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Review Article

Association between spontaneous intracranial epidural hematoma and craniofacial infections: A systematic literature review

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ABSTRACT

Background: Spontaneous and nontraumatic epidural hematoma (SEDH) is a rare entity. Etiology is various, including vascular malformations of the dura mater, hemorrhagic tumors, and coagulation defects. The association between SEDH and craniofacial infections is rather unusual.

Methods: We performed a systematic review of the available literature using the PubMed, Cochrane Library, and Scopus research databases. Literature research was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement. We exclusively included studies reporting demographic and clinical data, published until October 31, 2022. We also report one case from our experience.

Results: A total of 18 scientific publications, corresponding to 19 patients, met the inclusion criteria for the qualitative and quantitative analysis. Patients were mostly adolescents, with a clear male predominance. SEDHs frequently occurred in the frontal area, usually near the site of the infection. Surgical evacuation was the treatment of choice with good postoperative outcomes. Endoscopy of the involved paranasal sinus should be achieved as soon as possible to remove the cause of the SEDH.

Conclusion: SEDH may occur as a rare and life-threatening complication of craniofacial infections; therefore, prompt recognition and treatment are mandatory.

Keywords: Epidural, Hematoma, Infection, Intracranial, Spontaneous, Surgery and antibiotics

INTRODUCTION

An epidural hematoma (EDH) is an extra-axial collection of blood lying between the outer layer of the dura mater and the inner table of the skull. It is generally associated with head trauma, occurring in about 2% of all head injuries with mortality rates ranging from 1.2% to 33%.[22] Most of the cases originate from arterial bleeding, usually from a branch of the middle meningeal artery (MMA) (most common source); up to 10% of EDHs are related to venous bleeding. EDHs usually occur in young adults and males are more frequently affected than females.[9] On computed tomography (CT) scans, they classically appear as high-density biconvex-shaped masses.[16] In most cases, EDHs require prompt surgical evacuation. According to international guidelines, an EDH >30 cm^3 should be surgically evacuated regardless of the patient's Glasgow Coma Scale

(GCS) score, while EDHs <30 cm^3, with <15-mm thickness and with <5-mm midline shift, in patients with a GCS score >8 without focal deficits can be managed nonoperatively in a neurosurgical center.[2]

Spontaneous EDH (SEDH) is a rare entity. Even though it has been described as "spontaneous," an underlying cause has been recognized in all the reported cases. Etiology includes vascular malformations of the dura mater, hemorrhagic tumors, coagulation defects, and but also craniofacial infections.[7] In a few cases, SEDH has been reported in association with kidney diseases, systemic lupus erythematosus, and open-heart surgery.[12]

To date, only 19 cases of SEDH associated with craniofacial infections have been reported in the literature.[1,3-6,8,10,11,13-15,17,18,20,21,23-25]

We discuss the relevant literature and report one case from our experience.

MATERIALS AND METHODS

Study design

The present investigation consists of a case report and a systematic review of the literature conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.

Eligibility criteria

All written papers about the association between spontaneous intracranial EDH and craniofacial infections, reporting demographic and clinical data, diagnostic workflow, treatment, and prognosis were considered for eligibility. Articles lacking precise information were excluded, together with surgical and radiological technical notes, anatomical studies, abstracts from scientific meetings, and unpublished reports; articles discussing nonspontaneous EDHs were considered "not eligible" as well.

Publications written in languages other than English were considered in our paper only if the relevant information could be inferred from a complete abstract written in English.

Information sources and search strategy

The systematic review of the literature was performed on three different online medical databases (PubMed, Cochrane Library, and Scopus), using as search-terms "spontaneous," "epidural," "extradural," "hematoma," and "sinusitis" (being otitis and mastoiditis considered as falling under this definition) (All Fields), combined with the Boolean operators "OR" and "AND." The last search was conducted on October 31, 2022, and went back as far as data were available. The search strategy is summarized in Figure 1.

Data collection process and study selection

Abstracts and full texts were independently screened by two authors (Anthony Kevin Scafa and Massimo Corsini), and any discordance was solved by consensus with a third, senior author (Manolo Piccirilli). Reference lists of the included full texts ("forward search") were similarly screened and evaluated for inclusion.

RESULTS

After duplication removal, 54 papers were screened for this systematic review. Thirty-nine papers were excluded because "not pertinent" (exclusion criterion), and 15 full-text papers were, therefore, evaluated. Among these, seven papers were excluded according to our eligibility criteria so that eight papers, plus 10 from forward search, were finally included and analyzed[1,3-6,8,10,11,13-15,17,18,20,21,23,25] [Figure 1].

