Pediatric septic thrombosis of intracranial venous sinuses: from diagnosis to discharge. Twenty years of experience

Cristina Cortés-Ledesma, Alberto García-Salido, Lucía Garriga-Ferrer Bergua, Marta Cabrero-Hernández, Laura Palomino-Pérez, Gema de Lama-Caro Patón, Raquel Jiménez-García

Introduction. Septic thrombosis of intracranial venous sinuses (STSV) is a rare and severe complication of cranial infections.

Materials and methods. The main objective of this paper is to describe the clinical data, diagnostic procedures, treatment and evolution of a series of cases of STSV. In addition, the current literature is reviewed. Observational retrospective study by review of medical histories (January 1995-December 2016). The data collected were: clinical, analytical, epidemiological, microbiological, radiological, management and follow-up. A descriptive and statistical analysis of the data was done.

Results. Twelve children were included (86,832 admissions studied). They have a median age of 4.5 years (range 1-13) with a median time of symptoms of 6 days (range 1-25). At admission, the clinical data were: fever (11/12), vomiting (9/12) and headache (8/12). They also showed bad general status 12/12, 7/12 acute otitis media and 5/12 VI cranial nerve paresis. The lumbar puncture was pathological in 4/12. The most frequently microorganism isolated was *Streptococcus* sp. Prothrombotic mutations were confirmed on 2/12. Cranial computed tomography allowed diagnosis in 9/12; the magnetic resonance imaging achieves that in 12/12. Previous neurological signs or time to diagnosis did not influence the appearance of other image complications. All received antibiotic treatment, heparin 10/12 and 11/12 surgery. There were no sequels.

Conclusion. In our series otitis, headache, vomiting and fever were prevalent. Complementary tests allowed the suspect but the definitive diagnosis was obtained by neuroimaging. There were no sequels and the therapies were mainly wide broad-spectrum antibiotics, heparin, and surgical.

Key words. Cerebral venous sinus. Children. Heparin. Otomastoiditis. Septic thrombosis. Sinusitis.

Emergency Department (C. Cortés-Ledesma, L. Garriga-Ferrer Bergua); Pediatric Intensive Care Unit (A. García-Salido, M. Cabrero-Hernández, G. de Lama-Caro Patón); Pediatrics (L. Palomino-Pérez, R. Jiménez-García). Hospital Infantil Niño Jesús. Madrid, Spain.

Correspondence:
Dr. Alberto García Salido. Pediatric Intensive Care Unit. Hospital Infantil Universitario Niño Jesús. Av. Menéndez Pelayo, 65. E-28009 Madrid.

E-mail:
citopensis@yahoo.es

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Introduction

The septic thrombosis of intracranial venous sinuses (STSV) is an infrequent cranial infection complication. With an incidence of 0.67 cases per 100,000 children/year in the recent past it was the first cause of cerebral sinus thrombosis, with mortality nearly close to 100%. Nowadays, despite better management, STSV is usually described as an important cause of morbidity [1].

The absence of venous valves in the dural sinuses and cerebral emissary veins is the cornerstone of STSV physiopathology. Thrombotic emboli could easily move from near infected regions [2] like sphenoid and ethmoidal sinus [3].

The STSV diagnosis has been historically described through a typical triad: chemosis, proptosis and ophthalmoplegia. These signs and symptoms are not always presented in children so the attending clinical experience is a key to its suspicion. Also, the complementary tests are not always helpful. The blood counts or acute phase reactants usually does not help to identify STSV. Only imaging tests such magnetic resonance and computed tomography may allow an accurate diagnosis and not always in the very first moments of attendance [4].

In this paper, our group describe two decades of pediatric STSV. We made a review of clinical records, complementary tests, therapies applied and evolution. Also, we studied the complications derived from this disease.

