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Complications in craniosynostosis surgery in patients with rickets: illustrative case and systematic review of literature

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BACKGROUND Craniosynostosis (CSS) is the premature fusion of calvarial sutures associated with identified genetic mutations or secondary to alterations in intracranial pressure, brain, or bone growth patterns. Of the metabolic etiologies implicated in CSS, X-linked hypophosphatemic rickets (XLHR) is the most common, with dysfunctional bone mineralization leading to progressive hyperostosis and delayed synostosis. There is a paucity of literature discussing the unique surgical considerations for XLHR-related CSS.

OBSERVATIONS A 26-month-old male with XLHR-related sagittal CSS underwent cranial vault remodeling (CVR). Surgery was complicated by the presence of diploic hypertrophy with significant intraoperative estimated blood loss (EBL). EBL greatly exceeded reference ranges for CVR in all-cause CSS. As a result, the surgical goals were modified and the complete planned procedure aborted. Subsequent review of preoperative imaging revealed multiple fine vascular lacunae within the bone. A systematic literature review was conducted to identify reported complications relating to surgical intervention for rickets-associated CSS.

LESSONS Future considerations for patients with XLHR-related CSS should emphasize awareness of metabolic risk factors with associated complications, and the need for selection of approach and operative management techniques to avoid EBL. Further research is required to elucidate underlying mechanisms and determine whether the encountered phenomenon is characteristic across this patient population and potentially minimized by preoperative medical therapy.

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KEYWORDS rickets; craniosynostosis; cranial vault remodeling; endoscopic suturectomy

Craniosynostosis (CSS), the premature fusion of 1 or more calvarial sutures, occurs in association with known genetic mutations, syndromes, and metabolic disorders.[1] The diagnosis is most often clinical, based on compensatory dysmorphology on physical examination, but may also require radiographic confirmation in specific cases.[2] Treatment options include open cranial vault remodeling (CVR) for older children and endoscopic suturectomy for those diagnosed prior to 4 months of age.[3,4]

Rickets, mucopolysaccharidosis, pseudohypoparathyroidism, hypophosphatasia, and osteopetrosis are examples of metabolic disorders that have been implicated in the development of CSS.[5] Among these, rickets is most frequently associated with CSS and is characterized by a failure of bone mineralization linked to hypocalcemia and hypophosphatemia, resulting in long-bone growth disproportion and progressive hyperostosis of the calvaria leading to delayed synostosis.[2] With the increased availability of genetic testing, there has been a rise in the diagnostic prevalence of vitamin D deficiency disorders such as X-linked hypophosphatemic rickets (XLHR) and 1α-hydroxylase deficiency (vitamin D–dependent rickets), which are associated with dysfunctional osteophytes and deficient vitamin D

ABBREVIATIONS CSS = craniosynostosis; CT = computed tomography; CVR = cranial vault remodeling; EBL = estimated blood loss; ICU = intensive care unit; XLHR = X-linked hypophosphatemic rickets; 3D = three-dimensional.

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enzymatic metabolism, respectively. XLHR is the most common form, with an incidence of 1 in 20,000 individuals.[6]

Although small case series have reported an association between XLHR and CSS, there remains limited data regarding complications associated with surgical treatment of this patient population. As the development of CSS occurs in a delayed fashion in XLHR, surgical treatment is most commonly CVR as opposed to endoscopic suturectomy due to the typical age at diagnosis. Here we report on hypophosphatemic rickets and associated intraoperative blood loss in a case of sagittal CSS treated with open CVR. To our knowledge, this is the most detailed report on blood loss and associated genetic and radiographic findings in craniofacial surgery in a patient with CSS secondary to rickets. A review of the literature on surgical complications in this population was also performed to offer additional insight into the etiologies, risk factors, and patient characteristics surrounding this clinical entity, as well as steps for future management.

