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REVIEW

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Post-traumatic meningoencephalocele as a complication after head trauma and surgery: literature review focusing on the relevance of patient's history and radiological follow-up

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Abstract

Background Meningoencephalocele (ME) is an herniation of brain parenchyma covered by meninges through a bone defect and could be malformative or secondary. Except for rarer cases of spontaneous form, ME is usually due to endonasal or otologic infections and rarely after head trauma. In predisposed patients, even mild head trauma can lead to the formation of a ME.

Methods We performed a systematic review of literature with the aims to identify the clinical characteristics of all reported forms of post-traumatic ME and the best diagnostic and treatment strategy. We illustrated a case of a patient treated for a post-traumatic subdural hematoma who developed cerebrospinal fluid leakage 3 months after the trauma.

Results The search returned a total of 59 papers for the analysis, including radiological, clinical studies, technical note and the case reported from our experience. The total number of patients collected for this review was 61, with a mean age of 31.1 years. The diagnosis of ME could be heterogeneous in terms of timing and clinical onset after a head injury. Symptoms onset and subsequent radiological diagnosis of ME vary between 24 h to 43 years. The majority of traumas were reported in temporal site (52.45%). There were reported high variability of treatment strategies dependent on the location and extent of the defect: in the majority of cases (58%), duroplasty by the heterologous dural patch was the procedure of choice. There is a relative low rate of complications (6.5%) due to a delayed diagnosis of ME.

Conclusions When ME is associated with violation of meninges, the clinical presentation may be that of cerebrospinal fluid otorrhoea or otorhinorrhoea, consequently, delay in diagnosis can lead to neurological complications. The clinical effectiveness of ME treatment depends much more on the correct and timely diagnosis than on the type of procedure selected.

Keywords Meningoencephalocele, Brain injury, Head trauma, Chronic subdural hematoma, Neurosurgery

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Background

Meningoencephalocele (ME) is a herniation of brain parenchyma covered by meninges through a bone defect and could be malformative or secondary [1, 2]. The subsequent herniation of the brain with its meninges would weaken the dura with its rupture and a final cerebro-spinal fluid (CSF) leakage [2].

A bone defect can allow herniation of brain tissue through its meningeal covering, and frequently occurs in congenital forms of developmental anomalies in which arachnoid granulations can lead to direct contact with bone, resulting in its erosion. For the less frequent acquired forms, on the other hand, a chronic inflammatory status, iatrogenic injury, or rarely head trauma can result in herniation of the meningeal tissue. Chronic inflammations, previous surgery, neoplasms, and irradiation are well-known predisposing factors and can represent the most common causes of a secondary ME [1].

In particular, temporal MEs usually protrude into the middle ear by tegmen tympani. Except for rarer cases of spontaneous ME, it is usually secondary to infections, chronic inflammatory diseases, trauma, and surgery [3–5].

ME symptomatology is often mimicked by rino/otological issues, the diagnosis of spontaneous MEs relies on a high degree of suspicion in any case of unilateral clear leaking prolonged for many days, and rarely months. Consequently, delay in diagnosis can lead to neurological complications [6]. Rarely, forms of ME secondary to head trauma may occur at a late stage from the primary event making diagnosis and subsequent treatment. Such "delayed" forms often find anecdotal evidence in the literature making the correct choice of follow-up management, diagnosis and treatment complicated to manage.

With this review, we report all the cases described in the literature of delayed ME after head trauma analyzing the timing of clinical onset, treatment and complications of this rare event we also want to emphasize the importance of radiological follow-up in head trauma performed also with thin-layer computed-tomography (CT) scan with bone window, necessary to identify late secondary bone defects not visible at the time of diagnosis and eventual first treatment describing a representative case from our experience.

Methods

Eligibility criteria

We performed a review of the literature by analyzing all reported cases of diagnosed ME occurred after a head trauma reported from anamnesis with the aim of identifying the clinical features and a timing of the diagnosis in patients suffering from this uncommon complication.

Therefore, while screening the literature, we adopted the following inclusion and exclusion criteria:

Meta-analysis, case series, clinical study or clinical image reporting cases of patients who suffered from ME secondary to a head trauma;

Conversely, we excluded the following:

cases reported without detailed clinical features of patients; cases reported without description of radiological images; papers that report other pathologies (out of topic); and papers written in languages other than English.

Information sources and search

The English literature was systematically investigated using MEDLINE, the NIH Library, Pubmed and Google Scholar. The last search date was November 1, 2021. The following search terms were used: *Meningoencephalocele and/or Meningoencephalic herniation* with a research string of "*Meningoencephalic herniation*" OR "*Meningoencephalocele*" AND "*traumatic*".

For each case, we reported the number of patients analyzed, age, sex, initial diagnosis at the time of trauma, the time elapsed between the initial traumatic event and the radiological diagnosis of ME, the clinical onset at the time ME was suspected, the cranial region involved, the treatment performed, and the complications reported.

Results

The search returned a total of 92 papers, including radiological, clinical studies and technical note. To this initial cohort, the exclusion mentioned above criteria were applied to abstracts, eliminating 33 papers. The resulting 59 papers are included in our analysis. 37 Articles are subsequently excluded after complete revision of the paper. The list of articles is reassumed to the flowchart in Fig. 1.