Materials and methods

Retrospective observational study approved by the ethics committee of a tertiary pediatric monographic hospital. We reviewed the medical records

of children admitted with STSV diagnosis from January 1st, 1995 to December 31st, 2016. The data collected were under the Personal Data Protection Act, 15/1999.

Inclusion criteria

Both must be present:
- A STSV diagnosis at hospital discharge.
- STSV confirmed and described by a radiologist after a computed tomography (CT) or magnetic resonance imaging (MRI).

Data collected

Demographic data: age and gender.
- Clinical data: previous illness, pneumococcal vaccination, symptomatology in the emergency room, time of evolution, time from the onset of the first symptoms to suspect, previous treatments received.
  - In the group of patients with acute otitis media (AOM): antibiotic treatment before admission was considered 'adequate' if they had received at least 7 days of amoxicillin or amoxicillin-clavulanic at 80 mg/kg/day.
- Analytical data: blood count (leukocytes, neutrophils, hemoglobin), coagulation (prothrombin index, international normalized ratio or INR, time of cephalin and fibrinogen) and acute phase reactants (C reactive protein or CRP in mg/dL and procalcitonin or PCT in μg/mL). Prothrombotic study: levels of coagulant VIII factor (% at the acute time and during follow-up), antithrombin (%), anticardiolipin antibodies (IgM and IgG in UGPL/mL), antiBeta 2 antibodies (IgG and IgM in U/mL) and levels of lupus anticoagulant (acutely and during follow-up) and prothrombotic mutations.
  - In those patients with symptoms suggestive of meningitis, the data related to lumbar puncture (pathological/normal) are collected.
- Microbiological data: blood culture.
  - In those patients who undergo surgery, culture of the infectious focus if collected.
  - In case of lumbar puncture, cerebrospinal fluid (CSF) culture.
- Radiological data: presence of complications seen by image defined as abscesses or erosion of the bone cortex.
- Therapeutic data:
  - Antibiotherapy according to the center protocol (cefotaxime at 200-300 mg/kg/day, and vancomycin 60 mg/kg/day and/or metronidazole 30 mg/kg/day), treatment time and if this is subsequently changed to oral (amoxicillin-clavulanic at 80 mg/kg/day, or cefpodoxime proxetil at 10 mg/kg/day).
  - Anticoagulant treatment (low molecular weight heparin at a dose of 1 mg/kg/12 hours, with dose adjustment depending on the levels of anti Xa during the hospital stay, or acenocoumarol according to INR controls) and the time of administration.
  - Corticosteroid treatment (dexamethasone at doses 0.15-0.6 mg/kg/day).
- Evolution: days of admission and post-discharge complications (headache, motor deficits, epileptic seizures and school performance together with a regulated neurological examination).

Statistical analysis

The homogeneity of demographic variables, medical history and other clinical parameters were analyzed. The description is made by the mean, median, standard deviation of the quantitative variables as well as with the absolute frequency and relative frequency of the qualitative variables. For the quantitative variables, *t* Student tests are developed in case of the assumption of normality and nonparametric tests of Mann-Whitney *U*, otherwise. The qualitative variables are analyzed by homogeneity tests based on the χ^2 distribution when the expected values make it possible and by Fisher's exact test otherwise. The clinical variables are correlated with the evolution of the patient. The data is analyzed with the SPSS program version 20.0.

Results

Twelve children were included in the period analyzed (86.832 admissions). They were between 1 and 13 years old, with a median of 4.5 years. Seven patients were male. The symptoms and signs are summarized in the table.

Personal history, reason for consultation, clinical and analytical findings upon admission

In one patient, there was an underlying disease (IgA deficit). The pneumococcal vaccine had been administered on 8/12. The median number of days up to the consultation was 6 (range from 1 to 25). The reasons for consultation were: fever 11/12, vomiting 9/12 and headache 8/12. The most frequent clinical findings were ophthalmoplegia caused

by paralysis of the sixth nerve (4/12), acute otitis media (AOM) (7/12) and ptosis due to paralysis of the III pair in two children (2/12).