Methods

A retrospective chart review identified a case of sagittal CSS secondary to hypophosphatemic rickets treated at our institution using CVR. Relevant demographics, clinical history, imaging, and operative notes were reviewed. A systematic literature review was conducted to identify reported complications relating to surgical intervention for rickets-associated CSS. Using the string "rickets craniosynostosis," a search of the PubMed database was performed from database inception to May 2022. Articles were included for further analysis if they (1) reported on at least 1 human case of rickets-associated CSS and (2) provided information on the operative or postoperative course. Articles were excluded if they were (1) not original research, (2) published in a non-English

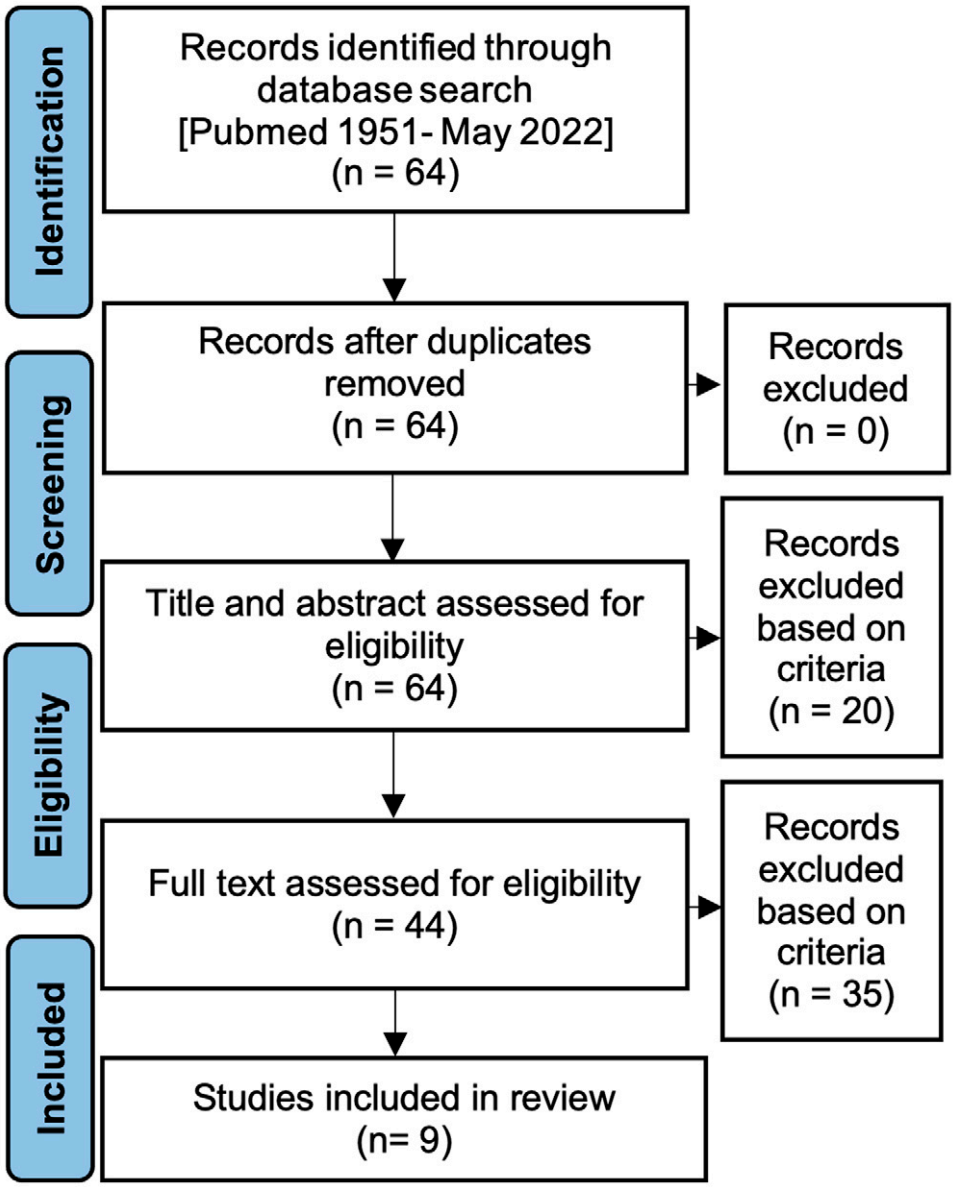

FIG. 1. PRISMA flow diagram. PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