The total number of patients collected for this review was 61 including a case from our experience, with a mean age of 31.1 years. Details are reported in Table 1.

The presence of ME is more reported in the male than the female sex (33/61, 54%) following the international epidemiology of head trauma in adults [7].

All reported cases have a history of initial diagnosis of mild to moderate head trauma in which CSF leak, rhinorrhea, otorrhea, or other signs of ME were not identified in the early stages (Table 2).

Only three patients were treated surgically at the time of the first access to the emergency room after the trauma in sites unrelated to the subsequent finding of ME (and therefore excluding iatrogenic formation of ME).

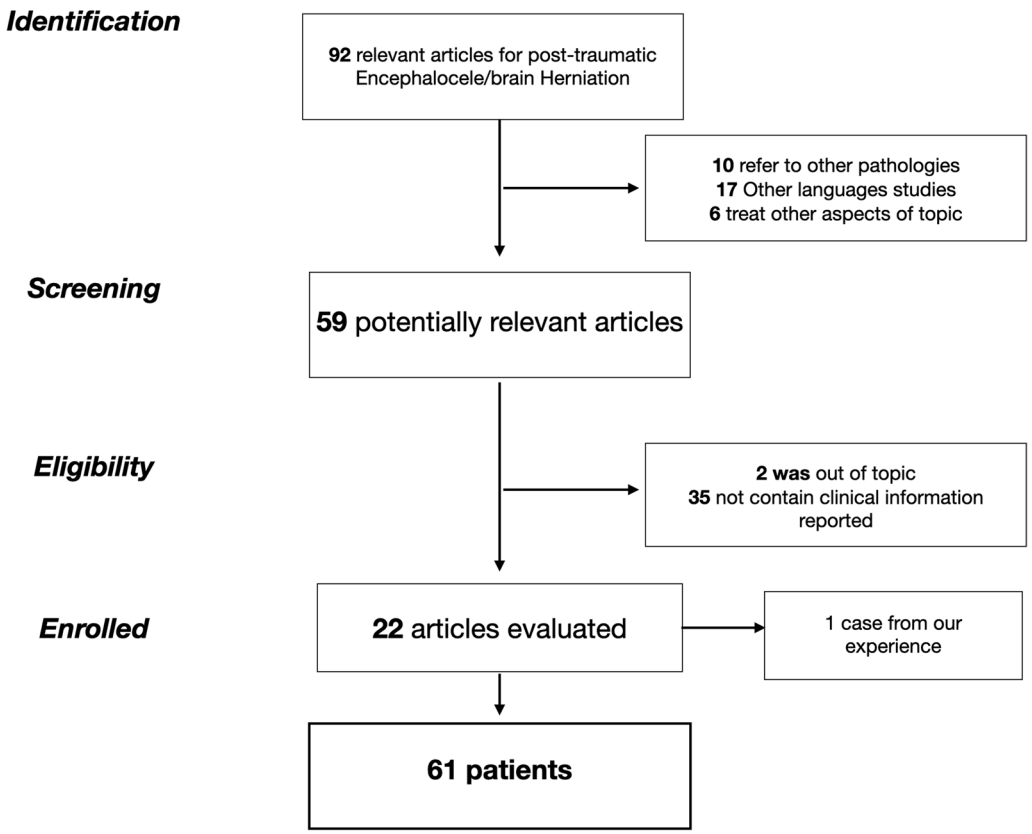

Fig. 1 A flowchart of research strategy in our literature review

Symptoms onset timing and subsequent radiological diagnosis of ME (obtained by magnetic resonance imaging, MRI) is reported as high variable, varying between 24 h (reported in 2 cases) to 43 years. In 15 patients, onset occurred in the range of days (with a mean of about 12 days); in most cases, the diagnosis remained silent for months or years. Trauma is reported in the frontal site in 19/61 cases (31.1%), in the parietal or parieto-occipital area in 10/61 cases (16.4%), and temporal site in 32/61 cases (52.45%). Therefore, wide variability of ME onset symptoms follows, wherein the majority of cases, there are otological symptoms (27/61, 44.26%), CSF leak (10/61, 16.4%), ocular symptoms (such as ecchymosis, exophthalmos, ptosis, in 8/61 cases, 13.1%). In comparison, there are less frequent cases of new-onset neurological disorders (3/61 seizure 4.9%, neurological deficits 2/61 3.3%, weakness 1/61 1.6%, loss of consciousness 2/61, 3.3%), headache and swelling of the injured area (3/61, 4.9% and 4/61, 6.55% respectively). 4 patients started with symptoms referable to meningitis (fever and neurological disorders, nuchal rigidity).

The treatment of this condition involves various reported treatment strategies highly dependent on the location and extent of the defect: in the majority of cases (29/50 procedures reported, 58%), duroplasty by the heterologous dural patch was the procedure of choice, followed by bone reconstruction alone frequently performed in cases of intradiploic herniation (8/50 procedures reported, 16%), autologous flap reconstruction with galea flap was performed in 6/50 cases reported (12%), 4 patients were performed with endoscopic nasal repair (8%), and 3 subjects reported received conservative treatment.

