

A rare case of early-onset cerebral syphilitic gummata with severe neurological decline in early-stage syphilis: A case report

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Abstract. Cerebral syphilitic gummata are rare granulomatous lesions associated with tertiary syphilis, typically developing several years after the initial infection. Early-stage syphilis with cerebral gummata and accompanying severe neurological symptoms is rare. Failure to recognize the lesions or delays in diagnosis may lead to marked and potentially irreversible central nervous system injury. In the present study, the case of a 60-year-old man with a 6-month history of syphilis who presented with progressive right-sided limb weakness and dysarthria is reported. Brain magnetic resonance imaging revealed multiple ring-enhancing lesions accompanied by extensive perilesional edema. Due to the severity of the symptoms and a notable mass effect, surgical intervention was performed to remove the gummata lesion, followed by histopathological confirmation of syphilitic granuloma. The patient was subsequently treated with high-dose intravenous penicillin, which led to notable improvement in neurological function and resolution of the symptoms; follow-up imaging demonstrated no recurrence of intracranial lesions. In conclusion, the present case illustrates the importance of early recognition and a multidisciplinary approach in the management of neurosyphilis with cerebral gummata. Increased awareness of this rare presentation may facilitate a timely diagnosis and appropriate treatment in similar clinical scenarios.

Introduction

Syphilis is a sexually transmitted infection caused by the spirochete *Treponema pallidum* (1,2). Despite its long history, syphilis continues to pose a notable public health challenge due to its ability to mimic a wide array of clinical conditions. Neurosyphilis (NS), a severe complication resulting from the invasion of *Treponema pallidum* into the central nervous system, can occur at any stage of syphilis. Historically, up to ~30% of untreated patients with syphilis developed NS; however, with antibiotic treatment, symptomatic NS is now estimated to occur in 1.5-13% of syphilis cases, and cohort data indicate that, among individuals with human immunodeficiency virus (HIV) infection, the incidence of NS is twice that of those without HIV infection (3,4). Cerebral syphilitic gummata (CSG) are rare manifestations typically associated with tertiary syphilis (5). In Cushing's (6) frequently cited case series of 2,203 intracranial tumors published in 1932, only 12 cases (0.5%) were identified as CSG. In a review of intracranial tumors by Grant (7) in 1956, which included 2,326 patients, only 5 cases (0.2%) were diagnosed as CSG. Likewise, Katsura *et al* (8), in a review of all neurosurgical cases in Japan prior to 1959, identified only 12 cases of syphilitic gummata among 3,312 patients, yielding an incidence of 0.4%. Although large-scale retrospective studies from recent years are lacking, the limited number of sporadic case reports published over the past two decades suggests that CSG has become an even rarer disease entity. The gummata are granulomatous lesions that may develop in various organs, including the brain, as a consequence of a chronic inflammatory response to persistent infection (9). Within the central nervous system, these lesions can give rise to diverse neurological symptoms depending on lesion size and location, including headaches, seizures, focal neurological deficits, cognitive impairment and behavioral disturbances. Owing to their rarity and heterogeneous presentation, CSG are frequently misdiagnosed or mistaken for other conditions, such as neoplasms, abscesses or other infectious or inflammatory disorders (10). The present study reports a case of CSG in a 60-year-old male patient who rapidly developed hemiplegia of the right upper and lower limbs over several days. The present case underscores the importance of considering NS in patients presenting with atypical neurological

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Abbreviations: NS, neurosyphilis; CSG, cerebral syphilitic gummata; MRI, magnetic resonance imaging; CSF, cerebrospinal fluid