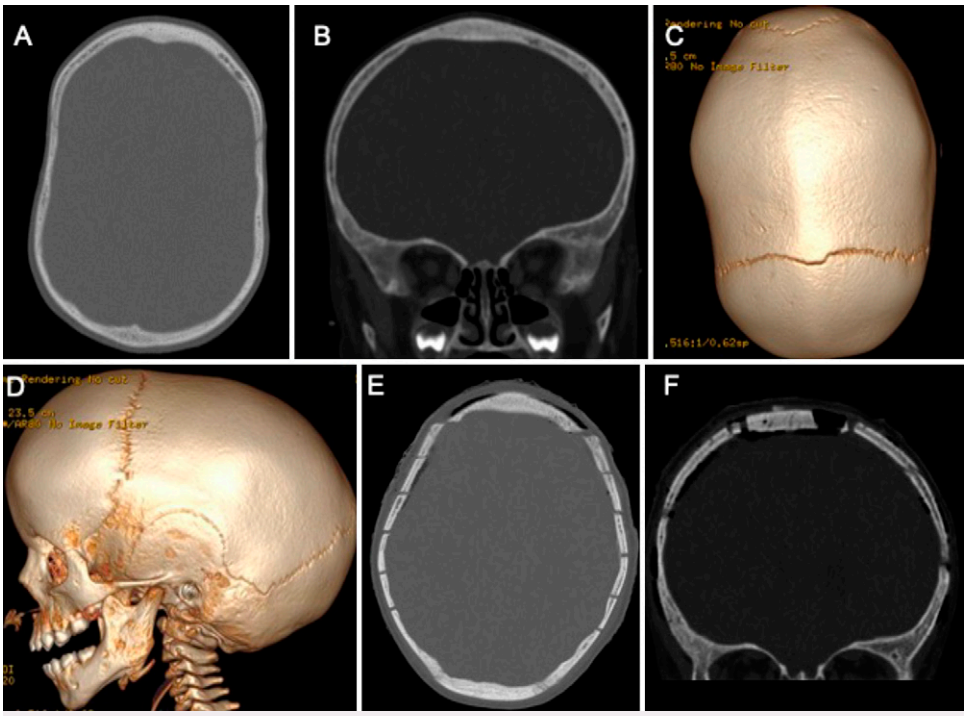

FIG. 2. Preoperative noncontrast CT axial (**A**) and coronal (**B**) cuts and axial (**C**) and sagittal (**D**) 3D reconstructions demonstrating premature closure of the sagittal suture with associated scaphocephaly. Postoperative noncontrast CT with axial (**E**) and coronal (**F**) cuts demonstrating cranial remodeling with correction of biparietal restriction and vertex elevation.

language, or (3) only mentioned the surgical approach taken without any further discussion.

Search results were compiled with duplicate studies removed. Title and abstract screening was conducted by the 2 first authors (M.N.L. and K.Z.) who then independently reviewed full-length articles. Conflicting inclusion decisions at any point throughout screening were discussed until the authors reached a consensus (Fig. 1). Variables extracted included author, year of publication, number of patients, average age, rickets etiology, suture corrected, surgical approach, blood loss, intra- and postoperative management strategies, as well as transfusion threshold and volume transfused when available.

Illustrative Case

Clinical Presentation

The patient was born at 37 weeks via vaginal delivery with a short neonatal intensive care unit (ICU) stay for respiratory distress with no sequelae. No family history of rickets, calcium, or phosphorus disorders; metabolic bone disease; short stature; or endocrinopathy was noted. At 1 week of age, the patient's head circumference was $>$ 99th percentile (38.8 cm) and body length was 95th percentile (54.2 cm).