From the perspective of outcome analysis, there is a low rate of complications due to delayed diagnosis of ME. The only 4 cases (6.5%) that presented problems in the follow-up (3 meningitis and one ocular pro-ptosis) were those for whom a bone-only or conservative reconstruction procedure was preferred.

Representative case

A 77-year-old male patient was subdued to a chronic subdural hematoma (CSDH) evacuation at our Neurosurgical Department. The postoperative course was uneventful, and the patient was discharged at home a week later. After four months, patient started to suffer a water-like leakage by the left ear. Anamnesis revealed a history of paroxistic ear leakage during youth after

Table 1 Patient's demographics

No.	Authors	Pts	Age/mean age	Sex	Past diagnosis	Time from trauma	Clinical debut of ME	Site	Treatment	Complications
1	Acakpo-Satchivi et al. [21]	1	1	M	Subdural chronic hematoma	8 months	Seizure	Fronto-polar region	Duroplasty	None
2	Canto et al. [34]	1	26	M	Facial fractures	18 days	Headache	Fronto-basal	Pericranium flap	None
3	Shi et al. [35]	1	45	M	Closed parietal skull fracture not surgically treated	39 years	Lower extremities weakness	Right para-sagittal dura defect	Duroplasty	None
4	Gupta et al. [36]	1	1	F	Parietal skull fracture	3 months	Hemiparesis	Left para-sagittal dura defect	Duroplasty	None
5	Zhao et al. [37]	1	30	M	Subdural acute hematoma	1 day	Loss of consciousness	Fronto-orbital	Pericranium flap	None
6	Arevalo-Perez [38]	1	84	F	Mild head trauma	Many years	Dizziness, Seizure	Right parietal defect	Duroplasty	None
7	Mohindra et al. [39]	2	1	M	Right parietal bone fractures	7 days	Swelling surgical site	Right parietal defect	Duroplasty	None
			1	M	SAH	5 days	Swelling surgical site	Righ parieto-occipital region	Duroplasty	None
8	Abhinav Aggarwal et al. [40]	1	12	M	Intraparenchimal hemorrhage	1 day	Loss of consciousness	Fronto-orbital	Conservative	None
9	Akin Akakin et al. [12]	1	39	M	Subdural chronic hemorrhage	1 year	Headache	Right parietal defect	Duroplasty	None
10	Antonelli et al. [28]	6	21	M	Orbital Roof Fracture	9 days	exophthalmos	Fronto-orbital	Bone reconstruction	None
			9	M	Orbital Roof Fracture	12 days	ecchymosis	Fronto-orbital	Bone reconstruction	None
			61	M	Orbital Roof Fracture	25 days	ecchymosis	Fronto-orbital	Bone reconstruction	None
			20	M	Orbital Roof Fracture	21 days	exophthalmos	Fronto-orbital	Bone reconstruction	None
			29	M	Orbital Roof Fracture	15 days	ecchymosis and exophthalmos	Fronto-orbital	Bone reconstruction	None
			25	M	Orbital Roof Fracture	5 days	ecchymosis and exophthalmos	Fronto-orbital	Bone reconstruction	None
11	Ha et al. [41]	2	66	F	Orbital Roof Fracture, Subdural acute Hematoma	3 days	Cognitive impairment, aphasia	Fronto-orbital	Pericranium flap	None
			48	M	Orbital Roof Fracture	4 days	exophthalmos	Fronto-orbital	Pericranium flap	None
12	Li Xue et al. [42]	4	56	M	lateral wall of the sphenoid sinus	\	Rhinorrea	Fronto-nasal	Endoscopic repair	None
			41	F	lateral wall of the sphenoid sinus	\	Rhinorrea	Fronto-nasal	Endoscopic repair	None
			28	F	Ethmoid roof	\	Rhinorrea	Fronto-nasal	Endoscopic repair	None
			1	M	Sieve plate	\	Rhinorrea	Fronto-nasal	Endoscopic repair	None
13	Moudrous W. et al. [43]	1	76	M	Left parietal region	43 years	Dizzines	Left parietal defect	\	None

Table 1 (continued)

No.	Authors	Pts	Age/mean age	Sex	Past diagnosis	Time from trauma	Clinical debut of ME	Site	Treatment	Complications
14	Kandemirli et al. [44]	1	11	M	Observation after mild trauma	8 years	Seizure	Left occipital defect	Conservative	None
15	Patil et al. [45]	1	64	M	None	1 year	Cranic mass	Left parietal defect	Duroplasy	None
16	Sanna et al. [46]	10	31.5	\	\	\	Hearing loss (9), CSF leak (3), Meningitis (1)	Temporal Bone defect	Bone reconstruction	2 meningitis
17	Arslan et al. [47]	1	5	F	Observation after mild trauma	2 days	Ecchimosis	Orbital	Resection and bone reconstruction	\
18	Grinblat et al. [48]	21	31.5	11 F	Head trauma	Mean 22.6 months	18 otologic symptoms, 3 Meningitis	Temporal Bone	Surgical repair with duroplasty	\
19	Satyarthee et al. [49]	1	4	M	Head trauma with linear fracture	3 years	Progressive swelling of frontal bone	Right frontal region orbit roof	Bone reconstruction	Orbital propoptosis
20	Menku¨ et al. [50]	1	30	F	Head mild trauma	5 years	Headache	Right parietal defect	Pericranium flap	None
21	Cullu et al. [51]	1	18	M	Head trauma	14 days	Rhinorrea	left frontal sinus inferior posterior and the left ethmoid sinus superior posterior wall	Conservative	Meningitis
22	Our case	1	77	M	Subdural chronic hematoma	4 months	Othorrea	Temporal Bone	Pericranium flap	None