Key words: CSG, early-stage syphilis, NS, granulomatous lesions, case report

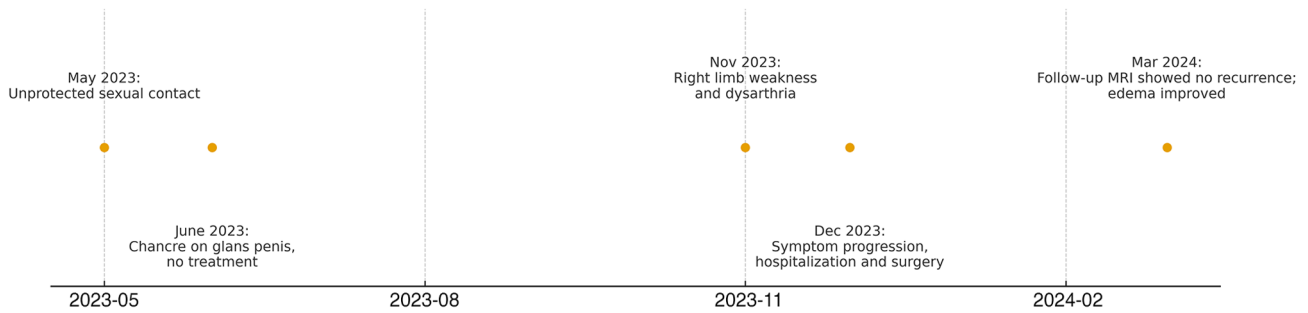


Figure 1. Timeline diagram of the clinical course. MRI, magnetic resonance imaging.

symptoms, particularly those with risk factors for syphilis infection.

Case report

Patient information. A 60-year-old man with a 6-month history of syphilis was admitted to Jiangnan University Medical Center (Wuxi, China) in December 2023 for progressive right-sided limb weakness and slurred speech over a 2-week period. On admission, the patient presented with complete right-sided hemiplegia. The patient's medical history was unremarkable, with no hypertension, diabetes or other chronic illnesses, and the patient was not taking any regular medications and denied any substance abuse. There was no family history of sexually transmitted infections or related conditions.

Because the lesion, which was detected during the present hospitalization on brain magnetic resonance imaging (MRI) as a left frontal parasagittal mass, exhibited a firm consistency during surgical resection, appearing grayish, poorly circumscribed and lacking a clear capsule, which differed from that of typical neoplastic lesions, a review of the patient's medical history was conducted after surgery and revealed that the patient had a single hard chancre, that was 0.5x0.5 cm in size, on the glans penis that appeared ~20 days after a sexual encounter 6 months prior to presentation at the hospital; however, the patient did not receive any treatment at the time. The clinical course is summarized in a timeline diagram (Fig. 1).

Clinical presentation. During the initial examination, the patient demonstrated slurred speech with dysarthria. Neurological assessment revealed right-sided hemiparesis accompanied by mild hyperreflexia. Cranial nerve function was intact, and the remainder of the physical examination was unremarkable. The patient exhibited no signs of meningismus, and the vital signs were stable.

Diagnostic workup. Initial laboratory tests, including complete blood count, basic metabolic panel and liver function tests, were within normal limits. Infectious disease screening showed a signal-to-cutoff ratio value of 26.91 for *Treponema pallidum* antibodies; testing for HIV was negative at the time of admission. Given the neurological deficits of the patient, brain MRI was performed, which demonstrated a nodule in the left frontal lobe with extensive surrounding edema. On contrast-enhanced imaging, the lesion appeared as an abnormally enhancing

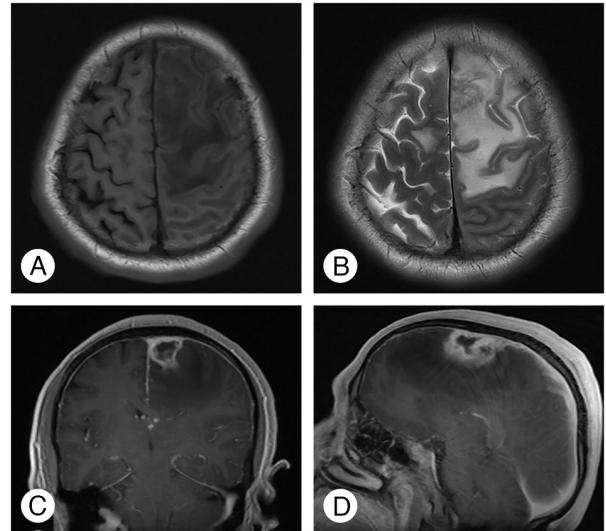


Figure 2. Preoperative brain magnetic resonance imaging scans. (A) Axial T1-weighted image demonstrating a hypointense mass in the left frontal lobe. (B) Axial T2-weighted image showing the left frontal lobe lesion with extensive surrounding hyperintense edema. (C) Contrast-enhanced coronal T1-weighted image revealing prominent ring enhancement of the lesion. (D) Contrast-enhanced sagittal T1-weighted image demonstrating the close relationship between the lesion and the adjacent dura mater.

mass measuring ~18x21x28 mm, involving the adjacent dura mater and the falx cerebri (Fig. 2).