The mean age of the investigated patients (n = 20, 19 from the literature review and 1 from our experience) was 19.3 (range 10–34 years). The M: F ratio was 2.3:1.

Setting of the hemorrhage was supratentorial in all cases; location was frontal (alone or in combination with adjacent sites) in 13 patients (65%).

The "cause" of the hemorrhage was an infection of the frontal sinus (alone or in combination with infection of the adjacent sinuses) in 12 cases (60%).

Clinical profiles were diverse and are reported in Table 1. An altered level of consciousness was found in 15 patients (75%), being the most common presenting neurological "symptom." In none of the analyzed cases were systemic signs of infection apparent.

Coagulation tests were normal in all the cases for which this information was available, apart from our case.

SEDH was diagnosed postmortem in three cases (15%); surgery was the treatment of choice in the remaining 17 patients (85%), with good postoperative outcomes in all cases.

Eight patients (40%) had positive intraoperative cultures (Gram-positive cocci in six cases, 75%). Results were negative in 5 (25%), not available in 7 (35%) [Table 1].

Information regarding antibiotic therapy was available in only seven cases [Table 1]: β-lactams and metronidazole were the most frequently employed drugs.

CASE REPORT

A 19-year-old male was admitted to the emergency department of a peripheral hospital complaining of headache, agitation, vomiting, diplopia, and fever. The patient suffered from left conjunctivitis, treated with antibiotic eyewashes, 3 weeks earlier, and had a history of headache in the past