Table 2 Group analysis

Total patients 61	
Age	Mean: 31 Min–Max: 1–84
Sex	M: 33/61—54%
	F: 28/61—47%
Main event (reported in 51 patients)	Skull linear fracture: 25/51: 49%
	Orbital roof fracture: 8/51: 15.7%
	Mild trauma without evident fracture: 7/51: 13.7%
	Facial bone fracture: 6/51: 11.7%
	Subdural hematoma: 5/51: 9.8%
	Intracranial hemorrhage: 2/51: 3.9%
Location of direct trauma	Frontal: 19/61 cases—31.1%
	Parieto-occipital 10/61 cases—16.4%
	Temporal: 32/61 cases—52.45%
Timing	Min: 24 h Max: 43 years
Onset symptoms at diagnosis of ME	Otological symptoms—27/61—44.26%
	CSF leak—10/61—16.4%
	Ocular symptoms—8/61—13.1%
	Seizure—3/61—4.9%
	Neurological deficits—2/61—3.3%
	Weakness—1/61—1.6%
	Loss of consciousness 2/61—3.3%
	Headache—3/61—4.9%
	Swelling—4/61—6.55%
Treatment	Duroplasty—29/50—58%
	Bone reconstruction 8/50—16%
	Galea flap 6/50—12%
	Endoscopic repair 8%
	Observation 3%
Complications	4—6.5%

recurrent ear infections. Leakage's chemical examination with high values of β2-transferrin revealed the CSF nature. A high-resolution CT scan (HRCT) evidenced the presence of phlogistic tissue in the left tympanic cavity and mastoid antrum with tegmen tympani erosion and a skull-base significant defect. Subsequently, a brain MRI was performed and confirmed the presence of a temporal ME through the skull base defect; moreover, signs of chronic flogosis were present, as tympanic cavity and mastoid cells obliteration were found. The aforementioned conditions necessitated a surgical repair with a subtemporal approach [8, 9], whereas the point of herniation was repaired by muscle, heterologous dural patch, and fibrin glue [10].

The postoperative course was uneventful, and the patient was discharged one week after. At one year follow-up, no signs of CSF leakage were present. (Fig. 2).

Discussion

In Neurosurgery centers with a high turnover of traumatic pathology, it is not unusual to find cases of delayed post-traumatic complications. From the literature search we conducted, the occurrence of late ME long after trauma is an under-described and under-reported occurrence; however, we believe that it may be more common than what is normally believed and that the issue is related to the reduced follow-up of hospital centers and by the misdirection of follow-up radiological examinations. In predisposed patients, even mild head trauma can lead to extrinsic theca defects that can lead to the formation of an ME. CSF pulsation through the dural defect may cause progressive erosion of the bone and enlargement of the extradural collection. On the other side, osteogenesis promoted by the dural and periosteal layers would limit the collection to the diploic space and