Treatment. Based on the clinical symptoms and imaging findings, a preliminary diagnosis of a left frontal lobe space-occupying lesion was made, with the consideration of a malignant neoplastic lesion or metastatic tumor; however, a high-grade glioma could not be excluded. Consequently, the patient underwent a craniotomy for the resection of the intracranial mass. Intraoperatively, a gray-yellow lesion was observed near the midline of the left frontal lobe, which was markedly adherent to the meninges and falx cerebri; the lesion had a firm texture and lacked a distinct capsule, and swelling of the brain tissue was evident. Under the microscope, dissection was performed along the surrounding edema zone of the lesion until a complete resection was achieved. During the craniotomy, the intracranial pressure was elevated and marked brain bulging was observed; consequently, the local bone flap was removed. The bone flap was not replaced at wound closure, resulting in a left frontal decompressive craniectomy (Fig. 3). During follow-up,

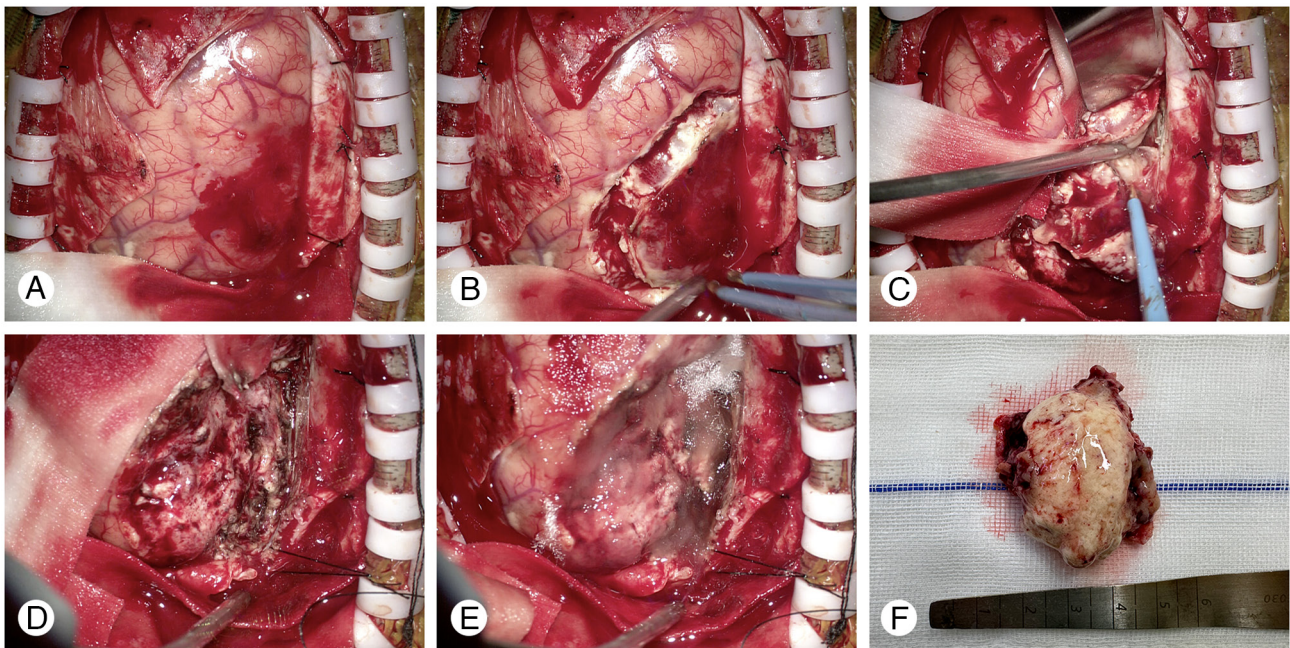


Figure 3. Intraoperative images of the surgical resection of the left frontal lobe lesion. (A) Intraoperative view showing the exposed left frontal lobe lesion without a discernible capsule. (B) Dissection plane along the margins of the lesion during surgical separation. (C) Intraoperative image demonstrating firm adherence of the lesion to the falx cerebri. (D) Intraoperative view confirming complete removal of the lesion. (E) Final operative field showing adequate hemostasis after tumor resection. (F) Gross appearance of the lesion after surgical resection.

