Editorial

Skull Base Osteomyelitis

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Skull base osteomyelitis may develop as a complication of paranasal sinusitis or other regional infectious process or as an unfortunate sequelae of iatrogenic injury or trauma. Afflicted patients generally have some form of systemic immunocompromise, most often diabetes, or a history of external beam radiotherapy for a head and neck malignancy with the radiation portal encompassing the area of the skull base.[1] Before the era of systemic antimicrobial therapy, skull base osteomyelitis was almost universally fatal. Acute osteomyelitis often results in rapid lytic destruction of the skull base bone commonly associated with cranial nerve palsy/paralysis, and rarely, with craniovertebral instability and intracranial abscess formation.[2] It may or may not be associated with a deep fascial, orbital or intracranial soft tissue component. Chronic osteomyelitis, as seen in granulomatous conditions, is often much more indolent and slowly progressive. Patients may still exhibit cranial nerve palsies, albeit usually more slowly. As such, they are often able to compensate for these losses over time and hence often delay their clinical presentation.

In contradistinction to malignant otitis externa (otologic skull base osteomyelitis), nonotologic skull base osteomyelitis may pose a significant diagnostic clinical challenge. There are no pathognomic findings on clinical or radiographic examination which confirm skull base osteomyelitis. Tissue diagnosis is the key to both diagnosis and management of this rare entity. Preoperative CT or MRI-guided fine needle aspiration of accessible lesions is warranted for all skull base lesions. Unfortunately, this is not feasible in many cases due to inaccessibility. Thus, advanced open and/or endoscopic craniofacial approaches are required to allow for safe exposure of the affected areas. Preoperatively, it may not be possible to differentiate infectious from neoplastic entities that can present in a similar fashion.[3] Once access has been achieved, confirmatory biopsy should be sent for pathologic analysis. If osteomyelitis is confirmed, then aggressive surgical debridement of grossly affected bone, preserving neurovascular anatomy whenever possible, followed by culture-driven systemic antibiotic therapy (generally for 6 weeks) is the mainstay of treatment. Initial or suspected nonotologic osteomyelitis should be treated empirically to provide coverage for *Staphylococcus aureus.*[1] This is in contradistinction again to otologic osteomyelitis where empiric coverage for *Pseudomonas aeruginosa* should be provided. Cultures should always be sent to evaluate for possible fungal and tuberculosis pathologies as well. Serial gallium scans may be useful to follow these patients postoperatively as a marker of response to treatment. Serial CT and MRI scanning seems to be less useful in this regard.

With aggressive surgical debridement facilitated by the broad exposure provided by modern craniofacial approaches, followed by tissue-guided systemic antimicrobial therapy, one can anticipate resolution of the progressive radiographic findings, and often times, note reversal or improvement of neural deficits (especially those of recent onset). Refractory cases and those arising in a radiated field may benefit from the adjunctive use of hyperbaric oxygen therapy (minimum of 30 dives).[4] The major difficulty encountered by the clinician treating this patient population remains that of establishing a sound diagnosis and differentiating this lesion from a neoplastic process.

References

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Please see "Idiopathic Osteomyelitis at the Base of the Skull" on page 1121 of this issue.

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