

CASE REPORT

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Meningitis and subdural empyema caused by group A streptococcal infection

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Abstract

Background Group A streptococcus (GAS) could lead to various disease types in children, but central nervous system (CNS) infections are uncommon. In this paper, we analyzed the clinical features of a GAS case with meningitis and subdural empyema, and characterized the GAS clone.

Case presentation A thirteen-year-old boy complained of fever, headache, and left hemiplegia. Physical examination also showed central facial palsy of left side. The examinations of blood and cloudy cerebrospinal fluid (CSF) showed bacterial meningitis. Blood cultures and metagenomic sequencing (mNGS) of CSF showed GAS, and GAS antigen of throat swab was positive. The first anti-streptolysin (ASO) was negative, but increased obviously after 2 weeks. The examination of emm type showed emm 12.0 isolate. The head MRI showed restricted diffusion in the right frontal lobe, subdural empyema in the right side of cerebral falx, and meningitis. The CT revealed rhinosinusitis and mastoiditis. Bacterial meningitis, subdural empyema, sepsis, and sinusitis were diagnosed, and vancomycin and ceftriaxone were given. The patient also received dexamethasone in the beginning. Gradual improvement was seen in the patient's clinical status, laboratory parameters (blood/CSF), and radiographic manifestations.

Conclusions The contiguous spread from rhinosinusitis could lead to meningitis and intracranial abscess in adolescent. GAS infection could be the pathogen for subdural empyema in patients with an abrupt onset of symptoms and rapidly deteriorating clinical course.

Keywords Group A streptococcus (GAS), Meningitis, Subdural empyema, Central nervous system (CNS), Sinusitis

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Background

Group A streptococcus (GAS) causes a number of disease manifestations in children, and superficial skin infections and pharyngitis are frequently seen [1]. Invasive GAS infections (iGAS) occur when the organism invades a normally sterile site in the body, including bacteremia, necrotizing fasciitis, empyema, pneumonia, meningitis, osteomyelitis, septic arthritis, and et al. [2]. GAS infections in central nervous system (CNS) are uncommon but often severe, and intensive monitor during hospitalization are required to reduce morbidity and mortality [3, 4]. We present a case of iGAS with sinusitis, bacteremia, meningitis, and subdural empyema, analyzed the clinical features, and characterized the GAS clone.

Case presentation

A thirteen-year-old boy was admitted complaining of fever for 8 days, headache for 4 days, and left hemiplegia for 2 days in January 2024. The patient suffered from cough, nasal obstruction, and running nose at the beginning. The patient had no medical history of rhinosinusitis/mastoiditis/congenital heart disease, no past history of head trauma/neurosurgery, and no family history of immunodeficiency. Physical examination showed normal pupillary reflex, central facial palsy of left side, stiff neck, obviously decreased muscle strength (Grade 0–1) of left side, and positive Babinski sign of left side. Blood tests revealed increased white blood cells (WBC, $17.59 \times 10^9/L$), C-reactive protein (CRP, >180 mg/L), and alanine transaminase (ALT, 65.2U/L). The immune function evaluation revealed normal lymphocyte subsets, serum immunoglobulin levels, and complement levels. Meantime, hyponatremia (128.6mmol/L)

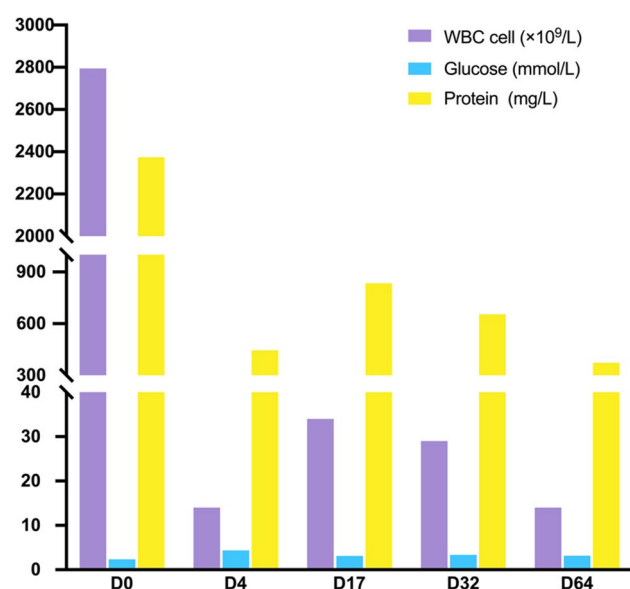


Fig. 1 The longitudinal changes of key CSF parameters. CSF, cerebrospinal fluid; WBC, white blood cell