In our series, 10 had mastoiditis and 2 sinusitis in ED. In those with mastoiditis, all had a recent history of AOM and 5/10 had received adequate antibiotic therapy for at least 7 days. Of the children who presented mastoiditis as an infectious focus, only two of them presented external signs of mastoiditis, the diagnosis of which was made by CT.

At hospital admission, leukocytosis was observed (median of 12,350/μL, range of 5,850/μL to 23,820/μL). The CRP at admission showed a median of 8.9 g/dL (range of 0.02 to 20.5 g/dL) and the PCT of 0.27 μg/dL (range 0.02 to 36 μg/mL). As for the leukocytosis figure at admission, it showed a median of 12,350/μl (range of 5,850/μl to 23,820/μl). Only two patients of 2 and 4 years old had anemia with hemoglobin of 10 and 9.8 g/dL respectively.

A lumbar puncture was performed in 7 patients, being pathological in 5 (1 bacterial meningitis, 2 pleocytosis without signs of infection, 2 increases in CSF pressure). All cultures were sterile.

Blood culture was extracted in all patients, being positive in 2 (*S. pneumoniae* and *S. intermedius*). Of the 11 cases in which surgery was performed, positive microbiological isolation was obtained in 4 (*S. pneumoniae, intermedius,* and *constellatus,* and *Staphylococcus hominis*).

A coagulation study was performed in 8 patients: 4 had elevation of coagulant VIII factor (up to a value of 174%) and 2 of them were positive for lupus anticoagulant. Both findings were normalized during follow-up. In one of the patients, a prothrombotic mutation was found in the prothrombin 20210A gene and in another Leiden factor V (Table).

Imaging tests

Brain CT and brain MRI were done to all cases. The CT was diagnostic in 9/12 and the MRI in 12/12. Mastoid occupation was observed in the three cases with not diagnostic CT. In one of them intracranial hypertension was also seen. About cases with sinusitis, one had sagittal sinus thrombosis and the other cavernous sinus thrombosis. Ten patients presented thrombosis of the sigmoid sinus, 5/12 also involvement of the transverse sinus and 3/12 jugular involvement.

The complications added to the thrombosis were: 2/12 epidural abscesses, 1/12 subperiosteal abscess, 1/12 cerebellar abscess and 1/12 erosion of the cortical bone. In case of image complications there were no significant differences in the appearance of neurological symptoms (p = 1.08 in Fisher's test) or time of admission (p = 0.6). In turn, in complicated cases it was observed a tendency to more extended time to diagnosis (10.6 ± 11.1 days on average in complicated VS 6.71 ± 3.3 days in uncomplicated).

Therapies

All the patients received wide-spectrum intravenous antibiotics. Antibiotherapy lasted a median of 21 days (range 10-42 days). Treatment with low-molecular-weight heparin was administered in 11 patients a median of 26 days (range 12-90). In 4 patients the anticoagulant treatment with oral acenocoumarol was completed until a total of 90 days. Seven patients received corticosteroids. There were secondary complications or worsening of the neurological symptoms in 5/7 of the cases in which they were administered. In 2 of them corticosteroids were initiated until negative CSF microbiology. Eleven patients required surgical intervention (Table).

Clinical course

The median of days of admission was 34 (range 10-81). No patient presented surgical complications. Four patients completed antibiotherapy at home orally. There were no sequelae of any kind in the patients in the clinical and radiological follow-up after hospital discharge. When coagulation alterations were observed, they disappeared in later controls.

Discussion

We describe a cohort of children with septic thrombosis of intracranial venous sinuses (STVS). As far as we know, our paper describes one of the largest pediatric series. We observed that at emergency department fever, vomiting and headache were the most frequent findings. Also, in more than half of cases neurological symptoms were observed. Mastoiditis was the principal previous diagnosis at ED and the MRI always confirmed the STVS. The treatment was based on broad-spectrum antibiotics, surgery and anticoagulation. We did not observe acute or late complications.