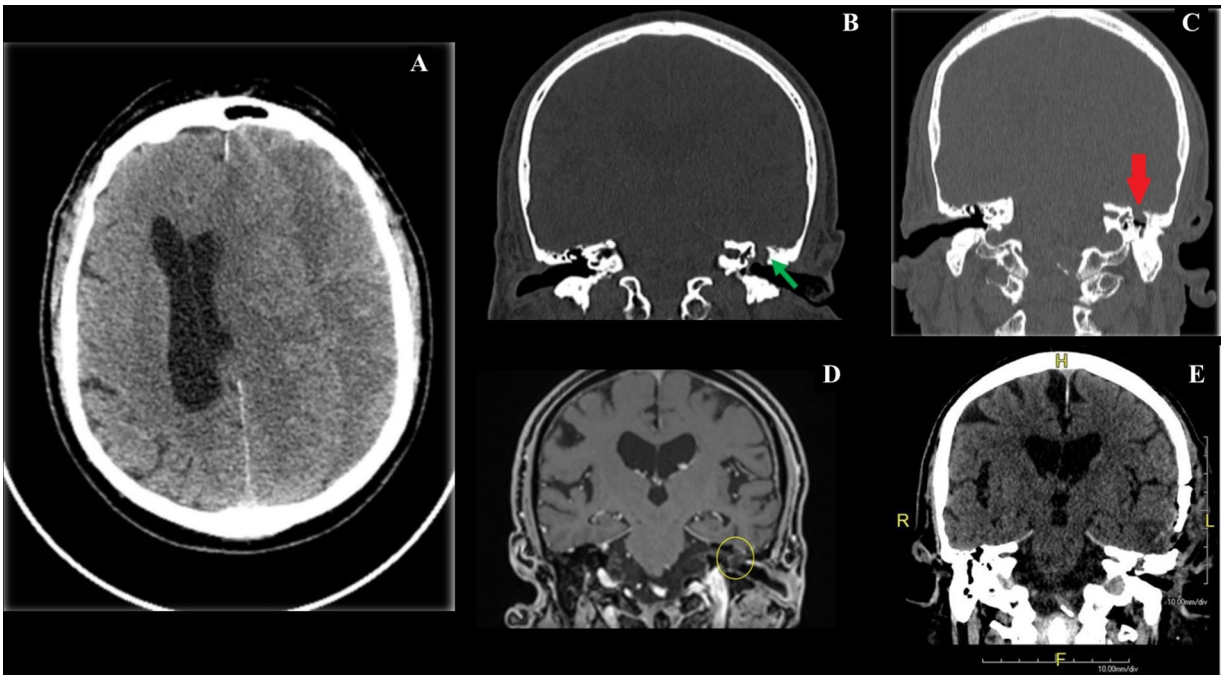

Fig. 2 **A** The patient following cognitive impairment and right sensorimotor neurological deficit underwent an initial cranial CT scan documenting large CSDH and subsequently underwent evacuation surgery by craniotomy hole. After four months, patient started to suffer from otorrhea of the left ear. A high-resolution CT scan (HRCT) evidenced the presence of phlogistic tissue in the left tympanic cavity and mastoid antrum with tegmen tympani erosion and a skull-base significant defect (**B**, **C**, red and green dot). A brain MRI was performed and confirmed the presence of a temporal ME through the skull base defect (**D**). The patient underwent surgical repair with a subtemporal approach, and the herniation site was repaired with muscle, heterologous dural patch, and fibrin glue. Postoperative follow-up HCT documented a successful procedure without complications (**E**)

eventually remodel the skull profile in a slow process. These factors probably account for the missed evidence of fracture at the diagnosis many months after the initial head injury and the underlying defects can result in post-traumatic ME often with clinical presentation of otorrhoea or otorhinorrhoea as consequence of violation of meninges and relative cerebrospinal fluid (CSF) leak (in more than 50% of cases). In addition, the defects provide a route for the spread of infection into the intracranial cavity, resulting in recurrent episodes of meningitis and brain abscess. Symptoms in delayed ME may be absent, particularly if the herniated cortex is non-eloquent, or may be subtle and chronic due to irritated cortex. Acute clinical onset after physiological conditions with the elevation of intracranial pressure has been anecdotally reported [18], and our analysis shows that neurological symptoms are infrequent compared with local symptoms such as rhinorrhea or delayed otorrhea.

Clinical history and radiological investigation with high-resolution CT scan (HRCT) and MRI may be the only way to make a diagnosis [1], although even with a bone thin-layer scan is not easy to define a bone defect [11], especially after orbit roofs fractures [28, 52] and after surgical treatments resulting from the trauma [15, 29–32]. In fact, surgical treatment can promote this phenomenon even in a minor procedure in some predisposed patients. An example is ME with a CSF leak that can be found after the treatment of a CSDH-patient with mild head trauma [12] as is reported in our illustrative case. Neuro-radiological examinations, including a thin-layer bone CT scan, revealed that a temporal ME was the cause of leakage. CSDH is a typical condition among 65 years old or older patients, and it is usually related to a mild trauma [22, 23]. The entire process would take about 20 days. Consequently, the patient becomes symptomatic when CSDH exerts a local mass effect on surrounding parenchyma with a subsequent intracranial pressure elevation [23–26]. After the uncomplicated discharge, patients referred us with otorrhea. A more profound patient interview revealed episodes of chronic otitis during his youth. Since the most common cause of acquired temporal MEs actually is chronic otitis media [19] and are also the most frequent secondary form of delayed ME; we argued that a congenital thinner tegmen tympani broke because of intracranial hypertension with a subsequent herniation of the brain, thus excluding head injury as a direct cause of ME, but defining it as a contingent and triggering cause. Nevertheless, the

initial dura covering was gradually damaged by a middle ear inflammatory process [33], leading to arachnoid disruption, and favored by the intracranial pressure variations, to the CSF otorrhea. Rarely a head trauma with CSDH can be caused by a spinal CSF leakage and intracranial hypotension, especially in younger patients, but in some case we believed that could be a predisposing factor for ME. Kenning et al. [13] suggested that patients with a CSF opening pressure > 20 cmH_2O, BMI > 30 kg/m^2, and other predisposing factors should be considered for ventriculoperitoneal shunting after temporal ME repairing. Although CSDH can cause intracranial hypertension, is not considered a typical risk factor for temporal ME, but in adult patients with recurrent CSH and prior presence of bilateral CSH, a survey for an underlying spinal CSF leak should be considered in the differential, especially on a remote trauma or inflammatory disease in clinical history [12].