and hypoalbuminemia (26.2 g/L) were also shown. The examinations of cloudy cerebrospinal fluid (CSF) showed increased WBC ($2794 \times 10^6/L$) and protein level (2374 mg/L), and decreased glucose level (2.36mmol/L, Fig. 1). The head MRI showed restricted diffusion in the right frontal lobe, subdural effusion and empyema in the right frontal/parietal/temporal part and right side of cerebral falx, and meningitis (Fig. 2A-C). The CT revealed sinusitis and mild mastoiditis (Fig. 2D-F). Bacterial meningitis and sinusitis were first diagnosed, and vancomycin and ceftriaxone were given. Mannitol and dexamethasone (5 mg q6h, for 4 days) were also prescribed.

Ten hours later, double blood cultures were both positive, and further tests showed GAS (clindamycin resistant, erythromycin resistant, Table 1). We tested emm type of the bacterium, and found emm 12.0 isolate. Metagenomic sequencing (mNGS) of CSF also showed GAS (reads 7, relative abundance 5.1%, no other suspicious pathogens detected), however, bacterial culture of CSF was negative. The GAS antigen of throat swab was positive. The first anti-streptolysin (ASO) was negative, but 2 weeks later, ASO was 5890IU/ml. Through treatment, the temperature was nearly normal on the second day. The blood inflammatory markers and CSF parameters improved significantly on the fourth day. Bacterial cultures of blood became negative after 3 days. On the fifth day, vancomycin was stopped based on drug susceptibility test (Table 1). The muscle strength of left side became normal (Grade 5) after 1 week, and facial movement also recovered. Brain MRI revealed localized subdural empyema in right frontal part and right side of cerebral falx, and obvious meningeal enhancement in the 17th day (Fig. 3A-C). Symptomatic treatment was stopped gradually. The otolaryngology team assessed the patient and determined no surgical indication given the significant clinical improvement. Neurosurgeons were consulted and declined intervention due to an insufficient volume of collection for safe drainage. During the therapeutic process, there was slight fluctuation of some CSF parameters (Fig. 1), but the conditions gradually improved. The intracranial lesions became smaller by brain MRI (Fig. 3D-F).

On the 33rd day, this patient was discharged from hospital, and was given oral linezolid and drugs for rhinosinusitis. On the 64th day, the CSF indicators became normal, and brain lesions regressed obviously. On the 95th day, there was no obvious abnormal findings by MRI, and linezolid was stopped. This patient was during regular follow-up, and no abnormal manifestations were found.

Discussion and conclusions

Intracranial GAS infection in children was uncommon, only 3.5% children with iGAS had intracranial infection

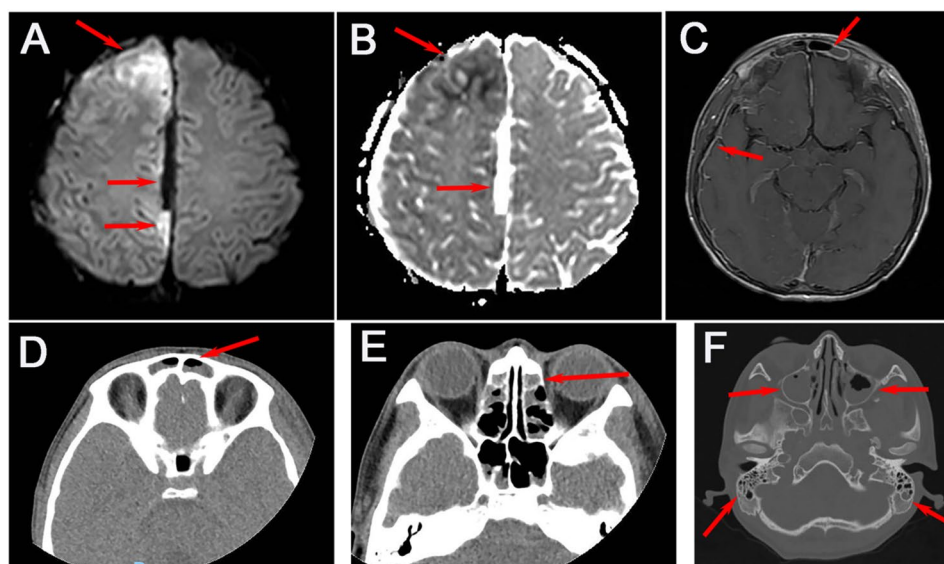


Fig. 2 The imaging manifestations of brain and ear/nose on admission. **A** (DWI) and **B** (ADC) showed restricted diffusion in the brain parenchyma of the right frontal pole, subdural effusion in the right frontal part and right side of cerebral falx, and subdural empyema in the right side of posterior falx (posterior arrow). **C** (enhanced T1) showed pachymeningeal enhancement of the right cerebral hemisphere and frontal sinusitis. **D**, **E**, and **F** showed frontal sinusitis, ethmoidal sinusitis, sphenoidal sinusitis, and mild mastoiditis by CT