At 8 weeks and 6 days of age, the patient was referred to the craniofacial clinic for evaluation of macrocephaly and frontal bossing. Assessment was notable for mild frontal bossing and torticollis. The patient was neurologically intact and meeting all developmental milestones, with a head circumference in the $>$ 99th percentile (42.5 cm) and body length in the $>$ 99th percentile (63.3 cm). No vertex slanting, biparietal restriction, sutural ridging, or occipital compensation were appreciated on examination. Head ultrasound showed bilateral symmetric extra-axial fluid in the frontoparietal regions, consistent with benign extraaxial subarachnoid spaces of infancy. The parents were advised to present back to the clinic if concerns regarding head shape or head circumference persisted in follow-up with their pediatrician.

At 23 months, the patient presented back to the craniofacial clinic due to persistent macrocephaly. Of note, he was lost to follow-up during the interval between his second-month visit and 23rd-month visit due to the coronavirus disease 2019 (COVID-19) pandemic and reliance on virtual pediatrician visits. At this time, his head circumference was 55 cm. He was now noted to have developed bow-leggedness with frequent falls but was otherwise reaching normal milestones. Physical examination demonstrated the development of scaphocephaly with progressive frontal bossing, distention of scalp veins, bicoronal saddling, vertex slanting, and occipital compensation. Noncontrast head computed tomography (CT) demonstrated closure of the sagittal suture compatible with sagittal synostosis (Fig. 2). Surgical correction with CVR was recommended. A CT venogram was not obtained preoperatively, as venous imaging for patients with nonsyndromic synostosis is not part of our standard of care.

The patient was referred to endocrine and his pediatrician for assessment for rickets based on these clinical findings and potential medical optimization prior to surgery. Systemic examination revealed several additional clinical findings, including multiple dental abscesses, confirmed bow-leggedness, and abnormal laboratory values consistent with hypophosphatemic rickets. Endocrine abnormalities included mild elevation in alkaline phosphatase, low-for-age serum phosphorous,

and normal 25-hydroxyvitamin D. Calcitriol and phosphorus therapy was initiated, with a plan to start treatment with burosumab postoperatively. Genetic testing for XLHR was performed. He also underwent treatment for his dental abscesses prior to proceeding with calvarial vault remodeling and continued on appropriate antibiotic therapy during the perioperative period.

Surgical Technique and Intraoperative Findings

Virtual surgical software was used to plan the surgical approach, including both posterior and anterior remodeling in order to restore normal calvarial volume and morphology (Fig. 3). Figure 3 shows a screenshot of the virtual surgical plan with the projected volumetric increase and postoperative morphology. The possibility of pursuing only posterior reconstruction was discussed with the family preoperatively as the posterior reconstruction accomplished the functional surgical goal of increasing intracranial volume while anterior reconstruction targeted a cosmetic goal with respect to forehead remodeling and correction of bossing. After prone positioning, a bicoronal incision was created with bony exposure to the superior orbital rim anteriorly and to the lambdoid sutures posteriorly. Burr hole osteotomies were created on either side of the midline at the anterior and posterior extent of the sagittal suture. With the first burr hole, excessive diploic hypertrophy and associated bleeding were encountered, with substantial blood loss elicited with any bone cut or manipulation of the epidural space. Surgical technique was therefore shifted to the creation of troughs to allow for controlled exposure of the epidural space and bony hemostasis during the creation of the necessary craniectomy. Hemodynamic stability was maintained using hemostatic agents and substantial fluid, blood products, and factor resuscitation while in constant communication with anesthesia. The remainder of the lateral barrel stave osteotomies and calvarial reconstruction were then completed. The anterior reconstruction was aborted once the sagittal suture was removed and intracranial volume had been increased with the functional goal accomplished, as anterior reconstruction at this point was felt to represent a cosmetic goal. Estimated blood loss (EBL) was 2150 mL, with an estimated preoperative circulating blood volume of 1035 mL. The patient was transfused packed red blood cells, platelets, plasma, and cryoprecipitate to maintain intravascular volume and to prevent coagulopathy as well as insulin and calcium phosphate to prevent cardiac instability secondary to hyperkalemia. The patient was hemodynamically stable throughout the procedure and remained intubated after surgery due to the large volume of transfused products. The patient was extubated and weaned to room air without issue on postoperative day 1, was neurologically intact on examination, and was discharged home on postoperative day 3. Postoperative noncontrast head CT showed expected postsurgical changes with increased intracranial volume and subarachnoid space (Fig. 2). The results of genetic testing 7 days postoperatively revealed the etiology to be XLHR (pathogenic variant, c.871 C $>$ T [p.291*] in the phosphate-regulating endopeptidase variant). A retrospective review of preoperative imaging with neuroradiology demonstrated multiple fine hypervascular-appearing lacunae in the middle table, within the diploic space of the skull, when compared with a patient with single sagittal synostosis without rickets (Fig. 4).