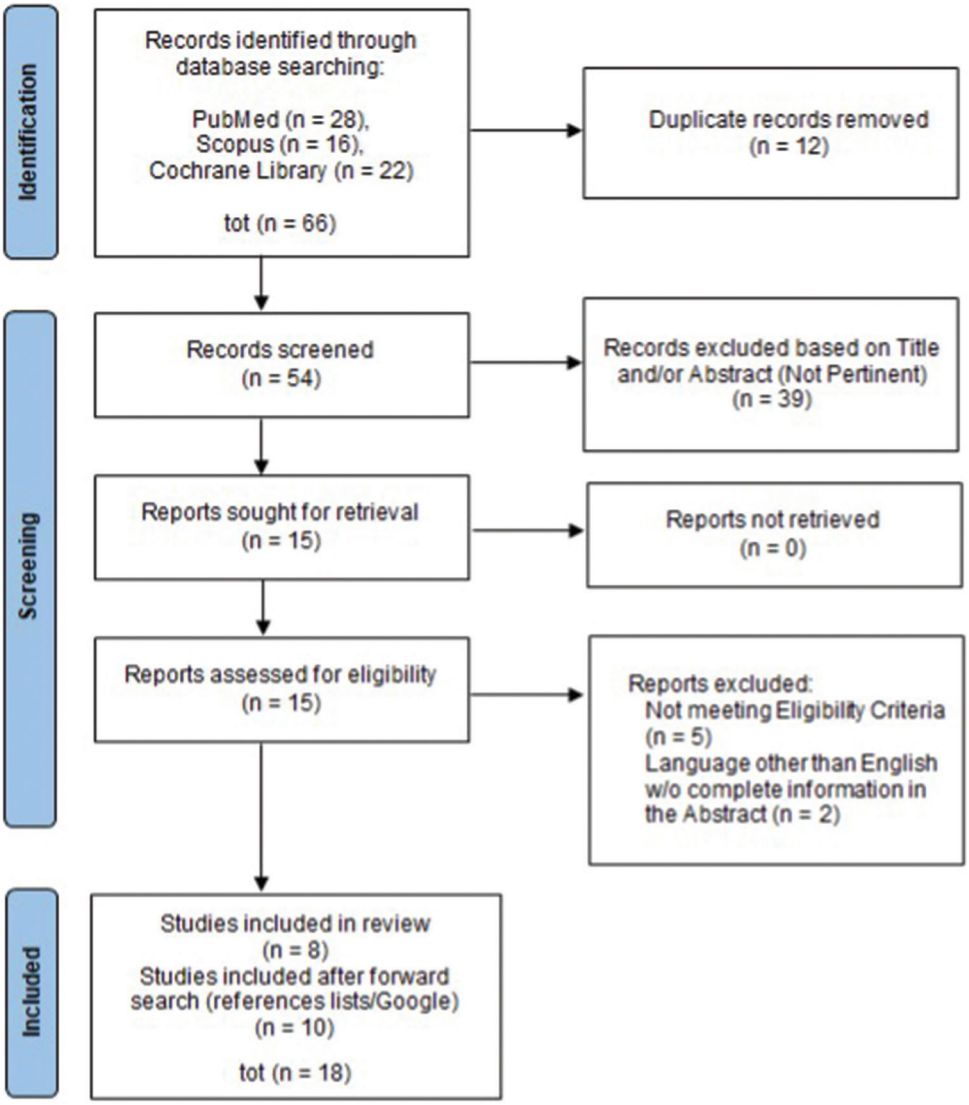

Figure 1: PRISMA flow-chart showing information sources, search strategy, and study selection. PRISMA: Preferred reporting items for systematic reviews and meta-analyses.

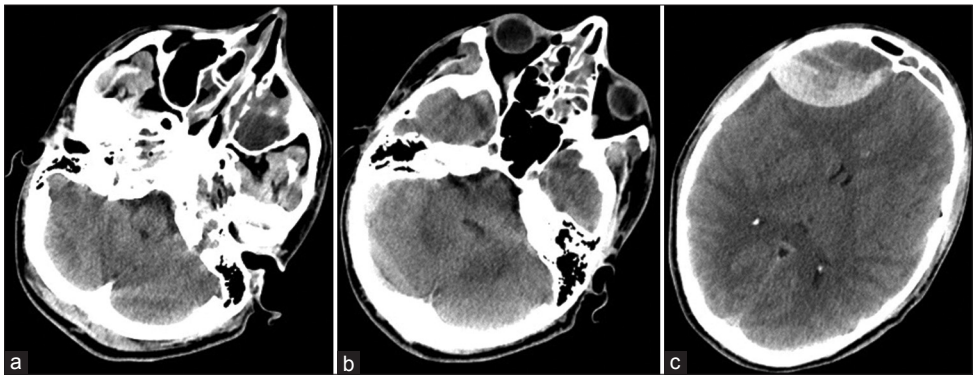

Figure 2: Preoperative brain computed tomography scan showing a voluminous right frontal extradural hematoma, along with inflammatory hypodense tissue in the left ethmoidal cells (b), maxillary (a), and frontal (c) sinuses. No evidence of skull fractures.

Table 1: Review table.

Authors and year	Age and sex	SEDH site	Associated CF infection/s	Clinical presentation	Coagulation tests	Surgery	SEDH culture	Antibiotic therapy	Outcome
Schneider and Hegarty, 1951[20]	21, M	R temporal	R (chronic) otitis media + mastoiditis	Draining R ear, lethargy, anisocoria (R>L), L UE weakness, bilateral Babinski	NA	N/ postmortem diagnosis	NA	NA	Dead
	21, M	R fronto-temporal	R ala nasi furuncle + frontal sinusitis	Fever, somnolence, mental confusion, R fronto-temporal headache, anisocoria (R>L), L faciobrachial weakness, meningismus	NA	Y	Hemolytic *S. aureus*	NA	Alive at 10 mo-FUP (seizures)
Novaes and Gorbitz, 1965[13]	26, M	L temporal	L otitis media	Lethargy, anisocoria (L>R), R hemiparesis	NA	Y	NA	NA	Good
Kelly and Smith, 1968[8]	11, M	L frontal	L frontal sinusitis	Fever, headache, L eye pain, purulent discharge from both nostrils	NA	Y	*H. influenzae*	IM penicillin 400,000 units every 6 h, postoperatively shifted to oral penicillin and chloromycetin (no mention of duration)	Good at 9 mo-FUP
Clein, 1970[5]	18, M	L fronto-parietal	L otitis media + mastoiditis	Severe L sided headache and vomiting; coma following spinal tap	NA	N/ postmortem diagnosis	Beta hemolytic Streptococcus	NA	Dead
Rajput and Rozdilsky, 1971[15]	18, M	L frontal	L frontal + maxillary sinusitis	Fever, epilepsy, coma	Normal	N/ postmortem diagnosis	*S. aureus*	IV ampicillin 500 mg every 6 h, IV sodium sulfadiazine 2 g every 8 h, im diphenylhydantoin sodium 100 g every 8 h, im streptomycin sulfate 1 g every 12 h	Dead
Sanchis *et al.*, 1975[18]	13, M	R temporal	R otitis media	Draining R ear, lethargy, anisocoria (R>L), bilateral Babinski	NA	Y	NA	NA	Good
Marks and Shaw, 1982[10]	31, M	L frontal	Pansinusitis	Somnolence, frontal headache, vomiting, L periorbital swelling	Normal	Y	Negative	NA	Good
Sekimoto *et al.*, 1985[21]	34, F	R temporal	(Pan)sinusitis+ orbital phlegmon	Headache, R periorbital swelling, lethargy	NA	Y	NA	NA	Good

(Contd...)

