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Sinus Pericranii: Diagnostic and Therapeutic Considerations.

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ABSTRACT

Sinus pericranii (SP) is a rare vascular anomaly of the venous drainage between intracranial and extracranial system via diploe of skull. It is a generally benign condition and presents as a soft scalp swelling in the region of major venous sinuses and appears with patient in recumbent position and disappears with erect position. Once suspected and treatment is planned, a detailed analysis of drainage pattern of the lesion and venous system must be done. Most of the time, it is innocuous, but a misdiagnosis can lead to disaster. The aim of this study was to report on five cases of sinus pericranii operated at our institute with consideration of the preoperative evaluation of surgical risk. Two patients of Sinus pericranii associated with craniosynostosis were not included in this study. The study population consisted of 5 patients, 4 patients with congenital and one with traumatic origin. There were three male and two female. Three of them were child of Eleven month, one and half years and another of three years. Other two were young adults (one male and other female). One child presented to us in hemodynamically unstable condition after wrongly diagnosed as epidermoid cyst and inadvertent incision of the lesion. All five sinus pericranii patients were resected without any postoperative morbidity. This was followed by a review of literature.

Keywords: sinus pericrania, anomaly, skull, sinuses

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INTRODUCTION

In normal conditions, there is no obvious connection between the intracranial and extra cranial venous circulation. Sinus pericranii is a rare venous varix in extracranial location connected to the intracranial venous system (1).

Sinus pericranii was first described by Hecker in 1845 as 'varix spurius

Circumscriptus venae diploicae frontalis' (2). In 1850 Stromeyer described this anomaly as cyst containing circulating blood and communicating with intracranial sinus and proposed the term sinus pericranii (3). It is found beneath or in the periosteum of the cranial vault and connected with an intracranial sinus through anomalous diploic veins of congenital or acquired origin. Sinus pericranii presents mostly in pediatric age group as small circumscribed swelling of the scalp. Some of cases though present from childhood can come for treatment in young adult as in one of our case. Most of sinus pericranii are of congenital origin. Some cases have been attributed to trauma, including one of our cases. It is appropriate now to reconsider this disorder, as new neuroradiological techniques have clarified the profile of this disease more precisely.

The aim of this study was to clarify this rare vascular entity, define the role of angiography(CT, MR and DSA) and other investigations in preoperative assessment. The authors also suggested a diagnostic and therapeutic protocol and role of surgery based on their experience.

METHODS

Eleven patients with vascular lesions in the regions of venous sinuses(Superior sagittal and Transverse sinuses) and suspected to be Sinus Pericranii were investigated after admission in our institute in seven years period (from January 2011 to January 2017). Seven cases were diagnosed as sinus pericranii. Out of these two cases were associated with craniosynostosis and were not included in this study. Rest five cases were operated. Patients were followed up in outpatient department at six months interval for evidence of recurrence. All cases are. The summary of these cases are given in table 1.

These cases are described in detail; the summary of this is given in table 1.

Case 1

One and half year old boy was admitted with soft swelling in right posterior frontal region in midline. The child was born normally at full term. His physical and mental development had been normal. He had no history of head trauma. His parents noticed swelling in mid parietal region when child was 6 month old. This swelling gradually increased in size.

In recumbent position the lesion was 2.5cmX 2.5cm in size. The boundaries of the lesion were well demarcated. The lesion was soft, fluctuant and elastic. The lesion did not move over cranium but skin overlying the lesion was movable. There was no discoloration of skin over the lesion. The palpation of skull under the lesion was also normal. In this case there was associated hemangioma on the face.

Plain X ray of skull was normal, CT scan showed isodense lesion with homogenous enhancement with contrast. CT angiogram could not demonstrate abnormal communication between lesion and intracranial venous sinuses.

Operation-After usual prepping and draping a slightly parasagittal incision was made near the vertex on the right side, taking care not to incise the venous anomaly. The lesion was adherent to skull and looked like collection of multi loculated cyst. Using bipolar and sharp dissections, numerous scalp veins and three large feeders to sinus pericranii through diploic veins and sagittal sinus was identified cauterized and ligated. The lesion was extirpated in enblock, with meticulous coagulation of tiny vessels. Bleeding from Diploic veins were stopped using bone wax. The pericranium was dissected upto skull defect through which sagittal sinus communicated with the lesion (sinus pericranii). After this, pulsation from the malformation disappeared. After surgery the absence of the residual sinus pericranii and abnormal scalp feeders and tributaries were confirmed using Valsalva maneuver and putting patient in the Trendelenberg position.

Macroscopically, the specimen was a disc of spongy tissue. The outer surface next to skin was locular and pinkish red. The inner surface next to bone was thin but tough. There were several holes corresponding to tiny channels between lesion and intracranial venous system. Microscopic examination showed fibrous connective membrane, irregular sinuses lined by endothelium. There were abundant small vessels in spaces between sinuses.

Patient was discharged home after 48 hours. At follow up the patient has no recurrence of the lesion after six years.



Fig 1: Case 1 One and half year old boy admitted with soft tissue swelling in left posterior frontal region near midline. Parents noticed swelling when child was 6 month old. This swelling gradually increased in size.