Whether this is an unusual associated primary injury [14–17], an effect of the chosen therapy, or a combination of these factors, is unclear [20]. Repair of the dural tearing is the aim of surgery, along with decompression of the herniated cortex if incarcerated or strangulated.

Treating physicians should be aware of this unusual complication, especially in the development of new symptoms during therapy [21]. As a spontaneous CSF leakage, literature agrees that a greater than one-week duration should represent an indication for surgical repair [1, 8, 9, 27].

Formation to that of growing skull fractures suggested that brain pulsations played a significant role in pushing the cortex through the neo-membrane in the same way that the leptomeninges are forced through dural calvarial defects in patients with growing fractures. This proposal seems logical, especially given that we could not find a report suggesting that this particular complication has been reported in the adult population [15, 29].

Further study and limitation

Our study has several limitations: first, we pointed out that most of the reported cases are either part of larger studies or report only the salient aspects of treatment focusing little on the timing of onset and delayed clinical onset. Second, little consideration is given to predisposing aspects of ME formation such as a history of chronic sinusitis or chronic otitis that may be risk factors even in mild trauma.

Conclusion

Various factors play a role in the etiopathogenesis of delayed post-traumatic ME, and a bony defect alone is not sufficient for meningoencephalic herniation to develop. We would alert physicians caring for a patient with mild head trauma or CSDHs to be aware of this unusual complication. Although the development of new symptoms should be a reason for patients to undergo neuroimaging, we would also suggest that the identification of areas of cortical irregularity along the inner surface of subdural collections may be an indication for regular and relatively frequent follow-up imaging executed with thin-layer bone CT scan even if a bone defect was not revealed at first examinations.

Abbreviations

ME	Meningoencephalocele
CSF	Cerebrospinal fluid
CT	Computed-tomography
CSDH	Chronic subdural hematoma
HRCT	High-resolution CT scan
MRI	Magnetic resonance imaging

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Author contributions

AP: manuscript, draft DA: data collection, research, GP surgical operator, AP: supervision.

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Availability of data and materials

On demand to the corresponding author.

Declarations

Ethics approval and consent to participate

All procedures performed in studies involving human participants were by the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any authors.

Consent for publication

Informed consent was obtained from all individual participants included in the study. The patient has consented to submitting this review article to the journal.

Competing interests

We wish to confirm that there are no known conflicts of interest associated with this publication, and there has been no significant financial support for this work that could have influenced its outcome. We wish to draw the attention of the Editor to the following facts, which may be considered potential conflicts of interest and to significant financial contributions to this work.