Table 1: *(Continued).*

Authors and year	Age and sex	SEDH site	Associated CF infection/s	Clinical presentation	Coagulation tests	Surgery	SEDH culture	Antibiotic therapy	Outcome
Ataya, 1986[1]	31, M	L frontal	(Chronic) pansinusitis	Somnolence, headache, L supraorbital swelling, diplopia	Normal	Y	NA	NA	Good
Sakamoto *et al.*, 1997[17]	16, F	L frontal	L maxillary sinusitis	fever, headache, L periorbital swelling and exophthalmos	NA	Y	*P. cepacia* (in extradural drain fluid)	NA	Good
Papadopoulos *et al.*, 2001[14]	17, M	R frontal	Frontal sinusitis	Fever, frontal headache, nausea, vomiting	Normal	Y	Negative	2 w-course of IV cefotaxime and metronidazole (no mention of dosage regimen)	Good at 3 mo-FUP
Griffiths *et al.*, 2002[6]	17, M	Frontal	Fronto-sphenoidal sinusitis	Fever, R nostril purulent discharge, lethargy	Normal	Y	*S. milleri*	5 w-course of IV amoxicillin (no mention of dosage regimen)	Good
Moonis *et al.*, 2002[11]	21, M	L temporal	Sphenoidal sinusitis	Fever, headache, nausea, vomiting, L V1/V2 loss of sensation	NA	Y	*S. aureus*	NA	Good at 2 mo-FUP (mild L V1/V2 decreased sensation)
Chaiyasate *et al.*, 2007[3]	14, F	R frontal	Pansinusitis	Fever, headache, vomiting, R periorbital swelling	Normal	Y	*S. anginosus*	6 w-course of IV ceftriaxone and metronidazole (no mention of dosage regimen)	Good
Takahashi *et al.*, 2010[24]	10, F	R temporal	Sphenoidal sinusitis	fever, lethargy, R CIII palsy, L hemiparesis	Normal	Y	NA	NA	Good
Cho *et al.*, 2011[4]	17, F	R temporal	Sphenoidal sinusitis	Fever, lethargy, R CIII palsy, anisocoria (R>L)	NA	Y	Negative	3-w course of IV ceftriaxone and metronidazole	Good at 1 mo-FUP
Spennato *et al.*, 2012[23]	12, F	R frontal	R (chronic) frontal sinusitis	Lethargy, R exophthalmos, L hemiparesis	Normal	Y	NA	NA	Good at 3 mo-FUP
Xiao, 2021[25]	18, M	L frontal	L fronto-ethmoido-maxillary sinusitis	Fever, headache, nausea, vomiting, confusion, lethargy	Normal	Y	Negative	4 w-course of IV vancomycin 1 g and metronidazole 500 mg every 12 h, then 4 w-course of oral cefixime 100 mg every 12 h	Good at 3 yr-FUP

(Contd...)

Table 1: (*Continued*).

Authors and year	Age and sex	SEDH site	Associated CF infection/s	Clinical presentation	Coagulation tests	Surgery	SEDH culture	Antibiotic therapy	Outcome
Our case	19, M	R frontal	L fronto-ethmoido-maxillary sinusitis	Lethargy, anisocoria (R>L)	Prolonged INR	Y	Negative	IV ceftriaxone 2 g every 12 h and metronidazole 500 mg every 6 h, postoperatively shifted to oral amoxicillin 875 mg/clavulanate 125 mg every 8 h (6 w-course)	Good at 2 yr-FUP

S. aureus: *Staphylococcus aureus*, *H. influenza*: *Haemophilus influenza*, *P. cepacia*: *Pseudomonas cepacia*, *S. milleri*: *Streptococcus milleri*, *S. anginosus*: *Streptococcus anginosus*, CIII: Cranial nerve III; CF: Craniofacial; F: Female; FUP: Follow-up; IM: Intramuscular; INR: International Normalized Ratio; IV: Intravenous; L: Left; M: Male; mo: Month/s; N: No (not performed); NA: Not available; R: Right; SEDH: Spontaneous epidural hematoma; UE: Upper extremity; V1/V2: Ophthalmic (V1) and maxillary (V2) divisions of the trigeminal nerve; w: week/s; Y: Yes (performed); yr: Year/s.