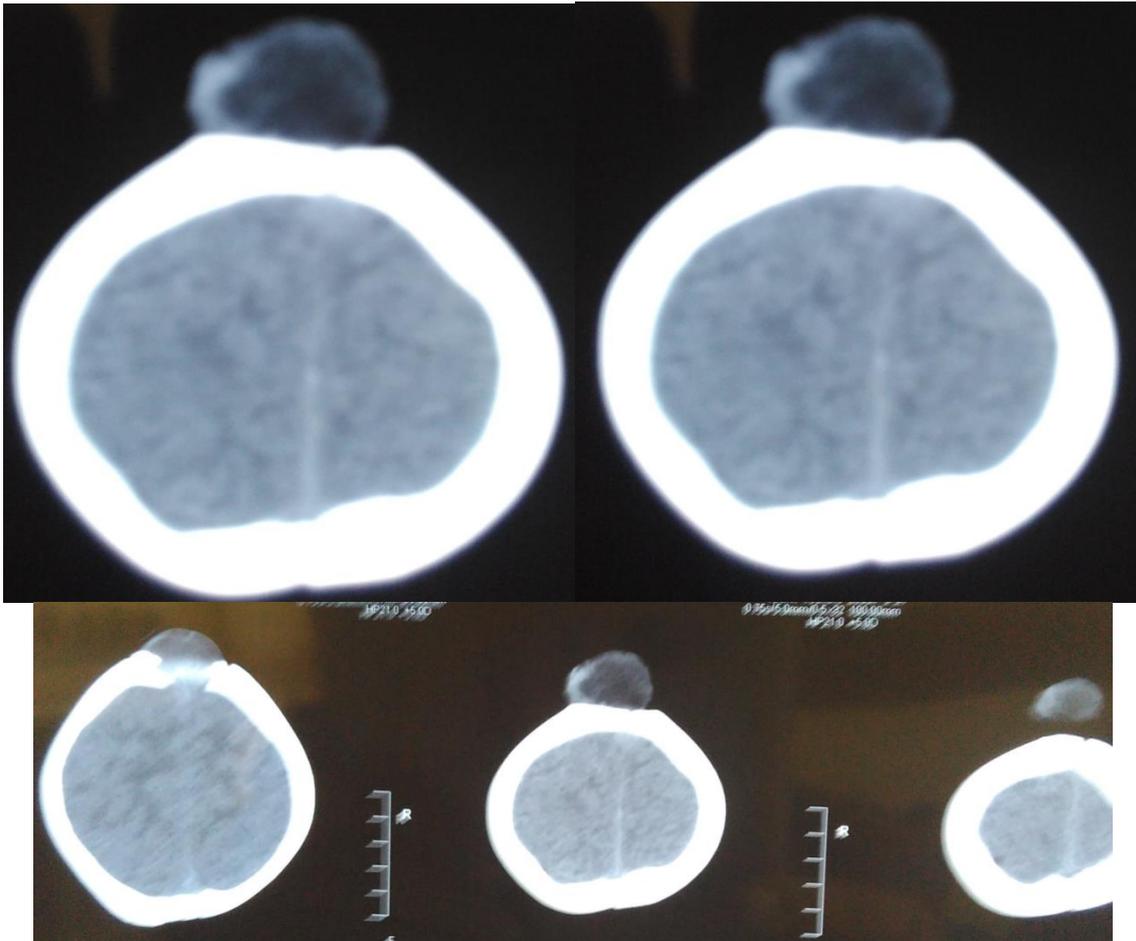


Fig 2: Case 1 Post contrast CT scan showed connection of the lesion to superior sagittal sinus.

Case 2

A 24 year female presented with soft tissue swelling in midline of frontal region since birth. Initially swelling was very small, but for last one year it has increased in size. Patient complained of extreme pain in forehead and scalp. Swelling was absent when patient was sitting or standing in upright position. It will appear only when patient will bend, cough or lie in prone position. On examination swelling was 2.8 X 2.8 cm in size, soft globular and fluctuant and reducible in pressure. The swelling increased in size while patient bent and with Valsalva maneuver. It was non pulsatile, but has positive impulse on coughing. The skin over lesion was not discolored and moved freely. The swelling could not be moved over cranium. Plain X ray skull showed irregular thinning of the frontal bone with small defect in midline corresponding to the location of lesion. CT scan showed an isodense lesion in mid frontal region. There was homogenous enhancement following contrast. CT angiogram was non contributory. MR angiogram showed normal arterial phase. The venous phase showed persistence of contrast. No abnormal vein was identified to communicate between lesion and sagittal sinus.

Operation- An eyebrow incision was made and separated from underlying cystic structure. Periosteum was divided at the boundary of the mass and carefully dissected toward the neck of mass. There were many small vessels communicating between the lesion and intracranial space through bone. Underlying bone was thinned out, but the surface was fairly smooth and bone cortex was preserved. Cyst could be excised completely.

Extirpated specimen contained multi loculated cystic structure. Microscopic examination showed periosteum connective tissue with some granulation. At one year follow up, patient had no recurrence of the lesion.

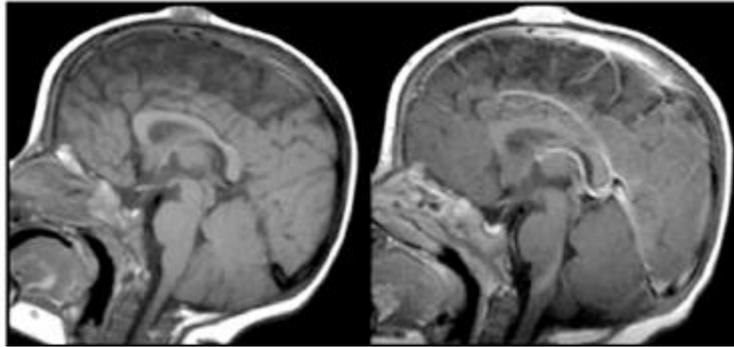


Fig 3: Case 2 24 year female presented with soft swelling in midline frontal region since birth. Initially swelling was very small, which had increased in size and had extreme pain in forehead and scalp for last one year. Swelling was absent in sitting or standing in upright position. It will appear only when patient will bend, cough or sneeze.