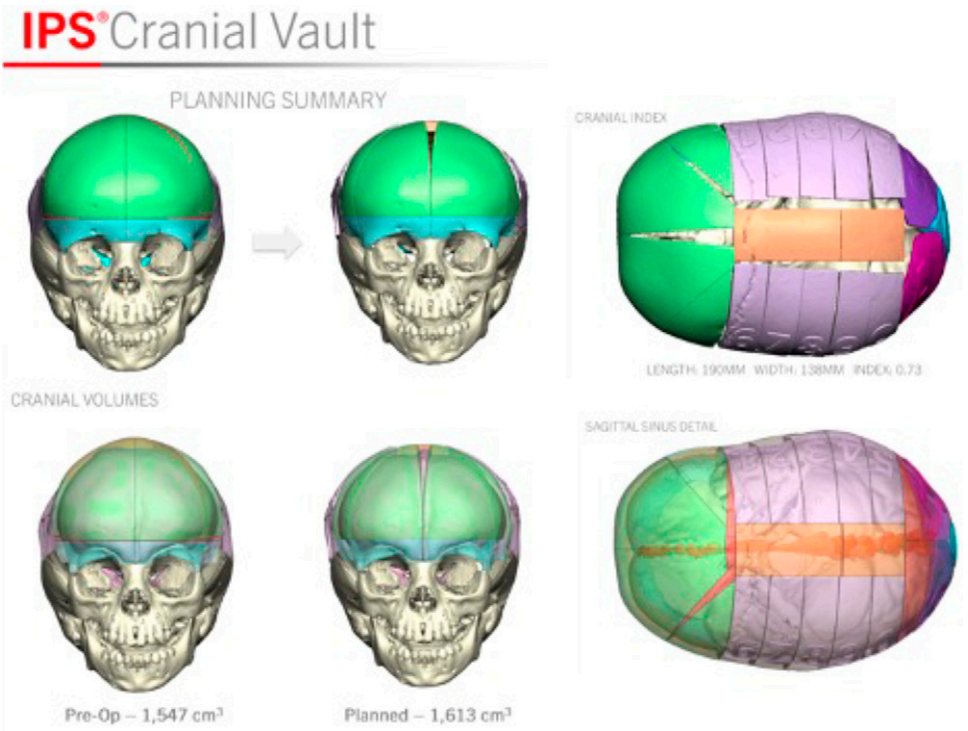

FIG. 3. Virtual surgical planning summary renderings compiled from the manufacturer's solution proposal show the patient's preoperative and planned cranial volumes, cranial index, and detail of the sagittal sinus. Pre-Op = preoperative.