Table 1 The antimicrobial susceptibility using Kirby-Bauer Disk Diffusion Susceptibility Test according to CLSI guidelines

Drugs	KB test	Results	Cut-off value
Penicillin	34	S	$S \geq 24$
Ceftriaxone	34	S	$S \geq 24$
Cefepime	36	S	$S \geq 24$
Erythromycin	6	R	$S \geq 21, R \leq 15$
Clindamycin	6	R	$S \geq 19, R \leq 15$
Vancomycin	21	S	$S \geq 17$
Linezolid	26	S	$S \geq 21$
Levofloxacin	24	S	$S \geq 17, R \leq 13$

[5]. According to an American study, intracranial GAS infections were most frequent among children aged < 1 year [5]. However, 83 adult GAS meningitis (16 years old or older) were revealed in a cohort study in 2024 [6]. In this paper, we presented an adolescent boy with meningitis and subdural empyema caused by GAS, and analyzed the clone, which help deepen the understanding of clinical features of intracranial GAS infections.

Subdural effusion was a relatively rare complication of community-acquired bacterial meningitis (3%), however, the proportion of subdural empyema in GAS meningitis was higher (22%) [6, 7]. Otitis (75%) and sinusitis (29%) lesions are common abnormalities in GAS meningitis [6]. Other studies also showed that contiguous infection in the middle ear or sinuses was one of the risk factors for intracranial GAS infections [3, 5]. In clinical practice, many infections caused by GAS were not differentiated from those caused by other pyogenic agents, which might favor underdiagnosis of the agent. However, it is worth noting that its high virulence should be considered

a potential etiological agent when in the presence of abscesses, notably in the head, neck, and abdomen, or when isolated in sterile sites [8]. Our patient suffered from bacteremia and sinusitis, however, we thought intracranial infections resulted from sinusitis, and not from blood dissemination. This thought was based on the consideration that the possibility of infection from blood to CNS was small, now that this patient was an adolescent boy whose development of blood-brain barrier was relatively perfect.

GAS emm type 1 (31%) and 12 (13%) were most common in available isolates among intracranial GAS infections in US children from 1997 to 2014 [5]. However, a study on community-acquired GAS meningitis in the Netherlands showed that the proportion of emm 1 isolates significantly increased in 2022–2023 (90%) compared with 2006–2021 (33%) [6]. In fact, emm 12 was a prevalent epidemic isolate of GAS in China, more than emm 1 strains in most years [9]. Our study showed that emm 12 could also lead to serious neurological lesions.

Antibiotics treatment was very important for patients with intracranial GAS infections, however, the evidence for the treatment duration was absent [3, 5]. The treatment duration of our patient was mainly based on the improvement of clinical symptoms, normalization of laboratory values, and resolution of radiographic abnormalities [10]. Additionally, surgical intervention to drain the intracranial abscess and the infected sinusitis also constitutes a vital component of the management strategy when feasible. Clindamycin resistance in GAS isolates increased gradually in US