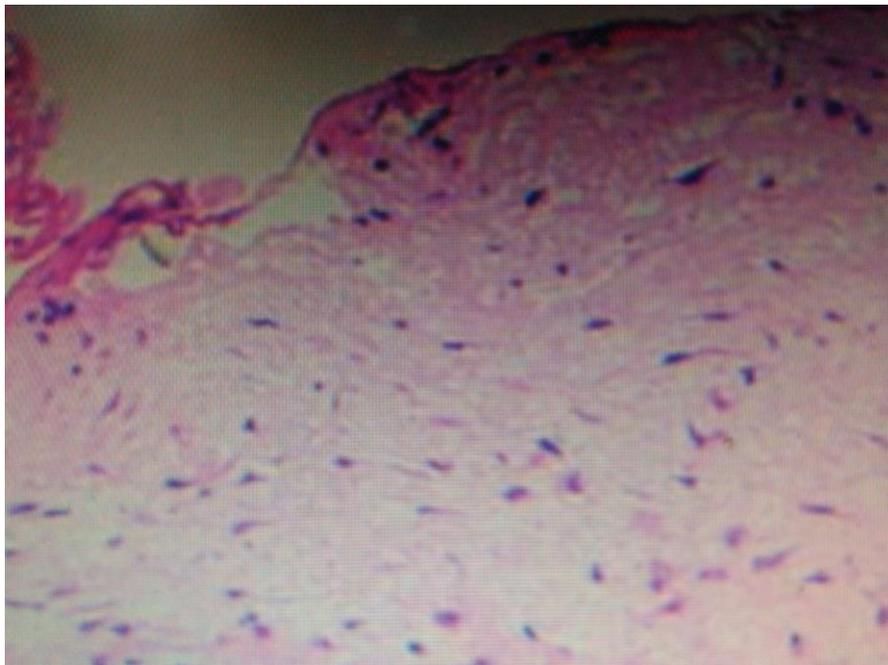


Fig 4: Case 2, Microscopic examination of the wall of the lesion showed thick fibrous connective tissue with large sinuses lined by endothelial cells and small non-muscular vessels. (Hematoxylin & Eosin X 80 original magnification).

Case 3

3 year old boy was referred from another Medical college in the city with history of swelling in midline in posterior frontal region since age of one year. It had recent increase in size. The child was seen at surgical OPD and was diagnosed to have epidermoid cyst. Patient had plain X-ray of skull which was normal. Routine hemogram and other routine investigations were normal. Patient was taken for surgery (excision of epidermoid cyst). During surgery, the wall of the lesion was inadvertently incised and it started bleeding torrentially. Somehow bleeding was controlled by packing and was sent to us.

When we received patient the lesion was not bleeding. Patient had very feeble pulse and BP was not recordable. We arranged four units of blood and after transfusing two units of blood, we decided to explore the lesion.

The patient was positioned supine with head end elevated to 30 degree under general anesthesia with endotracheal tube in situ. At the operation table previous dressing and packing was opened. The lesion was in midline in frontal region, just inside hairline. There was vertical surgical incision in the lesion and it was packed with gauze. Bleeding from incised wound was controlled by continuous manual pressure of the assistant. A semicircular incision was made on the right side of the lesion. The wall of the lesion was identified laterally. It was located beneath periosteum. It was cautiously detached from underlying skull surface and normal peripheral periosteum from all side. During the process of detachment many tiny venous communications through sieve like foramina were coagulated and cut between lesion and bone. Once these vessels were coagulated and cut, bleeding from the incised wound diminished significantly. Bleeding from large diploic emissary veins was closed with bone wax.

Fortunately, postoperative course was uneventful and was discharged from hospital after two week. Patient required one units of blood during surgery. Patient was not transfused in post operative period. Repeat CT scan of the child after 48 hours were normal. Histopathological examination of the lesion was consistent with sinus pericranii. Patient was doing well with no recurrence of lesion after follow up period of two years.