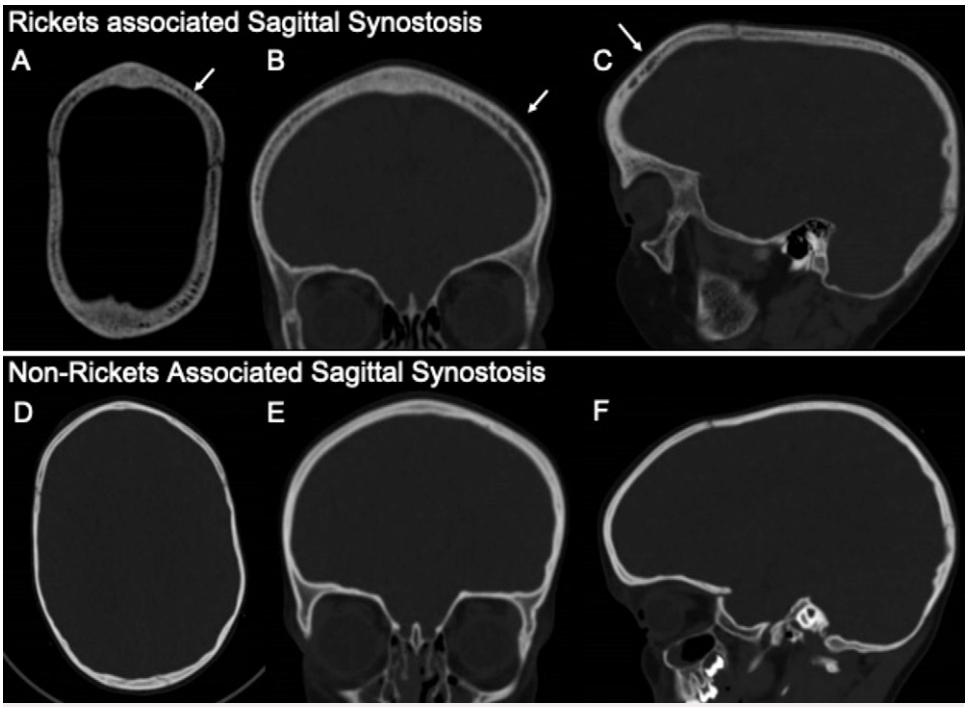

FIG. 4. Comparative CT in patients with sagittal synostosis demonstrating rickets-associated sagittal synostosis (**A–C**) versus non–rickets-associated sagittal synostosis (**D–F**). Axial (A), coronal (B), and sagittal (C) noncontrast preoperative CT scan of rickets-associated sagittal synostosis demonstrates fine lacunae in the middle table of the skull (*white arrows*) when compared with a similarly aged patient with sagittal synostosis without rickets on axial (D), coronal (E), and sagittal (F) cuts.

Literature Review

A systematic review of the literature using the string "rickets craniosynostosis" on PubMed yielded 64 records. Screening excluded 55 articles primarily due to the absence of data or discussion surrounding surgical outcomes. Nine studies reporting on 12 cases were included for data extraction and analysis (Table 1).[6–14] Initial compilation showed consistency with other literature discussing rickets-associated CSS, with a notable male preponderance (67%), previously postulated to reflect a more severe phenotype in males with XLHR, and an average age of 22.1 months (at diagnosis), which aligns with other studies reporting that diagnosis and management occur around 2 years of age.[2,5] Primary involvement of the sagittal suture in 7 of 12 cases (58%), followed by coronal involvement 3 of 12 cases (25%) and both coronal and sagittal involvement in 2 of 12 cases (17%) were noted. This distribution is also expected, with previous studies reporting a predominance of sagittal closure, followed by coronal synostosis.[3] Initial presenting characteristics of abnormal head shape and papilledema (our patient did not have papilledema reported) are also concurrent with other reports.[4] CVR was the only described surgical approach in all of the reviewed cases given the age at presentation.[3] Rickets etiology was also consistent with established trends, with 7 of 12 (58%) of the described patients having an XLHR form and 2 (17%) additional patients having a hypophosphatemic form of unknown etiology.[1]

D'Agostino et al.[7] described an EBL of 1000 mL in both of their patients, including excellent follow-up imaging affirming the value of surgical correction. While 1 reviewed case had a vitamin D–dependent etiology of rickets, the investigation described hyperemic bone but did not characterize the underlying bony morphology or radiographic characteristics, although they did hypothesize that the underlying metabolic demands of the bone may result in increased blood flow and ultimately hyperemic rickettic bone. Our report contributes to this suspicion, as the bone was noted to have essentially excess diploic space with hypervascularized lacunae leading to high EBL. While not encountering any complications themselves, Freudlsperger et al.[6] remarked that the rickettic skull may be more difficult to remodel surgically and that the possibility for bone defects is increased in patients over 1 year of age due to the attenuated capacity for reossification, potentially requiring more extensive craniectomies for cosmetic and pathological correction. However, surgical data in this review suggest that complication rates of CVR in these patients are infrequent, with 11 of 12 cases (91.7%) noting no complications. Unique surgical findings in this disease population include the report by Jaszczuk et al.[11] of soft bone and obliteration of the subdural space with endocortical scalloping of the inner table and Glass et al.[9] observing similarly diminished subarachnoid space with cortical interdigitation into the inner table.[12]

