Sinus Pericranii: CT and Color Doppler Ultrasound Imaging Findings in a Case of Spontaneous Thrombosis

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ABSTRACT: Sinus pericranii (SP) is a rare, usually asymptomatic condition characterized by communication of pericranial varicosities with an underlying dural sinus. We report a case of spontaneous thrombosis of sinus pericranii presenting as focally tender, nonreducible mass lesion different in character from the baseline venous abnormality. CT and colour Doppler Imaging were performed.

Key Words: sinus pericranii; computed tomography; color Doppler sonography; thrombosis

INTRODUCTION

Sinus pericranii (SP) is a rare, usually asymptomatic condition characterized by a large communication between the intra- and the extracranial venous drainage pathways in which blood may circulate bidirectionally through dilated veins of the skull. (1-3). Symptoms are infrequent but include headache, vertigo, feelings of fullness, or local pain (2). Most SPs become clinically apparent as nonpulsatile soft-tissue masses that are located in the frontal region along or close to the midline and connect pericranial veins with the superior sagittal sinus (SSS) through a bony defect (1-7).

We describe CT and color Doppler Imaging findings in a spontaneous thrombosis of SP case.

CASE REPORT

A ten months old infant presented with nodular lesion located along posterior parietal region in midline associated with significant overlying scalp tenderness. Parents told lesion was compressible up to few weeks ago. It was changing in diameter with the crying. Later on sudden increase in prominence and nonreducibility of lesion developed.

Physical examination revealed a noncompressible lesion unlikely to classical SP. Unenhanced head CT demonstrated a 1 cm bony defect of the calvaria in the posterior parietal region in midline (figure 1). Superficial to the bony defect was a prominent 13x 7 mm soft tissue mass. The attenuation of soft tissue was near to a cystic lesion. It was expanding intracranially through bony defect. With the clinical findings it has been diagnosed as spontaneously thrombosed SP. On the gray scale sonographic examination, a cystic lesion communicating with intracranial dural sinuses was seen (figure 2). Color Doppler imaging revealed a cystic lesion with no flow inside. Lesion was communicating with intracranial dural sinuses (figure 3).
Figure 1. A bone defect with 1 cm width is seen in the posterior parietal area in the CT. A prominent 13x 7 mm soft tissue mass is noticed superficial to the bony defect.

Figure 2. On this gray scale sonographic image, a cystic lesion communicating with intracranial dural sinuses can be seen.

Figure 3. Color Doppler imaging revealed a cystic lesion with no flow inside. Lesion was communicating with intracranial dural sinuses.
**DISCUSSION**

Sinus pericranii consists of anomalous extracranial drainage of the intracranial circulation occurring through diploic emissary veins into an enlarged varicoid venous pouch connected to the subgaleal and scalp venous systems was first reported by Hecker in 1845 and named by Stromeyer in 1850 (1,4,8).

Etiology consists of congenital, acquired and spontaneous development. Congenital development supported by accompanying anomalies (5,7). Our case had no history of trauma. Lesion considered as congenital.

In a recently published series, a deep venous anomaly was found in eight out of 15 patients with a sinus pericranii (3). This finding emphasizes the need for precise analysis of the cerebral venous anatomy when planning the treatment of a SP (1). CT angiography and conventional angiography can be applied for this purpose.

SP should be distinguished from other subcutaneous masses of scalp including subcutaneous haematoma, pseudomeningoele, cranioschisis, growing skull fracture, scalp dermoid cyst and skull tumor (4,5). Subepicranial varix may show blood flow but it has no communication intracranially. Return of venous blood and change in size with position and intrathoracic pressure (crying) are important clinical features. Our case had history of size change with crying. But because of thrombosis it was disappeared at presentation. As in our case thrombosis may cause a sudden alteration in character of the lesion from soft and painless to firm and painful is of concern to both the patient and physician.

CT may help to show bony defect at calvarium and color Doppler may help to show blood flow when there is no thrombosis.

SP is commonly asymptomatic, and classified as dominant if the major venous flow is through the SP and accessory if it concerns a minor part of the venous flow. If dominant lesion is diagnosed it should not be treated surgically.

**CONCLUSION**

Radiologists should remember SP at the differential diagnosis of subcutaneous soft tissue mass in scalp. Bone defect or thinning near to mass and change in the size with position should help differential diagnosis. Color Doppler should be used frequently to support diagnosis. Before treatment it is important to exclude cranial venous anomalies which may accompany SP.

**REFERENCES**