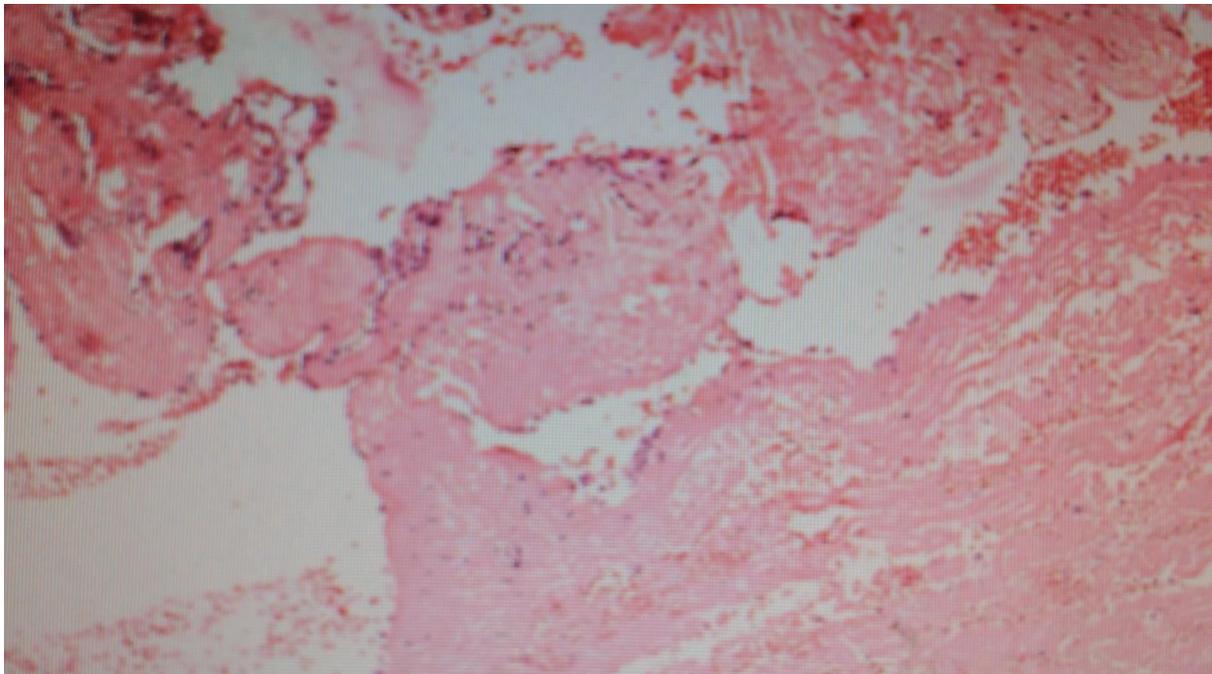


Fig 5: Case 3 Micro photograph of the wall of excised sinus pericranii showing the variable sized thin walled vessels lined by endothelium along with fibrocollagenous tissue and absence of muscular layer.(Hematoxylin and Eosin X 40 original magnification)

Case4

11 month girl child presented with a pinkish red mass of the size of 2cmX 2 cm in the midline in posterior frontal region. This has been present since birth. The mass varied in size with Valsalva maneuver or dependency. An underlying bony defect was palpable (due to non fusion of bones). There was no significant medical history. Physical examinations were normal. Plain skull films showed areas of irregular thinning of parietal bones without surrounding sclerosis. CT revealed a soft tissue density over the bone defect that enhanced after contrast administration. MRI (before and after IV gadolinium) demonstrated a soft tissue mass of mixed signal intensity with areas of signal void and enhancement indicating flowing blood. The diagnosis of sinus pericranii was confirmed by identifying communication of the vascular mass with underlying intracranial sagittal sinus. Due to risk of future complications, parents elected surgery. The operation was performed under general anesthesia. The soft tissue lesion was resected. The veins connecting lesion with underlying superior sagittal sinus were coagulated and excised. Residual bleeding from sinus wall was controlled using gelfoam wrapped with surgical and digital pressure over the sinus. Bleeding from a few emissary/diploic veins was stopped using bone wax. Histopathology examination showed cavernous vascular channels with thin vascular endothelium, which is typical for congenital sinus pericranii. Two month after operation repeat MRI showed total excision, with no residual lesion.



Fig 6: Case 4, A 11 month girl child presented with a pinkish red mass of the size of 2cmX 2 cm in the midline in posterior frontal region. This has been present since birth. The mass varied in size with Valsalva maneuver or dependency. An underlying bony defect was palpable (due to non fusion of bones)

