Do Dental Infections Really Cause Central Nervous System Infections?

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KEYWORDS

• Cavernous sinus thrombosis • Brain abscess • Dental • Odontogenic

In the post–World War I antibiotic era, the prevalence of central nervous system (CNS) infection is estimated to be 1 per 100,000 population.[1] The literature is replete with anecdotal case reports of CNS infections of apparent dental etiology. Conversely, it is widely cited that the incidence of CNS infection of dental etiology is only in the range of 1% to 2%.[2]

Before attempting to answer the question if dental infections really cause CNS infections, we define CNS infection as a condition that includes meningitis, subdural empyema, cerebritis, encephalitis, septic thrombophlebitis, and brain abscess.

Acute bacterial meningitis (viral, tuberculous, parasitic; fungal not included in this discussion of dental etiology) is the immediate effect of bacteria in the subarachnoid space causing an inflammatory reaction in the pia mater and arachnoid as well as in the cerebrospinal fluid (CSF). The large-scale inflammation of meningitis is secondary to the bacterial invasion as well as the immune reaction. The immune response is trifold, resulting in vasogenic cerebral edema, interstitial edema, and cytotoxic edema.

Subdural empyema is an intracranial suppurative process between the inner surface of the dura mater and the outer surface of the arachnoid. This condition is more common in men than women, a feature for which there is little explanation.

Subdural empyema usually originates in the frontal and sphenoid sinuses to the frontal lobe or in the mastoid air cells to the temporal lobe. A collection of subdural pus may range up to 200 mL. This collection may be visualized on computed tomography (CT) with increased meningeal enhancement.

Encephalitis is an acute inflammation of the brain. Encephalitis with meningeal involvement is known as meningoencephalitis. Although usually viral, bacterial and parasitic forms are common in immunocompromised patients. Typical neurologic signs and symptoms include fever, headache, nausea, vomiting, delirium, and seizures. CT or magnetic resonance imaging (MRI) may not be diagnostic. On lumbar puncture, the CSF contains elevated protein level, normal glucose level, numerous white blood cells, and antibodies to the offending organism.

Cerebritis is an inflammation of the cerebrum. It is a focal nonencapsulated purulent infection of the brain parenchyma or immature brain abscess. Cerebritis is characterized by vascular congestion, edema, petechial hemorrhages, and cerebral softening. This condition is ill defined on contrast-enhanced CT scan; it presents with an irregular nonhomogeneous central area of low intensity with mass effect and peripheral enhancement in the form of an incompletely formed ring. MRI is

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actually more sensitive than CT in the early cerebritis stage. If untreated, cerebritis will proceed to a brain abscess in 1 to 2 weeks.

Haymaker,[3] in his classic 1945 Army Institute of Pathology review of 28 fatal CNS infections after tooth extraction, cited 4 cases of meningitis, 3 cases of subdural empyema, and 1 case of encephalitis of dental etiology. Since 1945 in the postantibiotic era, the literature is largely devoid of these types of CNS infections of dental etiology.

Therefore, this article focuses on brain abscess and septic cavernous sinus thrombophlebitis of dental etiology.

CAVERNOUS SINUS THROMBOSIS

The dural sinuses drain blood from the brain ultimately into the internal jugular veins. The largest ones involved in septic thrombophlebitis are the superior sagittal sinus, lateral (transverse) sinus, straight sinus, and cavernous sinus. The cavernous sinus is the intracranial sinus most often implicated in dental or odontogenic infection.

The cavernous sinus is, in fact, a bilateral sinus cavity bordered by the temporal and sphenoid bones lateral to the sella turcica (**Fig. 1**). It receives blood from the superior and inferior ophthalmic veins, as well as the sphenoparietal sinus, superficial middle cerebral vein, and pterygoid plexus of veins. The cavernous sinus drains predominantly into the superior and inferior petrosal sinuses and ultimately into the internal jugular vein as well as the emissary veins passing through various cranial foramina.

Contents of the cavernous sinus include cranial nerves III, IV, V_1, V_2, and VI and internal carotid artery (**Fig. 2**). In fact, the cavernous sinus is the only place in the body where an artery travels completely through a venous structure. The abducens nerve passes below the sphenopetrous ligament, which forms a narrow fibrous canal, Dorello canal. Cranial nerve VI then runs with the internal carotid artery in the lateral wall of the cavernous sinus. Therefore, abducens nerve palsy is usually the first cranial nerve injury seen in cavernous sinus thrombosis (CST).

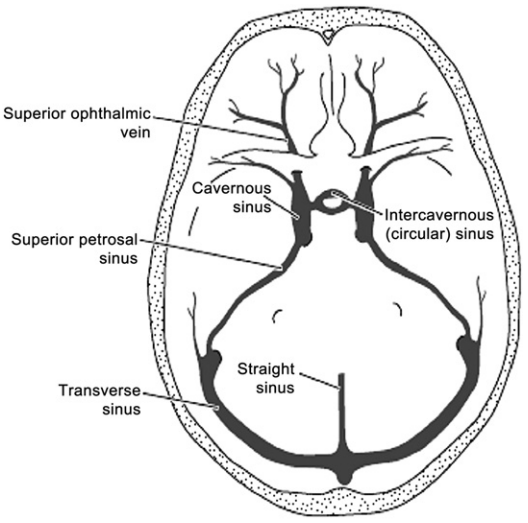

Fig. 1. Axial view of the cavernous sinus.

CST, or, more to the point, septic thrombophlebitis, implies an infected blood clot in the cavernous sinus. The patient with CST may present with high fever, headache, photophobia, nausea, vomiting, and signs of systemic toxicity. Obstruction of the ophthalmic veins leads to chemosis, proptosis, and edema of the ipsilateral eyelids, forehead, and nose. Engorgement of the retinal veins may be followed by retinal hemorrhages, papilledema, eye pain, and decreased visual acuity. Cranial nerve involvement (III, IV, V_1, VI) leads to ptosis, ophthalmoplegia, and supraorbital paresthesia. Within 24 to 48 hours, spread of the clot through the circular sinus to the contralateral cavernous sinus may result in bilateral signs and symptoms.

The cause of CST is 2-fold: hematogenous spread of septic emboli to the cavernous sinus via the blood supply of the head and neck or by direct extension of contiguous space infections. Most cases of CST are secondary to infections of the paranasal sinuses (ethmoid, sphenoid, frontal), nasal cavity, tonsils, middle ear, orbit, skin of the nose and face, or teeth.

Predisposing conditions to CST are similar to those for all CNS infections. These conditions include previous head trauma, particularly penetrating cranial injuries, even after intracranial neurosurgical procedures. Immunocompromised patients, including transplant recipients on immunosuppressive medications, patients with cancer on chemotherapy, and human immunodeficiency virus (HIV)-positive patients, are all at increased risk for CNS infection. Patients with nonoperated congenital cardiac cyanotic malfunctions are at increased risk for CNS infection due to septic emboli passing through a right-to-left shunt, bypassing the pulmonary filter. The same mechanism is seen in patients with hereditary hemorrhagic telangiectasia and pulmonary arteriovenous fistulas. Septic embolization from bacterial endocarditis or pulmonary embolism from tuberculosis or pneumonia increases the risk of CNS infection.

The 2 predominant pathogens in reported cases of CST are *Staphylococcus aureus* (50%–60%) and β-hemolytic streptococcus (20%), followed