Sinus pericranii

Sinus pericranii is a venous anomaly where a communication between the intracranial dural sinuses and dilated epicranial venous structures exists. That venous anomaly is a collection of nonmuscular venous blood vessels adhering tightly to the outer surface of the skull and directly communicating with intracranial venous sinuses through diploic veins. The venous collections receive blood from and drain into the intracranial venous sinuses. The varicosities are intimately associated with the periostium, are distensible, and vary in size when changes in intracranial pressure occur.

Classification

Both dominant and accessory sinus pericranii, as determined by the intracranial venous drainage pattern, have been described. The dominant variety drain a significant proportion of the intracranial venous outflow while the accessory variety have minimal or no role in this.

Etiology

The nature of this malformation remains unclear.

Congenital, spontaneous, and acquired origins are accepted.

The hypothesis of a spontaneous origin in the current case of SP is supported by no evidence of associated anomalies, such as cerebral aneurysmal venous malformations, systemic angiomas, venous angioma dural malformation, internal cerebral vein aneurysm, and cavernous hemangiomas.

Treatment

Accepted guidelines or recommendations concerning the management, diagnosis, and treatment of SP are still lacking.

Angiography plays a crucial role in the classification of SP and choice of the optimal treatment. Only accessory SP is amenable to treatment, whereas dominant SP must be preserved. The endovascular approach is becoming increasingly relevant and has proven to be safe and effective 1).

Ellis et al describe a simple and unique method for determining whether intracranial venous outflow may be compromised by sinus pericranii treatment. This involves performing catheter angiography while the lesion is temporarily obliterated by external compression. Analysis of intracranial venous outflow in this setting allows visualization of angiographic changes that will occur once the sinus pericranii is permanently obliterated. Thus, the safety of surgical intervention can be more fully appraised using this technique 2).

The surgical treatment involves the resection of the extracranial venous package and ligation of the emissary communicating vein. In some cases of SP, surgical excision is performed for cosmetic reasons. The endovascular technique has been described by transvenous approach combined with direct puncture and the recently endovascular embolization with Onyx.
Case series

2009

10 patients who had undergone surgery for SP and 6 patients with concomitant craniosynostosis and SP. The mean age of the patients at presentation was 3.7 years. To identify characteristics of SP with high operative risk, 8 cases in this report and 11 previously reported cases of SP with sufficient information were categorized on the basis of the number and size of SP, the number and size of transcranial channels, the venous drainage type, and the amount of blood loss. Hemorrhage amounts were classified into 3 grades based on the description of intraoperative blood loss.

Sinus pericranii not associated with craniosynostosis were resected without any postoperative morbidity. Sinus pericranii associated with craniosynostosis were preserved. After craniofacial reconstruction, 2 cases of SP with craniosynostosis regressed, completely in one patient and partially in another. These 2 patients with SP were confirmed to have compromised intracranial sinus before craniofacial reconstruction. Among a total of 19 patients, multiplicity or size (> 6 cm) of SP (p = 0.036) and multiplicity (> 3) or size (> 3 mm) of transcranial channels (p = 0.004) was associated with more severe hemorrhage grade. Sinus pericranii with peripheral venous drainage (drainer type) was not associated with hemorrhage grade after classification into 3 grades (p = 0.192). However, all 3 cases of SP with massive Grade 3 hemorrhage were the drainer type. Hemorrhage grade was correlated with the number of risk factors for SP (r = 0.793, p < 0.001).

Three risk factors of SP and the presence of compromised intracranial sinus are markers for highrisk SP. “Squeezed-out sinus syndrome” is suggested as a concept for SP associated with compromised intracranial sinus, mainly caused by craniosynostosis. Sinus pericranii in squeezed-out sinus syndrome probably serves as a crucial alternative to venous drainage of the brain with intracranial venous compromise. Conservative treatment for such patients with SP is recommended.

Case reports

2017

A 2-year-old male with achondroplasia, Sinus pericranii (SP), and craniocervical junction stenosis. The latter two defects required surgical correction. SP is an underappreciated malformation that Scott et al. propose may be induced by intracranial hypertension. This case appears to be the first report of this condition in achondroplasia.

1995

Cerqueira et al describe 2 cases of congenital sinus pericranii associated with intracranial venous anomalies. After a review of the literature concerning this association they consider the possible etiopathogenic mechanisms and treatment implications.