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References

1. Grinblat G, Dandinarasaiah M, Prasad SC, et al. Temporal bone meningo-encephalic-herniation: etiological categorization and surgical strategy. Otol Neurotol. 2018;39(3):320–32.
2. Nahas Z, Tatlipinar A, Limb CJ, Francis HW. Spontaneous meningoencephalocele of the temporal bone: clinical spectrum and presentation. Arch Otolaryngol Head Neck Surg. 2008;134(5):509–18.
3. Ramalingam KK, Ramalingam R, SreenivasaMurthy TM, Chandrakala GR. Management of temporal bone meningo-encephalocoele. J Laryngol Otol. 2008;122(11):1168–74.
4. Yang E, Yeo SB, Tan TY. Temporal lobe encephalocoele presenting with seizures and hearing loss. Singap Med J. 2004;45(1):40–2.
5. Stucken EZ, Selesnick SH, Brown KD. The role of obesity in spontaneous temporal bone encephaloceles and CSF leak. Otol Neurotol. 2012;33(8):1412–7.
6. Gubbels SP, Selden NR, Delashaw JB Jr, McMenomey SO. Spontaneous middle fossa encephalocele and cerebrospinal fluid leakage: diagnosis and management. Otol Neurotol. 2007;28:1131–9.
7. Dewan MC, Rattani A, Gupta S, Baticulon RE, Hung YC, Punchak M, Agrawal A, Adeleye AO, Shrime MG, Rubiano AM, Rosenfeld JV, Park KB. Estimating the global incidence of traumatic brain injury. J Neurosurg. 2018. https://doi.org/10.3171/2017.10.JNS17352.
8. Pelosi S, Bederson JB, Smouha EE. Cerebrospinal fluid leaks of temporal bone origin: selection of surgical approach. Skull Base. 2010;20(4):253–9.
9. Kutz JW Jr, Johnson AK, Wick CC. Surgical management of spontaneous cerebrospinal fistulas and encephaloceles of the temporal bone. Laryngoscope. 2018;128(9):2170–7.
10. Papanikolaou V, Bibas A, Ferekidis E, Anagnostopoulou S, Xenellis J. Idiopathic temporal bone encephalocele. Skull Base. 2007;17(5):311–6.
11. Armocida D, Pesce A, Frati A, Miscusi M, Paglia F, Raco A. Pneumoventricle of unknown origin: a personal experience and literature review of a clinical enigma. World Neurosurg. 2019;122:661–4. https://doi.org/10.1016/j.wneu.2018.11.050.
12. Akakin A, Yilmaz B, Eksi MS, et al. Recurrent cranial chronic subdural hematoma due to cervical cerebrospinal fluid fistula: repair of both entities in the same session. J Craniofac Surg. 2016;27(6):e578-580.
13. Kenning TJ, Willcox TO, Artz GJ, Schiffmacher P, Farrell CJ, Evans JJ. Surgical management of temporal meningoencephaloceles, cerebrospinal fluid leaks, and intracranial hypertension: treatment paradigm and outcomes. Neurosurg Focus. 2012;32(6):E6.
14. Doglietto F, Sabatino G, Policicchio D, Tirpakova B, Albanese A. Transcranial cerebral herniation after chronic subdural hematoma treatment with no dura closure. Neurology. 2006;67(3):493.
15. Kusano Y, Horiuchi T, Seguchi T, Kakizawa Y, Tanaka Y, Hongo K. Local brain herniation after partial membranectomy for organized chronic subdural hematoma in an adult patient: case report and review of the literature. Brain Inj. 2010;24(9):1118–21.
16. Naidich TP, Altman NR, Braffman BH, McLone DG, Zimmerman RA. Cephaloceles, related malformations. AJNR Am J Neuroradiol. 1992;13:655–60.
17. Ceccherini AF, Jaspan T. Cerebral herniation through a sub-dural membrane defect following non-accidental injury. Clin Radiol. 1999;54:550–2.
18. Chen JC, Levy ML. Causes, epidemiology, and risk factors of chronic subdural hematoma. Neurosurg Clin N Am. 2000;11(3):399–406.
19. Gupta A, Sikka K, Irugu DVK, Verma H, Bhalla AS, Thakar A. Temporal bone meningoencephaloceles and cerebrospinal fluid leaks: experience in a tertiary care hospital. J Laryngol Otol. 2019. https://doi.org/10.1017/S0022215119000203.
20. Scerrati A, Germano A, Trevisi G, et al. Timing of low-dose aspirin discontinuation and the influence on clinical outcome of patients undergoing surgery for chronic subdural hematoma. World Neurosurg. 2019;129:e695–9.
21. Acakpo-Satchivi L, Luerssen TG. Brain herniation through an internal subdural membrane: a rare complication seen with chronic subdural hematomas in children. Case report. J Neurosurg. 2007;107:485–8.
22. Sahyouni R, Goshtasbi K, Mahmoodi A, Tran DK, Chen JW. Chronic subdural hematoma: a historical and clinical perspective. World Neurosurg. 2017;108:948–53.
23. Lee KS. Natural history of chronic subdural haematoma. Brain Inj. 2004;18(4):351–8.
24. Iliescu IA, Constantinescu AI. Clinical evolutional aspects of chronic subdural haematomas—literature review. J Med Life. 2015;8:26–33.
25. Nakaguchi H, Tanishima T, Yoshimasu N. Factors in the natural history of chronic subdural hematomas that influence their postoperative recurrence. J Neurosurg. 2001;95(2):256–62.
26. Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. Am J Otol. 1997;18(2):188–97.
27. Motoyama Y, Isaka F, Nabeshima S. Internal intracranial encephalocele reduced by a subdural-peritoneal shunt. Case illustration. J Neurosurg. 2002;96:966.
28. Antonelli V, Cremonini AM, Campobassi A, Pascarella R, Zofrea G, Servadei F. Traumatic encephalocele related to orbital roof fractures: report of six cases and literature review. Surg Neurol. 2002;57(2):117–25. https://doi.org/10.1016/s0090-3019(01)00667-x.
29. Doglietto F, Sabatino G, Policicchio D. Transcranial cerebral herniation after chronic subdural hematoma treatment with no dura closure. Neurology. 2006;67:493.
30. Mazzucchi E, Bianchi F, Vigo V, Anile C, Frassanito P. Parietal intradiploic encephalocele in an adult: a delayed complication of pediatric head injury? Childs Nerv Syst. 2017;33(2):217–9. https://doi.org/10.1007/s00381-016-3323-8.
31. Loumiotis I, Jones L, Diehn F, Lanzino G. Symptomatic left intradiploic encephalocele. Neurology. 2010;75:1027. https://doi.org/10.1212/WNL.0b013e3181f25f31.
32. Lundy LB, Graham MD, Kartush JM, et al. Temporal bone encephalocele and cerebrospinal fluid leaks. Am J Otol. 1996;17:461Y9.
33. Feenstra L, Sanna M, Zini C, Gamoletti R, Delogu P. Surgical treatment of brain herniation into the middle ear and mastoid. Am J Otol. 1985;6:311–5.
34. Canto AMD, Pinotti MM, Maciel FA, Todeschini AB, Aguiar GB, Freitas RR. Treatment of a large traumatic encephalocele with titanium mesh. Craniomaxillofac Trauma Reconstr. 2017;10(2):145–50. https://doi.org/10.1055/s-0036-1582452.
35. Shi C, Flores B, Fisher S, Barnett SL. Symptomatic parietal intradiploic encephalocele: a case report and literature review. J Neurol Surg Rep. 2017;78(1):e43–8. https://doi.org/10.1055/s-0037-1599799.
36. Gupta DK, Mahapatra AK. Posttraumatic intradiploic pseudomeningocele: neuroimaging. Pediatr Neurosurg. 2006;42(3):200–1. https://doi.org/10.1159/000091869.
37. Zhao D, Tao S, Zhang D, Qin M, Bao Y, Wu A. "Five-layer gasket seal" watertight closure for reconstruction of the skull base in complex bilateral traumatic intraorbital meningoencephaloceles: a case report and literature review. Brain Inj. 2018;32(6):804–7. https://doi.org/10.1080/02699052.2018.1440631.
38. Arevalo-Perez J, Millán-Juncos JM. Parietal intradiploic encephalocele: report of a case and review of the literature. Neuroradiol J. 2015;28(3):264–7. https://doi.org/10.1177/1971400915592554.PMID:26246093;PMCID:PMC4757295.
39. Mohindra S, Chhabra R, Gupta R, Gupta SK, Khosla VK. A report of two "traumatic encephaloceles"; an unrecognized entity. Brain Inj. 2007;21(10):1087–91. https://doi.org/10.1080/02699050701598051.
40. Aggarwal A, Gupta AK, Aggarwal AK. Acute post-traumatic encephalocele in a child: CT and MRI features. BJR Case Rep. 2016;2(4):20150170. https://doi.org/10.1259/bjrcr.20150170.
41. Ha AY, Mangham W, Frommer SA, Choi D, Klinge P, Taylor HO, Oyelese AA, Sullivan SR. Interdisciplinary management of minimally displaced orbital roof fractures: delayed pulsatile exophthalmos and orbital encephalocele. Craniomaxillofac Trauma Reconstr. 2017;10(1):11–5. https://doi.org/10.1055/s-0036-1584395.
42. Xue L, Gehong D, Ying W, Jianhua T, Hong Z, Honggang L. Nasal meningoencephalocele: a retrospective study of clinicopathological features and diagnosis of 16 patients. Ann Diagn Pathol. 2020;49:151594. https://doi.org/10.1016/j.anndiagpath.2020.151594.
43. Moudrous W, Boogaarts HD, Grotenhuis JA. A meningoencephalocele caused by a chronic growing skull fracture in a 76-year-old patient. World Neurosurg. 2016;96:604.e1-604.e2. https://doi.org/10.1016/j.wneu.2016.08.052.
44. Kandemirli SG, Candan S, Bilgin C. Post-traumatic occipital intradiploic encephalocele. World Neurosurg. 2019;129:9–12. https://doi.org/10.1016/j.wneu.2019.05.174.
45. Patil AA, Etemadrezaie H. Posttraumatic intradiploic meningoencephalocele. Case report. J Neurosurg. 1996;84(2):284–7. https://doi.org/10.3171/jns.1996.84.2.0284.

