

REVIEW

The skull base in oncologic imaging

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Date accepted for publication 22 September 2003

Abstract

Radiologists are often called upon to solve diagnostic problems involving the skull base in cancer patients. This presentation focuses on three issues: the detection of osseous involvement by infiltrating neoplasms, the diagnostic approach to bone tumours and the demonstration of head and neck tumours showing intracranial spread.

Keywords: *Skull base imaging; fibrous dysplasia; radiation-associated tumours; perineural infiltration.*

Skull base erosion

The skull base can be infiltrated by adjacent head and neck tumours such as nasopharyngeal tumours, parotid cancers and malignancies originating in the sinonasal area.

It has often been mentioned that MRI is the modality of choice in delineating the soft-tissue component of a malignant neoplasm. However, the erosion of the bony structures of the skull base is best achieved by using high-resolution thin section CT. This belief holds for osseous structures that are thin and predominantly composed of cortical bone. However, for the bones forming the central skull base such as the clivus, body of the sphenoid bone, the pterygoid process and the petrous apex, MRI often shows early changes more convincingly^[1,2]. In fact, CT often underestimates the frequency and extent of malignant infiltration.

MRI easily detects the replacement of high-signal intensity fat by intermediate signal intensity neoplasm on T1-weighted images. These lesions are also enhanced following the administration of gadolinium. However, it is not always possible to separate tumour involvement from inflammatory reaction induced by the presence of tumour tissue as experience in imaging laryngeal neoplasms has shown^[3,4].

Skull base osseous lesions

MRI is increasingly used as the first modality in the assessment of the skull base. When a lesion arising from the skull base is first detected, one should always remember the possibility of localised fibrous dysplasia. The plain radiographic and CT findings of fibrous dysplasia are characteristic. Unfortunately, the MRI characteristics of fibrous dysplasia do not share the distinctive features seen on radiographs and CT. In fact, the MRI appearances of fibrous dysplasia often resemble that of tumours^[5].

The signal intensity of fibrous dysplasia has been reported to be low on T1-weighted images. However, the signal intensity of fibrous dysplasia on T1-weighted images may be intermediate thus resembling that of a soft-tissue tumour. The signal intensity of fibrous dysplasia on T2-weighted images is often variable ranging from low to high signals in some patients. These high-signal intensities on T2-weighted images correspond to non-mineralised areas and regions of cystic changes demonstrated on CT. When T2-weighted images show high signals the differential diagnosis should include an inflammatory lesion or a neoplastic process. A destructive pattern is not a feature of uncomplicated fibrous dysplasia. As fibrous dysplasia is typically a

painless anomaly, the presence of pain should also alert the radiologist to the presence of a more sinister process.

Like destructive osseous lesions elsewhere in the body, the most likely diagnosis of a destructive process in the skull base is a metastatic deposit. However, the possibility of a primary malignant lesion arising must also be considered. One useful method of approaching this problem is to describe the signs and ask ourselves what we would call the lesion if it were located around the knee. In this way, we could use a familiar method and apply it in an unfamiliar location. It should also be remembered that many of the primary osseous tumours encountered in the head and neck are associated with previous radiotherapy^[6-10]. Before this possibility is entertained, a radiologist should be familiar with the criteria for making a diagnosis of radiation-associated tumours. These criteria include: (a) a history of irradiation; (b) the second neoplasm must occur within the field of irradiation; (c) the histology of the second neoplasm must be distinctly different from the primary tumour; and (d) a latency period of many years, arbitrarily taken to be at least 5 years^[11].

Intracranial spread

Extracranial tumours can gain access into the intracranial cavity via the numerous skull foramina. These openings include the superior orbital fissure, foramen rotundum, foramen ovale, stylomastoid foramen and jugular foramen^[12]. Various sinonasal squamous cell carcinomas (SCCa) after invading the orbit continue to extend through the superior orbital fissure. Alternatively, tumours can spread from the nasal cavity through the sphenopalatine foramen and continue superiorly to the inferior and superior orbital fissures. In addition, tumours such as adenocystic carcinomas have a propensity to spread through the foramen rotundum along the maxillary nerve^[13-15].