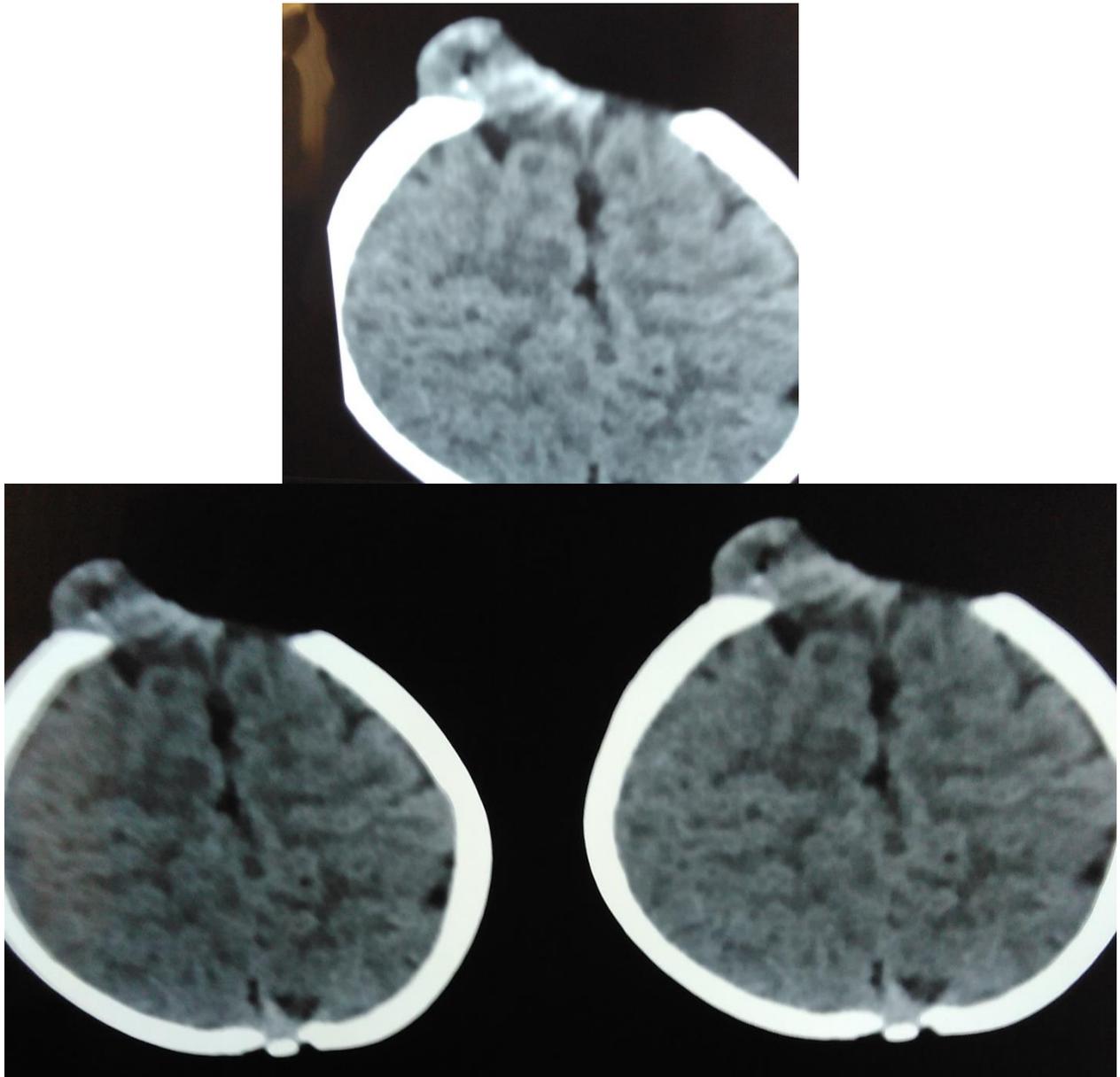


Fig 7: Case 4, CT scan of 11 month old child revealed a soft tissue density over the bone defect at sagittal sinus that enhanced after contrast administration

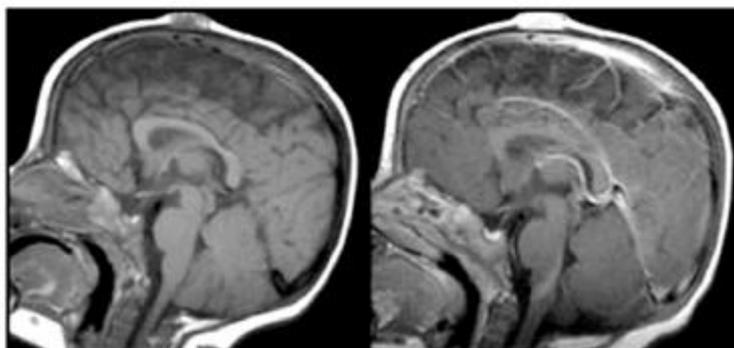


Fig 8: Case4, MRI sagittal T1WI and Sagittal T1WI Post gadolinium DTPA study showed evidence of sinus pericranii in frontal region.

Case 5

18 year boy was admitted with soft painless swelling in Median Parietal region. At the age of 8 year child had road traffic accident during which he was unconscious for some time and had injury to parietal region. Patient recovered completely after accident with no neurological deficit. Shortly after accident patient noticed, a soft mass in the parietal region in midline, when he was lying down. The mass was painless and disappeared with compression or while in sitting posture. It has not increased in size for last several years. Patient had occasional headache and vomiting and no other complaints.

On examination, patient had round soft painless mass 5 cm in diameter in right parietal region in midline in recumbent position. The mass disappeared in sitting position. There was no abnormality in overlying skin. The skull under lesion appeared depressed. Other physical and neurological tests and routine preoperative laboratory tests were normal.

Plain X-Ray of skull showed evidence of thinning of bone, beneath the lesion. CT scan showed evidence of isodense lesion in mid parietal region which enhanced with contrast. MR angiogram revealed connection of lesion to superior sagittal sinus through a significant vein.

At operation, a semicircular skin flap was made and separated from the underlying cystic structure. Periosteum was divided at the boundary of mass and was carefully dissected toward the neck of the mass. There were many small vessels communicating between the lesion and intracranial space. Bleeding could be controlled with difficulty. There were two large dilated emissary veins connecting lesion through the bone to sagittal sinus. These vessels were occluded with bipolar coagulation and were cut to free lesion from underlying bone. Bleeding from bony diploae was controlled with bone wax and prolonged manual pressure and raising head end. The lesion was excised from the skull bone. Patient had uneventful postoperative course.

Macroscopically it contained multiloculated cystic structure and microscopic examination showed periosteal connective tissue with some granulation tissue.

Table 1: Clinico radiological features, treatment and outcomes with patients with Sinus Pericranii

Sl. No.	Sex/Age	Type	Site	Clinical Presentation	Comorbidities	Imaging	Treatment	Outcome
1	Male/1 yr 6 mth	Accessory	Median posterior frontal	Soft tissue mass	Associated haemangioma on the face	Xray /CT/ CT angio/	Surgical Ligation	Permanent Disappearance
2	F/ 24 year	Accessory	Median Frontal	Soft tissue mass	Nil	Xray /CT/ CT angio/ MR / MRV	Surgical Ligation	Permanent Disappearance
3	Male/ 3 year	Dominant	Median Fronto parietal	Soft tissue cystic lesion, inadvertently incised and profusely bleeding with shock	Nil	Nil	Surgical Ligation and removal of cystic lesion	Permanent Disappearance
4	11 Month	Accessory	Median parietal region	Soft tissue mass in the midline at vertex, present since birth, varied in size with valsalva or dependency.	Nil	Xray /CT/ CT angio/ MR / MRV.	Surgical Ligation and removal of cystic lesion	Permanent Disappearance

				An underlying bony defect.				
5	Male/18 year	Accessory	Median Parietal region	Soft tissue cystic mass in posterior parietal region	Nil	Xray /CT/ CT angio/ MR / MRV/ DSA	Surgical Ligation	Permanent Disappearance

