Skull base osteomyelitis

In 1775, cranial osteomyelitis was first explained by surgeon Percival Pott as a collection of pus under the pericranium. Dr. Pott reported cranial osteomyelitis as a consequence of forehead trauma (bone contusion) and extradural hemorrhage ¹⁾ Later, it became known that the cause of such an infection was not an injury but the spread of infection from neighbouring structures, for example, paranasal sinuses. Meltzer and Kelemen first described skull base osteomyelitis (SBO) in 1959 in patients with a burn injury and osteomyelitis of the external auditory canal ²⁾.

Diagnosis

Skull base osteomyelitis (SBO) is a potentially life-threatening inflammation of cranial base bony structures of variable origin. Criteria for diagnosis and treatment are still controversial.

The diagnosis of SBO should be made according to four points: a high index of clinical suspicion, radiologic evidence of infection, repeated biopsies that are negative for malignancy, and positive results of microbiologic tests. SBO typically manifests clinically in patients with diabetes and recurrent otitis externa; the infection usually extends inferiorly to the compact bone of the infratemporal fossa, affecting the lower cranial nerve foramina. Several image-based techniques should be used to diagnose SBO. CT is the best option for evaluating bone erosion and demineralization, MRI can help delineate the anatomic location and extent of disease, and nuclear imaging is useful for confirming bone infection with high sensitivity. However, the standard diagnostic procedure for SBO is for patients to undergo repeated biopsies to rule out malignancy, with histopathologic signs of infection and detection of microorganisms in the biopsied bone or soft tissue indicating SBO. The ability to diagnose SBO can be increased by identifying patients at risk, recognizing the most important causes and routes of infection, describing the main radiologic findings, and always considering the differential diagnosis ³⁾

Case series

Czech et al. conducted a retrospective chart review of HNC patients diagnosed with SBO.

SBO was commonly diagnosed with nasal endoscopy showing mucosal breakdown between the naso/oropharynx and skull base and with characteristic changes on CT/MRI. Culture data were often polymicrobial, inclusive of naso/oropharyngeal flora, but half of the patients additionally had antibiotic-resistant or atypical pathogens. The mean duration of antimicrobial therapy was 117 +/- 94 days. Recurrent SBO was found in half of the patients, associated with Pseudomonas aeruginosa and with persistent defects in the mucosa abutting the skull base.

Diagnosis and management of SBO in HNC patients are challenging. Recommendations to aid in clinical care are proposed.

Level of evidence: 4, case series ⁴⁾

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Six proven cases. The diagnosis of SBO should be made according to four points: a high index of clinical suspicion, radiologic evidence of infection, repeated biopsies that are negative for malignancy, and positive results of microbiologic tests. SBO typically manifests clinically in patients with diabetes and recurrent otitis externa; the infection usually extends inferiorly to the compact bone of the infratemporal fossa, affecting the lower cranial nerve foramina. Several image-based techniques should be used to diagnose SBO. CT is the best option for evaluating bone erosion and demineralization, MRI can help delineate the anatomic location and extent of disease, and nuclear imaging is useful for confirming bone infection with high sensitivity. However, the standard diagnostic procedure for SBO is for patients to undergo repeated biopsies to rule out malignancy, with histopathologic signs of infection and detection of microorganisms in the biopsied bone or soft tissue indicating SBO. The ability to diagnose SBO can be increased by identifying patients at risk, recognizing the most important causes and routes of infection, describing the main radiologic findings, and always considering the differential diagnosis⁵

Demographics, predisposing factors, symptoms, imaging, and clinical, laboratory, histological, and microbiological data of patients managed for SBO at the University Hospital of Brescia (ASST Spedali Civili) between 2002 and 2017 were retrospectively reviewed. Patients were included in different etiological groups. The topographic distribution of magnetic resonance (MR) abnormalities was recorded on a bi-dimensional model of skull base, on which three different patterns of inflammatory changes (edematous, solid, or necrotic) were reported. In patients with a history of radiotherapy, the spatial distribution of SBO was compared with irradiation fields. The association between variables and etiological groups was verified with appropriate statistical tests. A classification tree analysis was performed with the aim of inferring a clinical-radiological diagnostic algorithm for SBO. The study included 47 patients, divided into 5 etiological groups: otogenic (n = 5), radio-induced (n = 16), fungal (n = 14), immune-mediated (n = 6), and idiopathic (n = 6). At MR, five types of topographical distribution were identified (central symmetric, central asymmetric, orbital apex, sinonasal, maxillary). In patients with a history of radiotherapy, the probability to develop SBO was significantly increased in areas receiving the highest radiation dosage. The analysis of patients allowed for design of a classification tree for the diagnosis of SBO. The integration of clinical and radiologic information is an efficient strategy to categorize SBO and potentially guide its complex management⁶.

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