Discussion

Observations

This case illustrates the intraoperative complications encountered in a 26-month-old male who underwent CVR for correction of sagittal synostosis secondary to XLHR. The clinical presentation was consistent with established trends for sex distribution, rickets etiology, presenting characteristics, sutural involvement, and elected surgical approach for other patients with CSS secondary to rickets.[2]

TABLE 1. Literature review of craniosynostosis cases for children with rickets that report intraoperative outcomes

Authors & Year	No. of Pts, Sex	Age at Op	Rickets Etiology	Suture Corrected	Surgery Type (CVR vs Endo)	Complications	Blood Loss	Intraop Findings	Reason for Referral
McCarthy & Reid, 1980[13]	1, F	17 mos	Hypophosphatemic	Coronal, sagittal	CVR	"Uneventful"	NA	NA	Abnormal head shape
Inman et al., 2008[10]	1, M	2 yrs	Unknown	Coronal	CVR	"Uneventful"	NA	NA	Abnormal head shape
Murthy, 2009[14]	1, M	32 mos	XLHR	Sagittal	CVR	"None"	NA	NA	Maternal hx & papilledema
Garg et al., 2010[8]	1, M	11 mos	Hypophosphatemic	Coronal	CVR	"Uneventful"	90 mL	NA	Abnormal head shape & proptosis
Glass et al., 2011[9]	1, F	3 yrs	XLHR	Coronal, sagittal	CVR	"Uneventful	NA	Diminished CSF & skull brain interdigitation	Papilledema
FreudIsperger et al., 2013[6]	1, M	18 mos	XLHR	Sagittal	CVR	"Uneventful"	NA		Abnormal head shape
Jaszczuk et al., 2016[11]	2, F; 1, M	2, 2, 3 yrs	XLHR	Sagittal	CVR	"Uneventful, without incident"	NA	Soft bone & subcortical space obliteration with scalloping	Papilledema & abnormal head shape
Johal et al., 2017[12]	1, M	7 mos	NA	Coronal	CVR	"Without consequences"	NA	Accessory (frontal wormian) bone attached to dura	Abnormal head shape
D'Agostino et al., 2019[7]	2, M	3 yrs	XLHR, VDDR	Sagittal	CVR	"High blood loss"	1000 mL	NA	Abnormal head shape

CSF = cerebrospinal fluid; CVR = cranial vault remodeling; Endo = endoscopic suturectomy; hx = history of; NA = not applicable; pts = patients; VDDR = vitamin D–dependent rickets; XLHR = X-linked hypophosphatemic rickets.

However, the distinct finding in our patient was the presence of increased, fine trabeculations throughout the diploic space leading to multiple vascular lakes. These lacunae produced extensive bleeding with any manipulation of bone. Additionally, epidural dissection was met with substantial venous bleeding, indicating potential venous hypertension or abnormal emissary formation. These factors led to an abnormally high intraoperative EBL of 2150 mL (155.8 mL/kg). This greatly exceeded the expected blood loss for CVR from our own previously published series, with a mean EBL of 207.4 mL (range: 20–625 mL), and the range of 200 to 400 mL in the reported literature.[15,16]

Venous Lacunae

Venous malformations creating abnormal vascular channels throughout the calvaria complicated the attempt at a total calvarial vault reconstruction. These vascular aberrancies did not seem to communicate through the meningeal layers or extend to the galea, suggesting this is a distinct entity from sinus pericrania, which is secondary to persistent communications between the sinuses and scalp, or persistent emissaries seen in other hyperostosis syndromes.[17,18] There are reports of idiopathic vascular lacunae lateral to the sagittal suture that are especially pronounced in children as venous channels are generally relatively larger at birth and recede during embryogenesis.[19] However, this phenomenon is likely not implicated in our patient, as lateral channels reside deep to the bone and are fed by the meningeal veins connecting to the superior sagittal sinus.[20]