10 days. His parents denied any history of trauma or intake of any kind of drug, except for nonsteroidal anti-inflammatory drugs (NSAIDs) (Ketoprofen 80 mg, twice a day, for 10 days). On neurological examination, the patient opened his eyes with verbal and painful stimuli; neither focal deficits nor nuchal rigidity was detected (GCS = 14). The CT scan of the brain (plain and bone windows) revealed a large hyperdense biconvex-shaped mass in the right frontal region (6.5 × 3.5 cm) which compressed the surrounding cerebral parenchyma, without evidence of skull fractures. The CT scan did not show signs of pneumocephalus. Furthermore, inflammatory tissue was highlighted in the left ethmoidal cells and in the frontal and maxillary sinuses [Figure 2]. Blood tests pointed out elevated white blood cell count ($13.33 \times 10^3/\mu L$), most of which were neutrophils (89.2%), high C-reactive protein levels (8.3 mg/dL), and an unusual elevated international normalized ratio (INR) (1.41).

Few hours later, the patient became drowsier and confused and developed right anisocoria (right: left pupillary diameter = 4:2). A new CT scan of the brain was immediately obtained, and an increase in the volume of the right frontal hyperdense mass was noticed (8 × 4.7 cm). The patient was intubated and immediately transferred to our neurosurgical operating theater.

A right frontal craniotomy was performed, and a voluminous EDH was evacuated. Intraoperatively, no skull fracture was detected. Similarly, neither dural nor bony vascular malformation was appreciated. The source of bleeding was found in a branch of the right MMA. Histological examination of the intraoperative material pointed out fibrin and leukocytes compatible with hematoma.

After surgery, the patient's anisocoria gradually decreased until he regained isocoria. The postoperative brain CT scan showed a good radiological outcome. The patient was, then, transferred to the Neurosurgical Intensive Care Unit and was started on a regimen of intravenous ceftriaxone 2 g twice a day and metronidazole 500 mg 4 times a day, as advised by the infectious disease specialist. He was extubated on postoperative day 2. The INR value spontaneously normalized 2 days later. Two weeks after the neurosurgical procedure, a high-resolution CT scan with the study of paranasal sinuses was performed [Figure 3], and a functional endoscopic sinus surgery was carried out. A tooth extraction was performed at the same time since an odontogenic origin for the pansinusitis was suspected.

Postoperative laboratory tests were negative for immunologic diseases and coagulopathies.

The patient fully recovered without neurological deficits and was discharged home on oral antibiotic therapy for a total of 6 weeks. A brain magnetic resonance imaging performed 1 month after neurosurgical treatment on suggestion of the

infectious disease specialist showed a good radiological outcome [Figure 4].

DISCUSSION

Craniofacial infections can lead to severe cerebral complications, such as extra- or intradural empyema, meningitis, cerebral abscess, and venous thrombosis. In some rare cases, these infections are associated with SEDHs.[1,3-6,8,10,11,13-15,17,18,20,21,23-25]

The first two cases of SEDH associated with craniofacial infection were described by Schneider and Hegarty[20] in 1951, one related to a middle ear infection, while the other to a furuncle of the ala nasi; the first case associated with sinusitis was indeed reported in 1968 by Kelly and Smith.[8]

As previously stated, SEDH is a rare entity and only nineteen cases associated with craniofacial infection have been described so far[1,3-6,8,10,11,13-15,17,18,20,21,23-25] [Table 1].

Patients are generally adolescents, and a clear male predominance has been demonstrated. SEDHs frequently occur in the frontal area, usually near the site of infection.

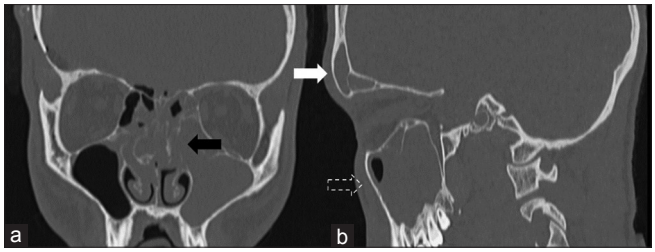

Figure 3: High-resolution paranasal computed tomography scan performed before functional endoscopic sinus surgery. Note the opacification of the left maxillary (b, spotted arrow), ethmoidal (a, black arrow), and frontal sinuses (b, white arrow).