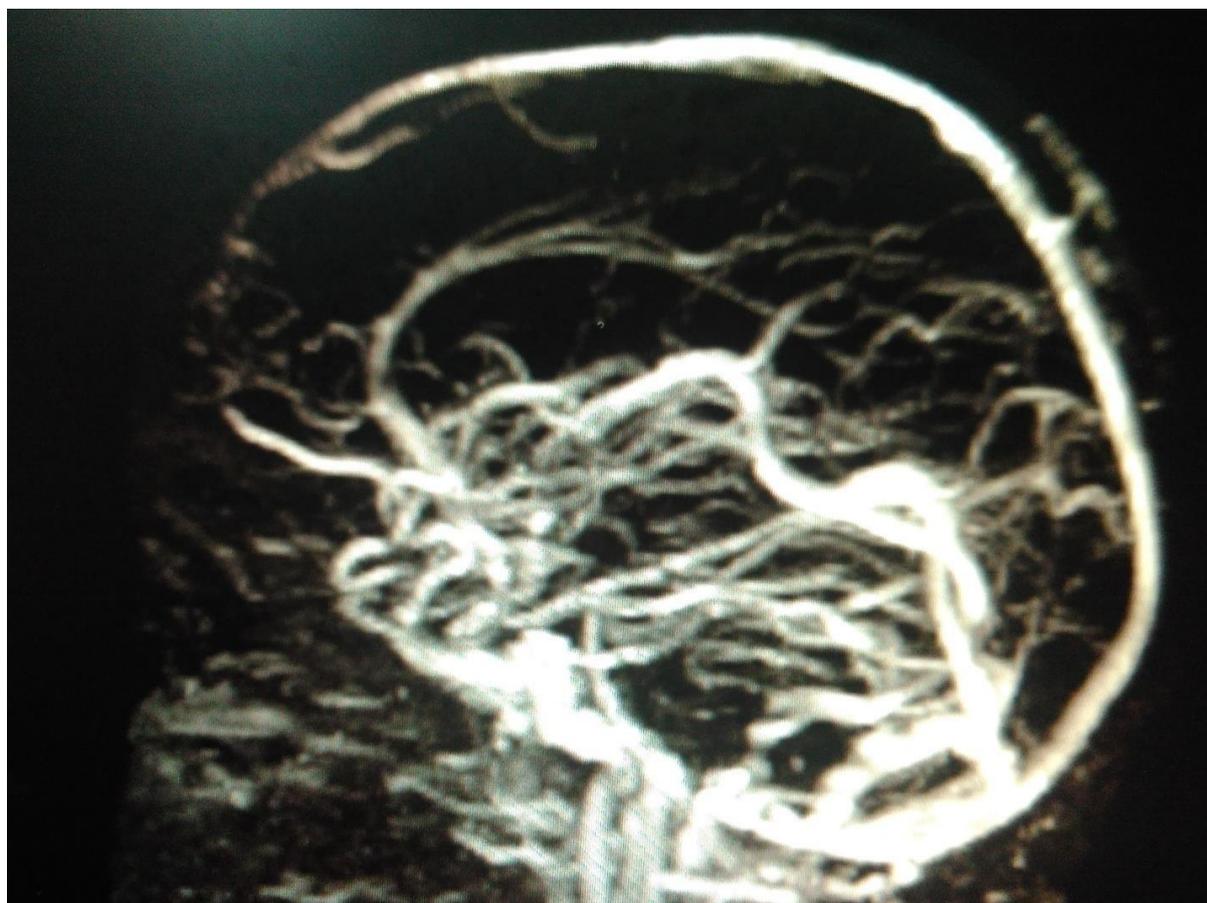


Fig 9: Case 5, Digital Substraction venogram showed connection of lesion from superior sagittal sinus through single vein

DISCUSSION

There are few published small series dealing with Sinus pericranii. Till date, approximately 150 cases were reported in the literature and most of them are individual case reports or small series of cases. The widespread agreement regarding the diagnosis, classification and management has not reached, due to a relative rarity of disease. Majority of cases are not classified with respect to dominance. It is now; appropriate to reconsider this disorder, as new neuroradiological techniques have clarified the profile of this disease more precisely. In this paper, we discuss our 7 year single centre experience with sinus pericranii. Out of seven cases seen in our institute two cases were associated with craniosynostosis and were not included in this study. All these cases were operated with good results.

Since the description by Hecker (1845) and Stromeyer (1850), many designation and classifications of this lesion had been reported (1, 2, 3 and 4).

Fevre and Modéc proposed the first classification system for Sinus pericranii in 1936. They described three pathophysiological types of sinus pericranii: 1) Closed system in which blood came from sinus and also

returned to it.; 2) Drainer system that act as collateral for intracranial flow and 3) Extracranial lesions draining into intracranial sinuses (5).

In 1967, Gerlach et al, defined sinus pericranii as a varix and classified into three types (varix simplex, varix racemous and varix herniosus) according to the size of venous communications between the extracranial venous sac and intracranial venous sinus (10). Newton TH and Troost BT believed that sinus pericranii was a venous malformation and described it as communication between intracranial and extracranial venous circulations via large tortuous, thinned wall vascular channels (12). Volkmann (1950) described two types sinus pericranii: "true sinus pericranii" which fills with increased intracranial pressure and disappears completely with compression of the tumor, and "pseudo sinus" which do not completely disappear with compression. They considered latter to be angioma or cavernoma of mainly venous components (13).