In classical series the STVS has been described as more prevalence in males [5,6]. In our paper 7/12 cases were male. As previously said a high in-

Table. Summary of patient's characteristics.

	Age in years at dx	Previous ATB	Symptoms	Focus	Image studies	Associated complications	LP	Cultures	Surgery
1	1	4 d amoxi	Fever, vomiting, mastoiditis signs	M	Dx; CT M + SS	Subperiosteal abscess	No	BC - IFC S. *Pneumoniae*	TTD + Mast
2	9	No atb	Fever, headache, vomiting	S	Dx: CT Sinusitis + SS	Epidural empyema	RM	BC S.*intermedius* IFC S.*intermedius*	Empyema drainage
3	13	no atb	Fever, headache, vertigo, papilledema	M	Dx: MRI M + SS+JV	Epidural empyema	No	BC - IFC S.*costellatus*	TTD+ Mast.
4	6	No atb	Fever, vomiting, meningeal signs, papilledema	M	Dx: CT M+ T+SS		RM	BC - IFC NR	TTD+ Mast
5	5	7 days amoxi	Fever, headache, vomiting, VI palsy	M	Dx: CT Mast SS+T+JV		Nor- mal	BC - IFC S.*hominis*	TTD+ Mast
6	2	8 days amoxi	Walk deviation, VI palsy, papilledema, dysmetria	M	Dx: CT Mast+ T+SS		No	BC - IFC S.*pneumoniae*	TTD+ Mast
7	2	No atb	Fever, vomiting, III palsy, papilledema	M	Dx:MRI M+CS		BM	BC - IFC NR	TTD
8	12	No atb	Fever, Vomiting Headache III, VI palsy	S	Dx: CT Sinusitis CS		No	BC S.*pneumoniae* IFC NR	NR
9	4	4d amoxi	Fever, headache, vomiting, VI palsy	M	Dx: CT M SS+T+JV		IH	BC - IFC -	TTD
10	1	16d cefuro- xime	Fever, repeated mastoditis	M	Dx: CT M +SS	Cortical erosion	No	BC - IFC -	TTD
11	11	10d a/clav	Fever, headache, vomiting	M	Dx: CT M +SS		No	BC - IFC -	TTD
12	4	14d a/clav	Fever, seizures, headache, vomiting	M	Dx: MRI M+ T+SS	Cerebellar abscess	Normal	BC - IFC -	TTD Empyema drainage

ACL: lupus anticoagulants; amoxi: amoxicillin; a/clav: amoxicillin/clavulanic; ATB: antibiotic; BC: blood culture; BM bacterial meningitis; Cef: cefotaxime; Clinda: clindamycine; CS: cavernous sinus; CT: computerized tomography; d: days; Dx: diagnosis; H: heparin; IFC: infectious focus culture; IH: intracranial hypertension; JV: jugular vein; LP: lumbar puncture; M: mastoiditis; mast: mastoidectomy; Metro: metronidazole; MRI: magnetic resonance image; NR: non-realized; RM: reactive meningitis; OAC: oral anticoagulants; SS: sigmoid sinus; T: transverse sinus; TTD: transtympanic drainage; U: unknown; Van: vancomycin.

dex of suspicion is essential in STVS diagnosis. We observed, with no statistical signification, a longer time to diagnosis in complicated cases. Our cases commonly showed fever accompanied by headache and vomiting, which have been previously described by other authors [7]. Also, almost half of cases presented some neurological symptom (7/12). About the thrombosis cause, the absence of signs of acute otomastoiditis in physical exam did not discard otic origin [8,9]. Thus, only in 2/10 patients with otomastoiditis had external signs. Collectively these findings suggest that classical clinical triad may not be present [7,10,11].