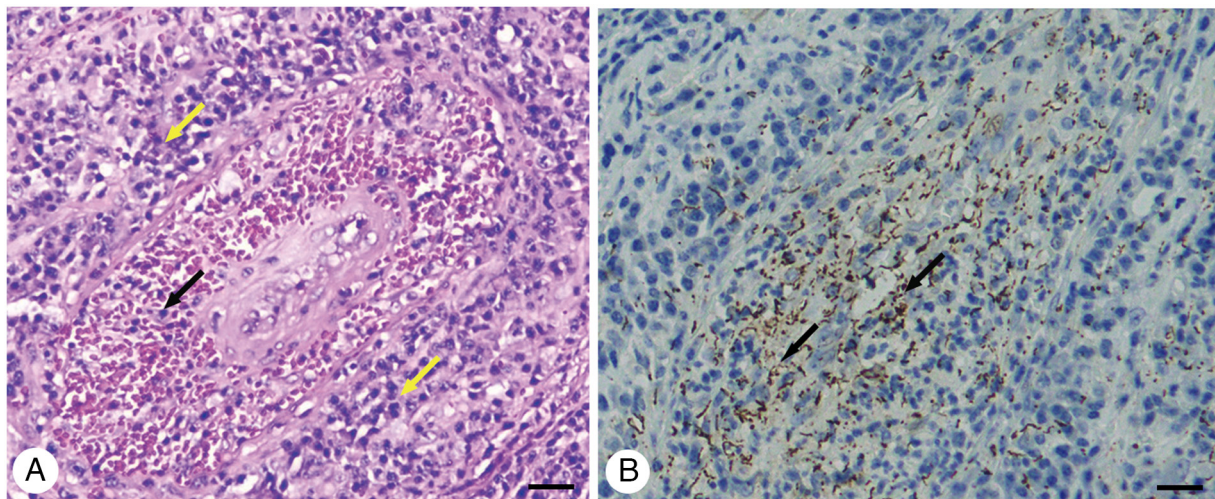


Figure 4. Postoperative pathological images. (A) The lesion consists of dense inflammatory infiltration composed of lymphocytes, plasma cells, granulocytes, histiocytes and fibroblasts. The black arrow indicates features of vasculitis and the yellow arrows show plasma cells surrounding the vasculitis (scale bar, 100 μ m). (B) Immunohistochemical staining with anti-syphilis spirochetes antibodies revealed numerous elongated, curved and spiral shaped brown *Treponema pallidum* on the blood vessel walls; black arrows reveal numerous *Treponema pallidum* (scale bar, 100 μ m).

cerebral edema gradually resolved; however, no secondary cranioplasty was performed because the patient declined cranial reconstruction. Postoperatively, the patient was treated with routine measures such as dehydration therapy to reduce intracranial pressure.

Intraoperatively, it was observed that the lesion was firmer than commonly encountered neoplastic lesions, and histopathological analysis confirmed the diagnosis of a gummatous lesion consistent with tertiary syphilis, showing granulomatous inflammation with central necrosis, surrounded by lymphocytes, plasma cells and macrophages. For histopathological

examination, the resected tissue was fixed in 10% neutral buffered formalin at room temperature for 24 h, embedded in paraffin and sectioned at a thickness of 4 μ m. Sections were stained with H&E using standard protocols at room temperature, with hematoxylin staining for 5 min followed by eosin staining for 2 min. Microscopic evaluation was performed using a light microscope at magnifications of x100 and x400. No evidence of malignancy or other infectious agents was found. Extensive plasma cell infiltration was observed around the small vessels in the brain tissue, leading to the formation of vasculitis.

Table I. Syphilis test results.

Sample	Test	Result	Reference range
Serum	TRUST	1:64	Negative
Serum	Syphilis Ab by TP-PA, S/CO	18.84	<1
Serum	HIV status	Negative	Negative
CSF	TRUST	Negative	Negative
CSF	Syphilis Ab by TP-PA, S/CO	23.30	<1
CSF	WBCs ($\times 10^6/l$)	5	0-8
CSF	Protein, mg/dl	48.2	15-45

CSF, cerebral spinal fluid; TRUST, toluidine red unheated serum test; WBC, white blood cell; S/CO, signal-to-cutoff ratio; TP/PA, *Treponema pallidum* particle agglutination; Ab, antibody; HIV, human immunodeficiency virus.

