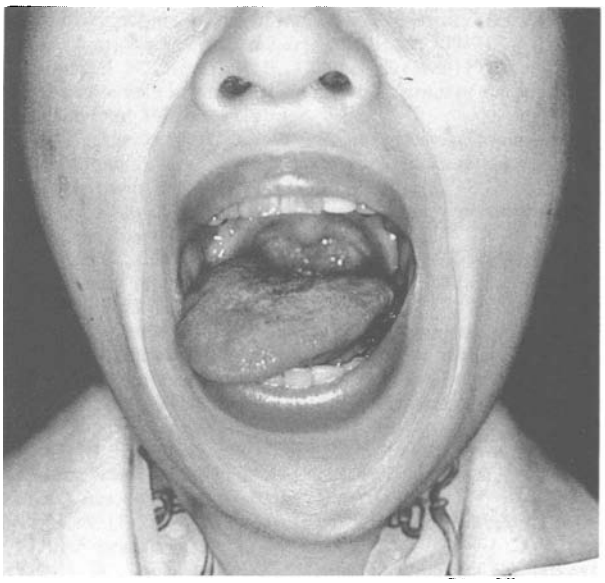

Fig. 2
Tongue deviating to the right on protrusion.

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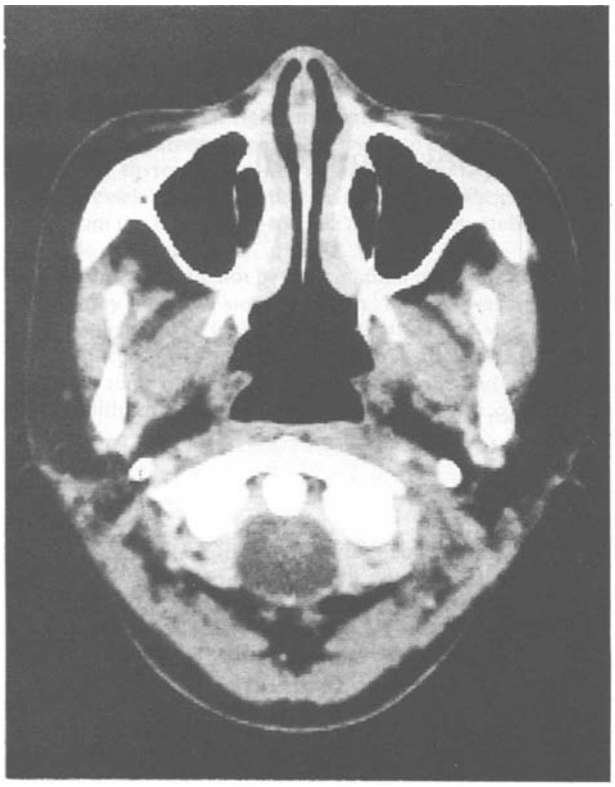

FIG. 3

CT scan on 16 March 1989, showing no recurrence of tumour of the nasopharynx.

side of the tongue; the tongue deviated to the right on protrusion (Fig. 2). On 8 August 1984, she developed slight hoarseness. Indirect laryngoscopy showed only slight mobility of the right vocal cord. On 9 January 1986, bilateral abductor vocal cord paralysis was noted. The cords were paralysed in the paramedian position. Fortunately, she did not require a tracheostomy, because there was only a slight problem with exercise tolerance. There were neither voice change nor aspiration. There were no other cranial nerve abnormalities. A chest radiograph revealed fibrosis of the right side of the upper lobe. However, computed tomography (CT) of the base of skull and extracranial soft tissues revealed no evidence of tumour recurrence (Fig. 3). She married, and became pregnant at the age of 25. In the eighth month of her pregnancy, a tracheostomy was performed for safety. She delivered a healthy male infant without any complications. Thereafter, decannulation was performed and there were no problems of breathing.

She has been followed up for an additional four years and remains in complete clinical and radiological remission.

Discussion

The nasopharynx is a roughly cuboidal open chamber below the base of skull with a rich lymphatic plexus. Therefore, NPC frequently invades the skull base and metastasizes to the cervical lymph nodes. Because of the anatomical location of the nasopharynx, surgical resection cannot ablate this disease. Radiation therapy is the primary treatment modality for most cases of NPC. However, radiation affects both the tumour tissue and also surrounding normal tissues. Thus, certain side effects cannot be avoided. In general, peripheral and cranial nerves are radioresistant structures. Radiation-induced cranial nerve palsy is rare (Mesic *et al.*, 1981). However, there are some reports of cranial nerve damage, especially of the hypoglossal (XII) nerve, after exposure of the neck to irradiation therapy (Ballantyne, 1975; Cheng and Schulz, 1975; Berger and Bataini, 1977; Saunders and Hodgson, 1979; Johnston *et al.*, 1989).

The mechanism by which radiation damages cranial nerves is unknown. Experimental irradiation of myelinated nerves in animals has shown a number of late sequelae. Focal nerve degeneration occurs and there is an increase in connective tissue with scar formation. Extensive vascular changes take place, particularly in small vessels with focal capillary obliteration. Schwann cells are affected, suffering a permanent depletion which may contribute to functional impairment (Janzen and Warren, 1942; Stoll and Andrew, 1966; Berger and Bataini, 1977).

In this patient, the appearance of XIIth nerve palsy was followed by bilateral vocal cord palsy. The interval between radiation therapy and the appearance of palsy may be inversely related to radiation dose (Berger and Bataini, 1977). Also, the XIIth nerve seems to be more susceptible to radiation injury than other cranial nerves (Ballantyne, 1975; Cheng and Schulz, 1975; Barger and Bataini, 1977; Saunders and Hodgson, 1979; Johnston *et al.*, 1989), although the reason is unclear.

The distinction between radiation-induced palsy and that due to tumour recurrence is critical. It is a distinction that can be inferred from the long latency period and confirmed by observation over a period of time. Radiation-induced cranial nerve lesions occur 2–10 years after the completion of radiotherapy (Berger and Bataini, 1977). At present there seems to be no way to eliminate this unfortunate sequela. We should follow patients after radiotherapy for head and neck tumours and be alert to late sequelae, among which cranial nerve palsies can be fatal.

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