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FUNDAMENTALS OF THE ETIOPATHOGENESIS, CLINICAL FEATURES AND PATHOMORPHOLOGY OF CAVERNOUS SINUS THROMBOSIS

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Abstract. Cavernous sinus thrombosis (CST) is a rare and potentially dangerous disorder that may complicate facial infections, sinusitis, orbital cellulitis, pharyngitis, or otitis, and may also develop after traumatic injury or surgery, especially in the presence of a thrombophilic disorder. Early recognition of CST, which is often accompanied by fever, headache, visual disturbances such as periorbital swelling, and ophthalmoplegia, is critical for a favorable outcome. Despite modern treatment with antibiotics and anticoagulants, the risk of long-term complications such as vision loss, diplopia, and stroke remains significant. Cavernous sinus thrombosis is usually infectious in origin, but may also be aseptic. Septic cases may follow central facial infections, particularly in the dangerous triangular facial area (from the corners of the mouth to the bridge of the nose). These include abscess or cellulitis, sinusitis (especially posterior sphenoiditis and ephemoiditis), dental infections, dental extractions or procedures (including posterior periosteal blocks penetrating the pterygoid plexus), upper facial surgery, otitis media, and mastoiditis. Aseptic causes are less common than septic causes and include trauma, surgery, or pregnancy.

Key words: sinusitis, loss, diplopia, stroke, fever, headache, ophthalmoplegia

Introduction. A variety of infectious organisms can cause cavernous sinus thrombosis, although most are bacterial. Staphylococcus aureus may account for up to two-thirds of cases, and methicillin resistance should be considered. Other common organisms include Streptococcus species (approximately 20% of cases), Streptococcus pneumoniae (5%), Gram-negative species such as Proteus, Haemophilus influenzae, Pseudomonas, Fusobacterium, Bacteroides, and Gram-positive species such as Corynebacterium and Actinomycetes. Some of these (Bacteroides, Actinomycetes, Fusobacterium) are anaerobes. Fungal infection in cavernous sinus thrombosis is

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less common but may include aspergillosis (the most common), zygomycosis, or coccidioidomycosis in immunocompromised individuals. Rare precursors to cavernous sinus thrombosis may include parasites such as toxoplasmosis, malaria, and trichinosis, as well as viral causes such as herpesvirus, cytomegalovirus, measles, hepatitis, and human immunodeficiency virus (HIV) [8, p. 14; 10, p. 159; 12, p. 46; 13; 14, p. 110]. Immunosuppression such as uncontrolled diabetes mellitus, steroid use, cancer, or chemotherapy may pose a risk not only for the development of cavernous sinus thrombosis but also for the development of complications.

The most significant risk factors are facial infections, acute sinusitis, and periorbital infections. Thrombophilia is a significant risk factor for cavernous sinus thrombosis. Women who are pregnant, postpartum, or taking oral contraceptives or hormone replacement therapy may have an increased risk. A variety of thrombophilic genetic disorders can lead to cavernous sinus thrombosis. These include factor V Leiden mutation, prothrombin G20210A mutation, deficiency of antithrombin III, protein C, or S, or increased factor VIII. Acquired disorders such as antiphospholipid antibody syndrome, hyperhomocysteinemia, heparin-induced thrombocytopenia, and obesity may also contribute to the risk of cavernous sinus thrombosis. Other risk factors for thrombosis include severe dehydration, such as in hyperosmolar non-ketotic state, nephrotic syndrome, and sheath cell disease.

Conclusions. Orbital cavity thrombosis is so rare that its prevalence is difficult to estimate. Since orbital cavity thrombosis accounts for approximately 1-4% of the overall incidence of cerebral venous thrombosis and sinus thrombosis, which has an annual incidence of about two to four per million persons per year, with a higher prevalence in children, it can be estimated that the annual incidence of orbital cavity thrombosis may be approximately 0.2 to 1.6 per 100,000 persons per year. It is unclear whether there is a male or female predominance among cases of orbital cavity thrombosis. Although there is a 3:1 female predominance among cases of cerebral venous thrombosis, this may not be the case for orbital cavity thrombosis. Weerasinghe reported a male to female ratio of 2:1 in 88 cases in adults with septic manifestations. Similarly, Tatai et al. and Smith et al. reported a slight male predominance in 35 and 12 cases, respectively.