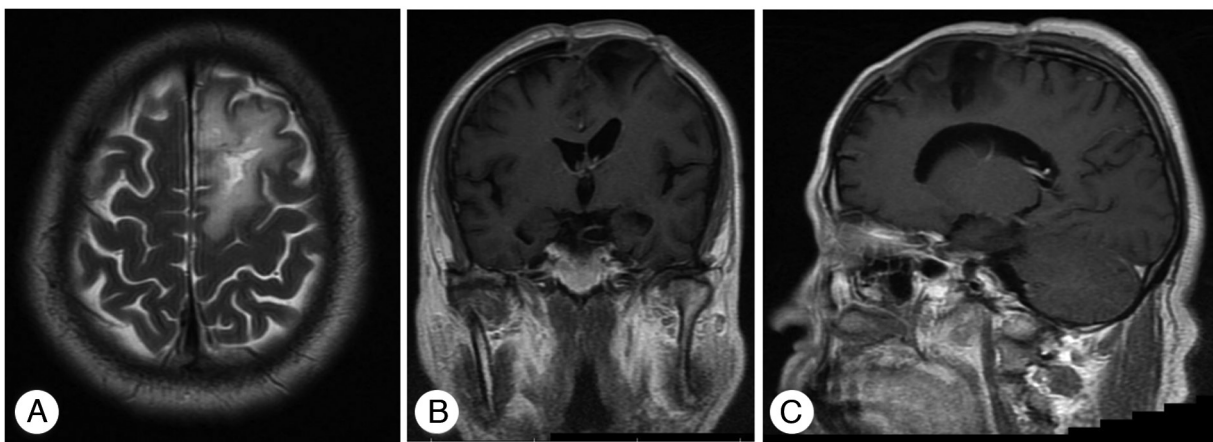


Figure 5. Follow-up magnetic resonance imaging scans 3 months postoperatively. (A) Axial T2-weighted image demonstrating marked reduction of edema in the frontal lobe. (B) Contrast-enhanced coronal T1-weighted image showing no residual contrast-enhancing lesion in the surgical area. (C) Contrast-enhanced sagittal T1-weighted image confirming the absence of contrast-enhancing lesions at the operative site.

For immunohistochemical analysis, the resected tissue was fixed in 10% neutral buffered formalin at room temperature for 24 h, paraffin-embedded and sectioned at a thickness of 4 μ m. Sections were deparaffinized in xylene and rehydrated through a descending ethanol series (100, 95, 85 and 75%), followed by rinsing in PBS (41404ES76; Shanghai Yeasen Biotechnology Co., Ltd.). Antigen retrieval was performed by heating the sections in citrate buffer (pH 6.0) at 95-100°C for 15 min. Endogenous peroxidase activity was quenched by incubation with 3% hydrogen peroxide. The sections were permeabilized with 0.1% Triton X-100 and blocked with 5% normal goat serum (36119ES10; Shanghai Yeasen Biotechnology Co., Ltd.) at room temperature for 30 min. Sections were incubated overnight at 4°C with a commercially available imported primary antibody against *Treponema pallidum* (dilution, 1:200; ZA-0199; Beijing Zhongshan Jinqiao Biotechnology Co., Ltd.). After washing with PBS (41404ES76; Shanghai Yeasen Biotechnology Co., Ltd.), an HRP-conjugated goat anti-rabbit secondary antibody (dilution, 1:500; ab6721; Abcam) was applied at room temperature for 30 min. Chromogenic detection was performed using 3,3'-diaminobenzidine. Immunostained sections were examined and imaged using a light microscope at magnifications of $\times 200$ and $\times 400$. Immunohistochemical staining with

anti-syphilis spirochetes antibodies revealed numerous brown spirochetes (Fig. 4A and B).

Routine cerebrospinal fluid (CSF) testing and biochemical analysis were performed postoperatively. The CSF was clear in appearance, with a normal cell count and a protein concentration of 48.2 mg/dl (reference range, 15-45 mg/dl). Serum and CSF toluidine red unheated serum test (TRUST) and *Treponema pallidum* particle agglutination (TP-PA) tests were performed; the serum TRUST test showed a result of 1:64, whereas the result in the CSF was negative. The syphilis antibody by TP-PA results for both serum (18.84) and CSF (23.30) were markedly higher than the reference range (<1); these results are presented in Table I.