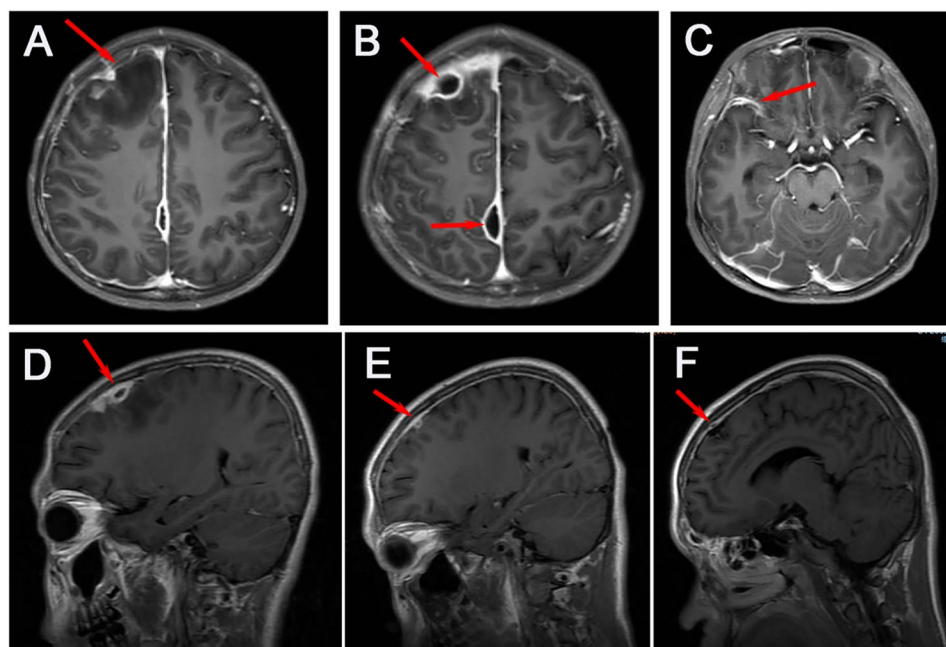


Fig. 3 The changes of intracranial lesions by enhanced MRI. **A-C** revealed pachymeningeal and parenchymal enhancement of right frontal lobe (**A**), localized subdural empyema of the right frontal part (12 mm×8.5 mm) and right side of cerebral falx (15 mm×6.5 mm, **B**), and obvious meningeal enhancement (**C**) in the 17th day. **D-F** revealed the improvement of intracranial lesions in the 30th day (**D**), 64th day (**E**), and 95th day (**F**)

and China, and this was due to expansion of several emm types [11–13]. The GAS isolate from our patient was resistant to clindamycin and macrolides, which might be related to ermB resistance gene harboured [12].

Our patient received third-generation cephalosporin and obtained good effects. However, the choice of oral therapy upon patient discharge posed a challenge. According to guidelines for bacterial meningitis, linezolid is not listed as a standard therapeutic option for community-acquired GAS meningitis, and only mentioned as an alternative for some CNS infections (*Streptococcus pneumoniae*, *Listeria monocytogenes*, *Staphylococcus aureus*, and *Enterococcus*) [14, 15]. We still chose oral linezolid after discharge mainly based on the fact that linezolid could achieve a satisfactory concentration in CNS compared with other oral antibiotics [16]. It should be noted that the selection of linezolid was pharmacologically reasonable but not evidence-based according to current guidelines, and represented an exception based on pharmacokinetic considerations. Coincidentally, a new study by Zhu et al. revealed that linezolid demonstrated favorable clinical efficacy and tolerability in pediatric CNS infections, with CSF concentrations correlating with plasma levels [17].

In summary, the bacterial meningitis is uncommon in adolescent, and contiguous spread from rhinosinusitis could lead to meningitis and intracranial abscess. GAS infection could be the pathogen for subdural

empyema in the patients with an abrupt onset of symptoms and rapidly deteriorating clinical course. Our patient with sinusitis, bacteremia, meningitis, and subdural empyema was caused by GAS emm type 12, who recovered after prompt and proper treatment. Our study could help the recognition and understanding of iGAS.

Abbreviations

ALT	Alanine transaminase
ASO	Anti-streptolysin
CNS	Central nervous system
CRP	C-reactive protein
CSF	Cerebrospinal fluid
GAS	Group A streptococcus
iGAS	Invasive GAS infections
mNGS	Metagenomic sequencing
WBC	White blood cells

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12887-026-06722-9>.

Supplementary Material 1.

Acknowledgements

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Authors' contributions

HX and YZ collected the clinical data and drafted the manuscript; LZ helped collect the clinical data; MG and KY tested emm type of the bacterium; FD saved the bacterium and finished the drug sensitivity test; XD analyzed the imaging material; GL revised the manuscript and approved the final version.

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Data availability

The raw sequence data reported in this paper have been deposited in the Genome Sequence Archive (Genomics, Proteomics & Bioinformatics 2025) in National Genomics Data Center (Nucleic Acids Res 2025), China National Center for Bioinformation / Beijing Institute of Genomics, Chinese Academy of Sciences (GSA-Human: HRA017044) that are publicly accessible at <https://ngdc.cncb.ac.cn/gsa-human>.

Declarations

Ethics approval and consent to participate

Our study was approved by the Ethics Committee of Beijing Children's Hospital, Capital Medical University, and performed according to the Declaration of Helsinki.

Consent for publication

A written informed consent for publishing this case was obtained from the parents of the patient. All authors have viewed and agreed to the submission.

Competing interests

The authors declare no competing interests.

Clinical trial number

Not applicable.

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