Many etiologies for sinus pericranii had been described. Mastinss' classification into congenital, spontaneous and traumatic is widely adopted (14, 15). Congenital pathogenesis was first proposed by Muller (16, 17). Spontaneous origin has been attributed to development of varix followed by pressure erosion of the skull or due to some chronic disease of skull vault (22). In regards to Pathogenesis of this anomaly Fractures of skull, injury to an emissary vein or direct sinus injury have been proposed as the pathogenesis of traumatic sinus pericranii. (3, 4, 20, 21).

Hahn described the histological appearance of a typical sinus pericranii, and stated that there was no histological difference between spontaneous and traumatic sinus pericranii (22).

Sinus pericranii is located most commonly in midline along the sagittal sinus. The most frequent site is frontal region followed by parietal region (1, 29, 37). Rarely has it been reported from a lateral location (23, 24, 25).

The exact location in relation to periosteum seems to be controversial. Ohata et al reported that lesion was beneath periosteum in three of their five cases. He described this condition as collection of venous blood vessels without tunica muscularis or 'venous hemangioma' adhering tightly to the outer surface of the skull bone and directly communicating with an intracranial sinus by way of many diploic veins of several sizes (6).

Most report suggests that Sinus pericranii is clinically benign lesions that predominantly raise cosmetic concern. There had been cases where, a case of sinus pericranii was misdiagnosed as epidermoid cyst of skull (27, 28) or Lipoma (1). We had also one case where, the sinus pericranii was misdiagnosed as epidermoid cyst and had iatrogenic injury to sinus pericranii leading to profuse bleeding leading to severe shock.

In Pre CT era it was diagnosed by conventional angiography and Sinography (direct injection of contrast medium to 'tumor') (1, 6, 18, 24). CT scan allows direct visualization of bony defect. Osteolytic lesions can be defined well using bone windows. It will show a subcutaneous mass that is slightly hyper dense compared to brain and subcutaneous tissue. This mass can be enhanced by intravenous injection of contrast medium depending on its extent of vascularity (26, 30, 31). MRI with or without MR angiography provides a definitive diagnosis of Sinus pericranii and its drainage into dural sinus. MR imaging will show signal void. Multiplanar MR images aid in diagnosis by showing the relationship of the lesion with the adjacent sinus (29, 30, 31). Sadler, et al presented the case studied by MR imaging that showed a mixed intensity signal, caused by turbulent flow. They suggested that signal would increase after the intravenous injection of gadolinium (30).

Digital subtraction angiography (DSA) is gold standard in diagnosis as it shows sinus in venous phase. Angiography can separate Sinus pericranii into two patterns, dominant and accessory. Gondalfo and colleagues proposed a more clinically useful categorization based on angiography into dominant and accessory. They explained that dominant sinus pericranii are untreatable, because they serve as a major venous outflow channel to intracranial compartment. In comparison, accessory sinus pericranii are highly treatable because only a small portion of intracranial venous outflow traverses them. Classification of sinus pericranii as dominant and accessory has been most important criterion for determining whether this anomaly can be safely obliterated (7, 33).

The sinus pericranii had been reported to be associated with vascular malformation, hemangioma of cerebellum, or retina as part of von Hippel Lindau syndrome, blue nevus syndrome, venous cavernoma of scalp, hemangioma of tongue and craniosynostosis (6, 18, 25, 35)

Although spontaneous regression of sinus pericranii had been reported (36, 37), most cases had been treated because of cosmetic reasons. There are reports, where sinus pericranii presented with hemorrhage (spontaneous, traumatic), infection, air embolism, intracranial hypertension and sinus thrombosis. In one of our cases, patient was misdiagnosed, leading to incision of the lesion with disastrous result. This led us to pursue aggressive intervention. (1, 7, 8, 38).

CONCLUSION

Sinus pericranii is a challenging diagnosis for clinician. This rare vascular anomaly which is seldom symptomatic and is often misdiagnosed. Most report suggests that Sinus pericranii is a clinically benign lesion that predominantly raises cosmetic concern. There are reports, where sinus pericranii presented with hemorrhage (spontaneous, traumatic), infection, air embolism, intracranial hypertension and sinus thrombosis. In one of our cases, patient was misdiagnosed, leading to incision of the lesion with disastrous result. This led some clinician to pursue aggressive intervention. Neuro imaging plays important role in the diagnosis of this uncommon condition. If a treatment is contemplated, analysis of drainage pattern should be done. Treatment should be avoided in case of dominant Sinus pericranii. We have presented our experience with five operated cases of sinus pericranii with excellent results.