Malignant perineural infiltration of the mandibular nerve is also frequently encountered in head and neck oncologic imaging. SCCa originating in the oral cavity can spread via the lingual nerve to the foramen ovale. Nasopharyngeal carcinomas in particular have a propensity to spread into the intracranial cavity via the mandibular nerve or the foramen ovale^[16,17]. It should be noted that the foramen lacerum, although a large opening, is covered with fibrocartilage. Intracranial spread through the foramen lacerum does take place but not as frequently as once thought. This is because fibrocartilage is one of the most resistant tissues to malignant infiltration.

Infiltration around the foramen lacerum and pterygopalatine fossa often involves the Vidian canal. The Vidian canal is located at the junction of the body of the sphenoid bone and the pterygoid process. It transmits the

Vidian nerve (formed by the greater superficial petrosal nerve and the deep petrosal nerve). Hence tumour can spread intracranially to the geniculate ganglion and the seventh cranial nerve^[18].

Conclusion

Imaging plays a central role in the management of cancer patients with skull base involvement. This is an extremely difficult region to evaluate clinically. The radiologist, therefore, plays a crucial role in determining disease extent and the choice of appropriate treatment methods.

References

- [1] Chong VF, Fan YF. Skull base erosion in nasopharyngeal carcinoma. *Clin Radiol* 1996; 51: 625-31.
- [2] Chong VF, Fan YF. Detection of recurrent nasopharyngeal carcinoma: MRI vs CT. *Radiology* 1997; 202: 463-70.
- [3] Becker M, Zbaren P, Laeng H, Stoupis C, Porcellini B, Vock P. Neoplastic invasion of the laryngeal cartilage: comparison of MR imaging and CT with histopathologic correlation. *Radiology* 1995; 194: 661-9.
- [4] Curtin HD. Importance of imaging demonstration of neoplastic invasion of laryngeal cartilage. *Radiology* 1995; 194: 643-4.
- [5] Chong VF, Khoo JB, Fan YF. Fibrous dysplasia involving the skull base. *Am J Roentgenol* 2002; 178: 717-20.
- [6] Modan B, Baidatz D, Mart H *et al.* Radiation-induced head and neck tumors. *Lancet* 1974; 1: 277-9.
- [7] van der Laan BF, Baris G, Gregor RT, Hilgers FJ, Balm AJ. Radiation-induced tumors of the head and neck. *J Laryngol Otol* 1995; 109(4): 346-9.
- [8] Mark RJ, Bailet JW, Poen J *et al.* Post-radiation sarcoma of the head and neck. *Cancer* 1993; 72(3): 887-93.
- [9] Goh YH, Chong VF, Low WK. Temporal bone tumours in patients irradiated for nasopharyngeal neoplasms. *J Laryngol Otol* 1999; 113: 222-8.
- [10] Lim LH, Goh YH, Chan YM, Chong VF, Low WK. Malignancy of the temporal bone and external auditory canal. *Otolaryngol Head Neck Surg* 2000; 122: 882-6.
- [11] Cahan WG, Woodward HG, Higinbotham NL, Stewart FW, Coley L. Sarcoma arising in irradiated bone: report of eleven cases. *Cancer* 1948; 1: 3-29.
- [12] Chong VF, Khoo JB, Fan YF. Imaging the nasopharynx and skull base. *Magn Reson Imaging Clin N Am* 2002; 10: 547-71.
- [13] Curtin HD, Williams R, Johnson J. CT of perineural tumor extension: pterygopalatine fossa. *Am J Neuroradiol* 1984; 5: 731-7.
- [14] Daniels DL, Pech P, Pojunas KW *et al.* Trigeminal nerve: anatomic correlation with MR imaging. *Radiology* 1986; 159: 577-83.
- [15] Chong VF, Fan YF. Maxillary nerve involvement in nasopharyngeal carcinoma. *Am J Roentgenol* 1996; 167: 1309-12.
- [16] Chong VF, Fan YF, Khoo JB. Nasopharyngeal carcinoma with intracranial spread: CT and MR characteristics. *J Comput Assist Tomogr* 1996; 20: 563-9.
- [17] Russo CP, Smoker WR, Weissman JL. MR appearance of trigeminal and hypoglossal motor denervation. *Am J Neuroradiol* 1997; 18: 1375-83.
- [18] Ginsberg LE, De Monte F, Gillenwater AM. Greater superficial petrosal nerve: anatomy and MR findings in perineural tumor spread. *Am J Neuroradiol* 1996; 17: 389-93.