Related to blood complementary tests the leukocytes median value was lower than the value usually considered as leukocytosis. Also, there were only

ATB	Corticotherapy	1. Anticoagulation 2. Coagulation study
Cef + Van 30d	No	1. H 90d 2. Normal
Cef + Van +metro ,42d	No	1. H 90d 2. U
Cef + Van +metro, 42d	No	1. H 40d 2. U
Cef + Van , 35d	Yes	1. H 21d 2. U
Cef + Clinda, 30d	Yes	1. H+OAC 90d 2. VIII + Factor V Leiden
Cef + Clinda, 30d	Yes	1. H+OAC 90d 2. VIII+ACL + Mut20210A
Cef + Clinda, 42d	No	1. H+OAC 90d 2. VIII+ACL
Cef + Van +metro, 21d	Yes	1. H+ACO 100d 2. VIII
Cef+ Metro, 35d	No	1. NR 2. Normal
Cef, 25d	Yes	1. H 90d 2. U
Cef + Van +metro, 30d	Yes	1. H 90d 2. U
Cef + Van +metro, 30d	Yes	1. H 90 d 2. U

two positive blood cultures despite it has been described a 70% positivity in the literature [10]. This could be related to a low volume of blood extracted and previous antibiotherapy administration [12]. It has been described that CSF analysis is helpful to diagnose the thrombus septic origin or to rule out an associated bacterial meningitis [10]. In previous reports up to 85% of CSF are described as pathological, with 55% being reactive meningitis and 30% bacterial meningitis. Besides, CSF culture is described as positive in almost 20% of cases [7,10]. We observed pathological findings in 5/12 cases but the CSF culture did not show bacterial growth in any case. A precocious antibiotherapy administration could affect this observation. As can be observed, these findings did not change the empiric antibiotic treatment done.

Related to coagulation, Sébire G, et al [13] and Javed et al [11] describe the presence of microcytic anemia as a thrombosis risk factor [11,13]. In our study, it was observed in two patients. In STVS cases a hypercoagulability study is always recommended [14]. Prothrombotic alterations are described in one out of every three patients [13]. These observations are in most cases secondary to the infection and will later normalize. As can be seen in the table surprisingly we found four cases with coagulation-related disorders that required subsequent follow-up.

As said, the imaging tests confirm the STVS diagnosis. The CT scan may show hyperdense areas in deep veins, signs of mastoiditis or sinusitis, bone erosion, and sometimes the sign of '*empty deltha*'. The MRI is considered as the gold standard diagnosis and is essential to assess the vascular system and discard intracranial complications [5,6,8,10,15]. In our cases we did not found concordance between the image and the clinical findings. Also, there was no alteration in the contralateral venous flow in any child. The absence of this, linked to a worse prognosis, may explain the good evolution of all our cases [6,8].

The STVS treatment is heterogeneous. Firstly, the empiric broad-spectrum antibiotherapy should be early initiated [16,17]. This treatment should last at least 21 days, intravenously initiated and later orally. The most frequently isolated pathogens are *Staphylococcus* spp., *Streptococcus* spp. and anaerobes [8]. Therefore, in our work the most common antibiotics were vancomycin, cephalosporins and metronidazole. In immunosuppression cases antifungal drugs must be considered [4]. As a complement, the primary infection drainage may be necessary in case of mastoiditis, dental infections, brain abscess or subdural empyemas [18]. In our series, myringotomy and placement of transtympanic drainage were performed in all patients with mastoiditis. Mastoidectomy was done only in those with complicated neuroimaging or complicated clinical symptoms.

The use of corticosteroids is controversial and its use is associated with better functional recovery of

the cranial nerves [7,16]. In our series, corticosteroids were used in seven patients. The influence on their evolution cannot be inferred by our study. It is retrospective and we do not have a control group. About anticoagulant agents seem to be associated with a decreased thrombus propagation and may improve neurological prognosis. Nowadays, there are not based evidence data about the optimal duration of this treatment [19-22], it is usually maintained between four and six weeks [6,7]. In this study 10/12 patients received anticoagulant without presenting hemorrhagic complications.