46. Sanna M, Fois P, Russo A, Falcioni M. Management of meningoencephalic herniation of the temporal bone: Personal experience and literature review. Laryngoscope. 2009;119(8):1579–85. https://doi.org/10.1002/lary.20510. Erratum in: Laryngoscope. 2010;120(1):217. Paolo, Fois [corrected to Fois, Paolo]
47. Arslan E, Arslan S, Kalkısım S, Arslan A, Kuzeyli K. Long-term results of orbital roof repair with titanium mesh in a case of traumatic intraorbital encephalocele: a case report and review of literature. Craniomaxillofac Trauma Reconstr. 2016;9(3):255–9. https://doi.org/10.1055/s-0036-1581064.
48. Grinblat G, Dandinarasaiah M, Prasad SC, Piras G, Piccirillo E, Fulcheri A, Sanna M. Temporal bone meningo-encephalic-herniation: etiological categorization and surgical strategy. Otol Neurotol. 2018;39(3):320–32. https://doi.org/10.1097/MAO.0000000000001693.
49. Satyarthee GD, Kumar A. Posttraumatic giant fronto-orbito encephalocele causing cosmetic disfiguring forehead swelling with proptosis: management. J Pediatr Neurosci. 2016;11(4):341–3. https://doi.org/10.4103/1817-1745.199476.
50. Menkü A, Koç RK, Tucer B, Akdemir H. Is skull fracture necessary for developing an intradiploic pseudomeningocele as a complication of head injury in adulthood? Acta Neurochir (Wien). 2004;146(6):623–7. https://doi.org/10.1007/s00701-004-0263-y. (**discussion 627**).
51. Cullu N, Deveer M, Karakas E, Karakas O, Bozkus F, Celik B. Traumatic fronto-ethmoidal encephalocele: a rare case. Eurasian J Med. 2015;47(1):69–71. https://doi.org/10.5152/eajm.2014.50.
52. Becelli R, Palmieri M, Facchini V, Armocida D, Pesce A, Kapitonov A, Zappalà M, Colangeli W, Bozza F, Salvati M, Santoro A, Frati A. Management of frontal sinus fractures: a comprehensive review and treatment algorithm from Sapienza university of Rome. Interdiscip Neurosurg. 2021;26:101318. https://doi.org/10.1016/j.inat.2021.101318.

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