While not previously associated with rickets or other metabolic disorders, our case does resemble the excessive bleeding encountered by D'Agostino et al.;[7] their case noted "hyperemic" or hypervascular bone, similar to the vascular lacunae seen in our patient. Jaszczuk et al.[11] may also have recognized some coincidental manifestation of the process, with increased blood flow having some relation to the softer-than-usual bone they noted.

Future Management and Considerations

The findings in this case hold important considerations for preoperative planning and intraoperative prevention of excessive bleeding in rickets-associated CSS. The most conservative operative plan to achieve the surgical goals may need to be considered. Preparation for high intraoperative blood loss with the anesthesia team is critical; parents should be consented for blood transfusion, the patient should be cross-matched with blood available in the operating room at the start of the case, and the patient should have appropriate intravenous access established prior to incision. The postoperative fluid status of the patient is also important to consider, as our patient remained intubated due to the high volume of resuscitative products; both parents and pediatric ICU should be aware of complications related to high-volume transfusion such as pulmonary edema and prolonged intubation, coagulopathy, cardiomyopathy, and anaphylaxis.

The demographic and geographic epidemiology of CSS secondary to rickets has large implications on global awareness, public health measures, and interventional screening programs.[21] Patients with delayed presentation of CSS in geographic regions where rickets is common, such as the Middle East, Africa, and Asia, should potentially be screened for nutritional deficiencies and genetic abnormalities associated with rickets.[22] Additionally, intraoperative complications of craniofacial surgery secondary to rickets should have a greater awareness within the global neurosurgery community. Early diagnosis and treatment of XLHR and craniosynostosis are associated with better outcomes, both cosmetically and functionally.[23,24] Initiation of treatment of XLHR before 1 year of age increases the likelihood that patients will achieve normal growth, especially since synostosis in rickets tends not to develop until after 1 year of life, therefore making a total vault reconstruction the most viable treatment strategy for these children.

While the mechanism is unclear, the presentation of venous lacunae may be predictable based on the detection of diploic hypertrophy.[25] When there is concern for hypophosphatemic rickets, or another metabolic disorder that may confer aberrant venous anatomy, preoperative evaluation of the patient with helical three-dimensional (3D) head CT may be a useful preoperative adjunct.

Lessons

In conclusion, XLHR can result in delayed secondary synostosis, and awareness of potential surgical complications and methods to mitigate these risks is important. The described case reiterates risk factors and presenting characteristics similar to the trends reviewed in the literature; however, significant blood loss was reported in only 1 case. We recommend screening for rickets in patients with delayed development of secondary CSS in regions where rickets is prevalent. In addition, preoperative planning for patients with rickets undergoing craniofacial surgery would optimally consist of preoperative helical 3D head CT for detection of diploic hypertrophy, modified operative approach and surgical goals, preoperative planning for excessive bleeding, early consent for blood transfusion, and postoperative monitoring for complications related to high-volume transfusion. Further research is needed to determine whether the presence of venous lacunae is a consistent feature of a rickets-associated process and should be considered when performing craniofacial surgeries in this population, or a confluence of idiopathic factors in proximity to the superior sagittal sinus.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Hoffman, LaValley, Zappi, Guadix, Imahiyerobo. Acquisition of data: LaValley, Zappi, Giantini-Larsen, Garton, Heier, Imahiyerobo. Analysis and interpretation of data: Hoffman, LaValley, Zappi, Giantini-Larsen, Heier, Imahiyerobo. Drafting of the manuscript: LaValley, Zappi, Guadix, Giantini-Larsen, Garton, Imahiyerobo. Critically revising the manuscript: all authors. Reviewed submitted version of the manuscript: Hoffman, Zappi, Guadix, Garton, Heier, Imahiyerobo. Statistical analysis: Zappi. Study supervision: Hoffman, Imahiyerobo.

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