A parapharyngeal meningioma presenting as a neck mass and multiple cranial nerve palsies

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Case report

A 20-year old Malay female presented with swelling at the angle of the mandible on the left side of 7 months duration. It was associated with neck discomfort, dysphagia and few episodes of aspiration. Examination showed a diffuse fixed swelling at the angle of mandible on the left side. It was non-tender and lymph node was not palpable. Multiple left cranial nerve palsies were elicited involving glossopharyngeal, vagus, accessory and hypoglossal nerves. Intraorally the left tonsil was medialized. A CT scan showed a left parapharyngeal tumour encasing the left carotid artery and left internal jugular vein (Figure 1). It measured 2.6 cm × 4.6 cm × 4.5 cm. The cerebral angiogram showed tumour blush which was not suggestive of a glomus tumour. The feeding vessel was from ascending pharyngeal branch of the external carotid artery. A CT guided trucut biopsy revealed a meningothelial meningioma. She underwent subtotal tumour resection (Figure 2).

Discussion

Meningiomas tend to occur intracranially owing to the presence of meninges. In some rare circumstances, it can be found extracranially mainly in the structures adjacent to the cranial cavity. Extracranial meningiomas represent 2% of all meningiomas [1]. The lesion can be primary in nature or extension from an intracranial origin.

There are at least four mechanisms to explain the origin of parapharyngeal meningiomas: (a) extracranial extension from an intracranial tumour by way of neural foramina, (b) extracranial growth from the arachnoid within the cranial nerve sheaths, (c) extracranial growth from ectopic or embryonic arachnoid cell rest, and (d) distant metastases from intracranial meningioma [2,3]. The second and third mechanisms are more likely in this patient as the brain CT did not show abnormalities to suggest intracranial origin. Most of the parapharyngeal meningiomas are secondary from intracranial extension. Parapharyngeal space is an uncommon site for primary meningioma.

In cases of benign intracranial meningiomas, the completeness of the resection is the major prognostic factor determining the outcome [4]. As an adjunct

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treatment, external beam radiotherapy has been shown to control the residual microscopic disease after definitive surgery. In addition, radiation therapy is also used to control grade III meningiomas or as a palliative modality in inoperable or elderly patients [5,6].

References


An unusual manifestation of amiodarone toxicity: lung mass mimicking a lung carcinoma

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Introduction

Amiodarone is a widely used antiarrythmic agent with well known toxic side effects that can involve lung, liver, thyroid and neuromuscular system. Amiodarone related pulmonary toxicity commonly presents as pneumonitis causing lung fibrosis. However, amiodarone toxicity can present as a lung mass [1, 2]. Such lesions can mimic a lung carcinoma both on chest X-ray and CT.

Case report

A 65-year old man with ischaemic heart disease developed progressive weakness of the lower limbs over six months which made him bed-bound. He had features of peripheral neuropathy with grade I muscle power. The nerve conduction studies showed a demyelinating sensory motor neuropathy. Liver enzymes were elevated (AST 108 U/l, ALT 132 U/l.). Thyroid functions were normal.

The chest X-ray demonstrated an ill-defined opacity in the apex of the right lung. Contrast enhanced CT scan of the chest and abdomen revealed a spiculated, irregular, hyperdense mass lesion measuring 2.5 cm in diameter, in the apex of the right lung suspicious of a malignant lung mass. There were bilateral thick, linear, pleural based hyperdensities, predominantly distributed in the lung bases. A few small high density peripheral lung consolidations were also present. Liver and spleen showed diffuse, high density in the non contrast CT scan (85-90 Hu compared to 50-60 Hu of normal liver).

CT features of peripheral consolidations and subpleural deposits raised the radiological diagnosis of amiodarone toxicity which was supported by the findings of increased density of liver and spleen. Subsequent inquiry revealed that the patient was taking amiodarone 200 mg three times a day for more than 10 years. He had defaulted clinic follow up for 10 years. Fine needle aspiration (FNAC) of the suspicious lung mass under CT guidance showed cytology consistent with amiodarone toxicity. Amiodarone was discontinued and patient was given limb physiotherapy.

Four months after discontinuing amiodarone, patient was able to walk with support and CT scan showed marked reduction in size of the lung mass, almost complete regression of subpleural hyperdense deposits and return of liver and splenic densities to normal levels.

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