Finally, we did not observe sequelae in any patient. This may be related to early treatment and the absence of injuries seen by the image test. Probably, as said, this was a selection bias of good prognosis cases. Given the small number of cases, we should not draw conclusions or grant external validity [23,24] .

This study presents significant limitations. It comprises a small number of patients, it is unicentric and retrospective. There was no diagnostic or therapeutic protocol established which favors the heterogeneity of clinical attitudes. It was not either possible to recover the exact moment of realization of the complementary tests and starting time of antibiotherapy. Knowing their relationship would allow to better define the precocious nature of the treatment and to explain the negativity of the cultures carried out. The different therapeutic approaches cannot be compared or valued based on their effect on the evolution of cases. There is no control group. The retrospective nature of the study precludes ensuring the correct record of the variables which has negatively influenced its quality. At the same time, the data related to the evolution of patients are heterogeneous and do not allow a longer-term follow-up.

In conclusion, and according to our series, STVS is a rare complication that should be suspected when there is a previous history of otitis or sinusitis. The presence of headache, vomiting and fever in ED could help to its diagnosis. The cerebral MRI is useful to get to the definitive diagnosis and a CT scan may be performed if the MRI is not available. Early treatment with broad-spectrum antibiotic therapy should be started and anticoagulant treatment should be considered. At the same time, surgical treatment will be assessed individually. Given the low prevalence of STVS, it would be of great interest to carry out multicenter studies that, by grouping cases, could allow a better clinical and evolutionary description of these patients.

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Trombosis séptica pediátrica de senos venosos intracraneales: del diagnóstico al alta. Veinte años de experiencia

Introducción. La trombosis séptica de los senos venosos intracraneales (TSSV) es una complicación rara y grave de las infecciones craneales.

Materiales y métodos. El objetivo principal de este trabajo es describir los datos clínicos, procedimientos diagnósticos, tratamiento y evolución de una serie de casos de TSSV. Además, se revisa la bibliografía actual. Es un estudio retrospectivo observacional mediante revisión de historias médicas (enero de 1995-diciembre de 2016). Los datos recogidos fueron: clínicos, analíticos, epidemiológicos, microbiológicos, radiológicos, de manejo y de seguimiento. Se realizó un análisis descriptivo y estadístico de los datos.

Resultados. Se incluyó a 12 niños (86.832 ingresos estudiados). La mediana de edad fue de 4,5 años (rango: 1-13), con un tiempo medio de síntomas de 6 días (rango: 1-25). En el momento de la admisión, los datos clínicos fueron: fiebre (11/12), vómitos (9/12) y dolor de cabeza (8/12). También mostraron mal estado general, 12/12; otitis media aguda, 7/12; y paresia del VI par craneal, 5/12. La punción lumbar fue patológica en 4/12. El microorganismo más frecuentemente aislado fue *Streptococcus* spp. Se confirmaron mutaciones protrombóticas en 2/12. La tomografía computarizada craneal permitió el diagnóstico en 9/12; la resonancia magnética lo logró en 12/12. Los signos neurológicos anteriores o el tiempo de diagnóstico no influyeron en la aparición de otras complicaciones de la imagen. Recibieron tratamiento antibiótico 12/12; heparina, 10/12; y cirugía, 11/12. No hubo secuelas.

Conclusión. En nuestra serie, la otitis, el dolor de cabeza, los vómitos y la fiebre fueron frecuentes. Las pruebas complementarias permitieron el diagnóstico de sospecha, pero el diagnóstico definitivo se obtuvo por neuroimagen. No hubo secuelas y las terapias fueron principalmente antibióticos de amplio espectro, heparina y cirugía.

Palabras clave. Heparina. Niños. Otomastoiditis. Seno venoso cerebral. Sinusitis. Trombosis séptica.