The most common *neurological* reported "symptoms" are headache, confusion, diplopia, and drowsiness; *general* "symptoms" such as fever, nausea, and vomiting are reported as well.

None of the previously analyzed cases was related to a history of head trauma. Moreover, none of the patients enrolled had evidence of an increased bleeding risk condition or of a vascular malformation.

Two pathogenetic hypotheses can explain the origin of the SEDH in these patients.

First, craniofacial inflammatory conditions – like frontal sinus infections – may spread through diploic vascular channels and weaken meningeal vessels, causing arteritis. Damaged vessels are prone to bleed, even after mild trauma such as "barotrauma" (coughing, sneezing, or similar). The histological examination can confirm this theory, showing the presence of polymorphonuclear cells infiltration of intracranial tissues.[14] Second, exudate and air – which are usually produced in such infections – can erode the skull and accumulate in the epidural space, causing dural detachment from the inner table of the skull itself.[6] This mechanism may explain why SEDHs usually occur in younger patients, being dura mater more difficult to detach in older ones.[24]

The first theory is probably the most appropriate for our case, given the absence of pneumocephalus and of any interruption in the bony wall of the inflamed sinus. Yet, it is also true, as reported in the literature, that, in most cases, potential bone defects cannot be macroscopically identified.[23]

We believe that the use of nonsteroidal anti-inflammatory drugs (NSAIDs) plays a key role in both these circumstances. However, NSAIDs reduce platelet aggregation by inhibition of the enzyme cyclooxygenase and the production

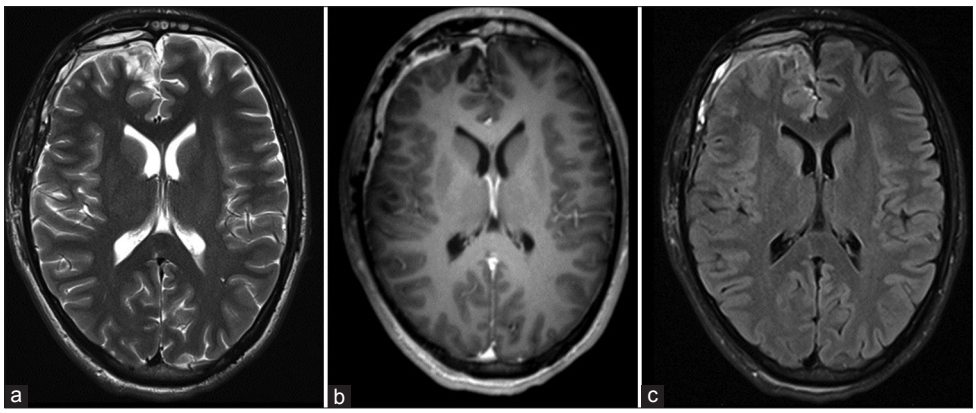

Figure 4: Postoperative brain magnetic resonance imaging (a: T2-weighted image, b: T1-weighted image after injection, c: fluid-attenuated inversion recovery section) showing no recurrences of the epidural hematoma, in the absence of images consistent with vascular malformations of the dura mater.

thromboxane A2.[19] This can lead to an increase in the risk of bleeding, even with "subclinical" trauma, especially after prolonged use.

Although rare, SEDHs should be promptly recognized and surgically treated to avoid permanent neurological deficits and fatal consequences. Laboratory and imaging examinations are important to rule out other causes of SEDH. In the case of a patient with sinusitis and acute onset of headache, nausea and vomiting, neurological deficits, or drowsiness, a brain CT scan should be immediately carried out to exclude a SEDH. If an EDH is diagnosed, immediate neurosurgical treatment coupled with antibiotic therapy can lead to a full patient's recovery.

At present, no guidelines exist for the antibiotic treatment of this condition, due to the scarcity of cases within a consistent lapse of time. However, it is still advisable to give antibiotics even in case of negative cultures since, as pointed out by Xiao,[25] SEDHs may serve as a medium for microorganisms, leading to the development of epidural abscesses.

To conclude, endoscopic treatment of the involved paranasal sinus should be achieved as soon as possible to remove the supposed primary cause of the EDH.[3,4,6]

CONCLUSION

Spontaneous and nontraumatic EDH is a rare entity. The association between this problem and craniofacial infections is rather unusual. Prompt recognition and treatment are mandatory to avoid permanent neurological morbidity and mortality.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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