REFERENCES

- [1] Bollar A, Allut AG, Prieto A, Gelabert M, Becerra E. Sinus pericranii: Radiological and etiopathological considerations. *J Neurosurg* 1992; 77: 469-472
- [2] Arrues MA, Dick man GH, Pataro VF .Sinus pericranii (Five cases). *Angiology*. 1956; 7:186-193
- [3] Hecker CF: Erfahrungen und Abhandlungen im Gebiete der chirurgieund ugenheil kunden. Erlangen, Enke, 1845
- [4] Stromeyer L: Ueber Sinus pericranii. *Dtsch Klin* 1850; 2:160-161.
- [5] Desai K, Bhyani R, Goel A, Muzumdar D. Sinus pericranii in the frontal region: a case report. *Neurol India* 2001; 49: 305-307.
- [6] Ohata T, Waga S, Handa H, Nishimura S, Mitani T. Sinus pericranii. *J Neurosurg* 1975; 42:704-712.
- [7] Pavanello M, Melloni I, Antichi E, Severino M, Ravegnani M ,Piatelli G, Cama A Gandolfo C: Sinus pericranii: diagnosis and management in 21 pediatric patients. *J Neurosurg Pediatr* 2015; 15:60–70.
- [8] Park SC, Kim SK, Cho BK, et al. Sinus pericranii in Children: report of 16 patients and preoperative evaluation of surgical risk. *J Neurosurg Ped*. 2009; 4:536-42.
- [9] Fe'vre M, Modéc L: Sinus pericranii et tumeurs vasculaires extracraniennes communniquant avec la circulation intracarnienne. *J Chirug* 1936; 47:561-588.
- [10] Gerlach J, Asperger H, Jensen HP, Kraus H. in *Pädiatrische Neurochirurgie 1967 – Thieme* (cited in reference 11)
- [11] Wakisaka S, Okuda S, Soejima T, Tsukamoto Y. Sinus pericranii. *Surg Neurol* 1983; 19:291-298.
- [12] Newton TH, Troost BT: Arteriovenous malformation and fistula in Newton TH and Potts DG (eds.): *Radiology of the Skull and Brain, vol II. Specific Disease process* St. Louis: 1974, pp 2490-2565.
- [13] Volkmann J: Ein Beitrag zum sogenannten sinus pericranii (stromeyer). *Zentralbi Chir*75:1389-94, 1950.
- [14] Mastin WM: Venous blood tumors of the cranium communicating with the intracranial venous circulation, especially the sinuses of dura mater. *JAMA* 7:309-320,1886.
- [15] Mastin WM: Venous blood tumors of the cranium in communications with intracranial venous circulation, esp. through the medium of superior longitudinal sinus. *Ann Surg* 1885; 1:324-340.
- [16] Muller E:Ueber Sinus pericranii. *Berlin med Wochenscher*.1914, 40:1372-76.
- [17] Muller E: Zur Frage des Sinus pericranii. *Z Ang Anat*. 1918; 3: 93-130.
- [18] NakayamaT, Matsukado Y: Sinus pericranii with aneurismal malformation of the internal cerebral vein. *Surg Neurol*.1975; 3:133—137.
- [19] Poppel MH, Roach JF, Hamlin H: Cavernous hemangioma of the frontal bone with a report of case of sinus pericranii. *AJR*.1948; 59:505-510.
- [20] Heinke W: Die chirgischen krankheitendes Kopfes. *Dtsch Chir*. 1948; 31:56-66.

- [21] Grossekkettler F: Sinus Pericranii. Roentgenpraxis.1930; 2: 368-373
- [22] Hahn EV: Sinus pericranii (Reducible blood of the cranium). Its origin and its relation to hemangioma and abnormal arteriovenous communications: report of a case. Arch Surg.1928; 16:31-43.
- [23] Nozaki J, Kawano H, Kabuato M et al: lateral sinus pericranii. Surg Neurol. 1986; 25:487-490.
- [24] Vaquero J, de Sola RG, Martinez R: Lateral sinus pericranii. Case report. J Neurosurg 1983; 58: 139-40, 1983.
- [25] Rizvi M, Behari S, Singh RK, Gupta D, Jaiswal AK, Jain M, et al. Sinus Pericranii with unusual features: Multiplicity, associated dural venous lakes and venous anomaly, and a lateral location. Acta Neurochir(Wein) 2010;152(12):2197-204.
- [26] Witrak Bj, Davis PC, Hoffman JC. Sinus pericranii. A case report. Pediatr Radiol1986; 16:55-56.
- [27] Little FM, Seagall HD, McComb JD. Sinus pericranii discovered at surgery for anticipated epidermoid cyst of the skull: A case report. J Child Neurol.1982;2(1):71-72.
- [28] Manaka S, Izawa M, Nawata H: Skull tumor simulating sinus pericranii. J Neurosurg 1977;46:671-673.
- [29] Wakisaka S, Okuda S, Soejima T, et al: Sinus pericranii. Surg Neurol1983; 19:291--298.
- [30] Sadler LR, Tarr RW, Jungreis CA, et al. Sinus pericranii: CT and MR findings. J Comput Assist Tomogr . 1990; 14: 124-127.
- [31] Carpenter JS, Rosen CL, Bailes JE, Gailloud P: Sinus pericranii: Clinical and imaging findings in two cases of spontaneous partial thrombosis. AJNR.2004; 25:121-125.
- [32] Jung S, Lee JK, Kim SH, Kang SS, Lee JH. Parietal Sinus pericranii: case report and technical note. Surg Neurol 2000; 54:270-273.
- [33] Gandolfo C, Krings T, Alvarez H, Ozanne A, Schaaf M, Baccin CE, et al. Sinus pericranii: Diagnostic and therapeutic considerations in 15 patients. Neuroradiology 2007;49:505–514.
- [34] Buxton N, Vloeberghs M: Sinus pericranii. Report of a case and review of literature. Pediatr Neurosurg 1999, 30:96-99.
- [35] Spektor S, Weinberger G, Constantani S, Gomori JM, Beni adani L. Giant lateral Sinus Pericranii: Case report. J Neurosurg 1998; 88:145-7.
- [36] Hayakawa I, Fujiwara K, Sasaki A, et al. Spontaneous regression of sinus pericranii, report of a case. Neurological Surgery 1978;6:91-5.
- [37] Rozen WM, Joseph S, Lo PA, et al. Spontaneous involution of two sinus pericranii-A unique case and review of literature . Journal of Clinical Neuroscience 2008;15: 833-835.
- [38] Akram H, Prezarakos G, Haliasos N, O'Donovon, Low H. Sinus pericranii: an overview and literature review of a rare cranial venous anomaly (A review of the existing literature with case examples). Neurosurg Rev 2012;35:15-26