Historically, cerebral venous thrombosis has been more common in children and neonates than in adults, although the impact of routine vaccination and frequent use of antibiotics (eg, for otitis media) on this association remains unclear. Incidence and mortality may be declining, most likely due to the availability and use of antibiotics. The orbital cavities (one on each side of the sella turcica, above and to the side of the sphenoid sinuses, anteriorly by the periosteum of the

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superior orbital cleft, posteriorly by the rocky part of the temporal lobe of the parietal lobe) are interwoven cavities formed by layers of the dura mater and filled with venous blood. This blood drains through the superior and inferior orbital veins and the superficial cortical veins anteriorly, and then drains into the lateral jugular plexus posteriorly through the superior and inferior jugular veins.

The orbital cavity has been called an "anatomical jeweler's box" because it has intimate relationships with several important structures. The orbital cavity contains the horizontal segment of the internal carotid artery, the sympathetic plexus, and the cranial nerves VI (abductors) in the middle. The outer layers of the lateral wall of the carotid ganglion are crossed by cranial nerves III (oculomotor), V (first and second branches, ophthalmic and maxillary, trigeminal), and IV (geniculate).

Septic thrombosis of the orbital cavity occurs in the following cases:

Local extension, often from veins without valves of the face and orbit.

Associated infections such as sinusitis (perhaps the most common cause), especially sphenoiditis and ethmoiditis (Press).

Cellulitis or facial abscess (especially in the dangerous triangle of the face formed by the corners of the mouth and bridge of the nose).

Periorbital and orbital cellulitis.

Pharyngitis.

Angina.

Otitis.

Mastoiditis.

Dental infections.

The mechanism is as follows:

Embolization of bacteria and other infectious organisms that cause thrombosis, which can trap the infection within the orbital cavity.

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Thrombosis of the orbital cavity leads to decreased blood flow from the facial vein and the superior and inferior orbital veins, resulting in facial and periorbital edema, ptosis, exophthalmos, chemosis, discomfort and pain when moving the eye muscles, swelling of the optic nerve, dilation of the retinal veins and loss of vision. The absence of valves in the cerebral sinus system allows blood to flow through the communicating veins into the orbital cavity and the thrombus can spread to the cerebral sinuses. Also, the communication between the right and left orbital cavities via the intercavitary sinuses anterior and posterior to the sella turcica allows the spread of thrombus and infection from one side to the other. Local compression and inflammation of the cranial nerves can cause several partial or complete cranial nerve lesions, including: Diplopia due to partial or complete external ophthalmoplegia caused by compression of the VI (abductors), III (oculomotor), and IV (geniculate) nerves. Limited abduction of the eye in VI nerve palsy is the most common initial manifestation, often progressing to inability to move the eye in any direction when nerves III, IV, and VI are involved. Internal ophthalmoplegia (non-reactive pupil) caused by loss of sympathetic fibers from the short ciliary nerves (resulting in miosis) and/or loss of parasympathetic fibers from cranial nerve III (resulting in mydriasis).Numbness or tingling (around the eyes, nose, forehead) and loss of the corneal blink reflex from the ophthalmic nerve, a branch of the trigeminal nerve (V).Pain, tingling, or numbness of the face from compression of the maxillary branch of the trigeminal nerve. Septic thrombosis of the orbital cavity can lead to central nervous system complications or infectious pulmonary lesions. Since the jugular vein and luminal vascular space have no valves, this venous blood can interact with the cerebral sinuses and cranial and articular veins, which can lead to meningitis, purulent inflammation of the jugular brain, or cerebral abscess. Infection can spread through the jugular vein to the pulmonary vasculature, causing septic emboli or abscess, pneumonia, or purulent pleurisy. Stroke can occur with stenosis of the carotid artery, vasculitis, or hemorrhagic infarction due to progression of thrombosis of the cortical veins. Hypopituitarism can occur due to ischemia or direct spread of infection.

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