After a definitive pathological diagnosis, the patient was started on high-dose intravenous penicillin G (24 million units per day, administered as 6 million units every 6 h) for 14 days to address the remaining infection in the central nervous system. Corticosteroid therapy was also initiated to reduce postoperative cerebral edema and mitigate the risk of the Jarisch-Herxheimer reaction, a common inflammatory response to the treatment of syphilis. Dexamethasone was administered at a dose of 10 mg/day intravenously for 3 days, followed by gradual tapering over the subsequent 5 days.

Table II. Literature review on early-onset cerebral syphilitic gummata.

First author, year	Age, years	Sex	Onset time, months	Lesion location	Past history	Treatment	Outcome	(Refs.)
Sasaki <i>et al</i> , 2019	47	M	9	Left frontal and temporal lobes	Type 2 diabetes	Penicillin G	No neurological deficits or residual lesions on MRI.	(13)
Zhang <i>et al</i> , 2017	56	M	7	Left parietal lobe	-	Operation and penicillin G	Edema resolved, no lesion recurrence.	(17)
Mu <i>et al</i> , 2024	65	F	12	Multiple intracranial lesions	Liver abscess	Penicillin G	Symptoms resolved, lesion disappeared	(20)
Tsuboi <i>et al</i> , 2016	21	M	5	Left frontal lobe	HIV(+)	Penicillin G	Symptoms improved, lesion shrunk	(21)
Present case	60	M	6	Left frontal lobe	-	Operation and penicillin G	Symptoms improved, no lesion recurrence	-

M; male; F, female; HIV, human immunodeficiency virus; MRI, magnetic resonance imaging.

Outcome and follow-up. The patient recovered well from surgery, with gradual improvement in neurological function. The headaches resolved and there was marked improvement in speech function and right-sided hemiparesis. On the seventh postoperative day, the patient's muscle strength of the right upper and lower limbs reached grade IV (grade II preoperatively), and speech was clear. The patient was discharged on oral amoxicillin (500 mg every 8 h) for an additional 2 weeks. Regular follow-up was arranged with neurology and infectious disease specialists at 1, 3 and 6 months after discharge, including neurological examinations, repeat serological testing for syphilis and follow-up brain MRI to assess lesion resolution. At the 3-month follow-up, the patient remained asymptomatic, with no recurrence of neurological symptoms. The 3-month follow-up MRI showed a marked reduction in the size of the cerebral lesions and surrounding edema, indicating a favorable prognosis (Fig. 5A-C).

Discussion

NS complications occur in 4-10% of untreated or inadequately treated patients with syphilis. The neurological manifestations in advanced NS are diverse (11); early NS often presents without neurological symptoms and primarily affects the meninges and blood vessels, whereas late NS can involve the meninges, blood vessels and brain parenchyma. This progression can lead to conditions such as meningitis, gummas or cerebral infarctions (12-14). CSG is a rare clinical manifestation of NS. In a review of 286 reported cases of NS, Drago *et al* (4) identified only 10 cases of CSG (3.5%). In a more recent cross-sectional study, the incidence of CSG was found to be 1.8% among 397 patients with NS (15); however,

in both large studies, the cases of CSG occurred years or even decades after the initial infection. In the present study, the case is unusual in that the patient developed a gummatous lesion in the brain within just 6 months of being diagnosed with syphilis, representing an atypical and rare presentation of this condition in the early stage of the disease. To the best of our knowledge, this rapid progression is rarely documented in the literature and it underscores the variability in the clinical manifestations of syphilis (16,17).

The radiological appearance of syphilitic gummas frequently mimics other intracranial pathologies. Gliomas, especially high-grade variants, may present with irregular enhancement, marked perilesional edema and a mass effect similar to that observed in gummas. However, gliomas tend to show infiltrative margins and elevated perfusion metrics, whereas gummas more often demonstrate well-circumscribed granulosomatous enhancement with a surrounding inflammatory halo.

Reports have suggested that penicillin treatment can reduce the size of CSG lesions, thereby avoiding the need for surgery (18,19). However, in the present case, the condition of the patient deteriorated rapidly, making it impractical to wait for the time required for penicillin therapy. Therefore, in the present case, the decision to pursue surgical intervention was based on the severity of the patient's symptoms and the need to alleviate the mass effect caused by the gummatous lesion. Surgical removal of the lesion not only provided symptomatic relief but also allowed for histopathological confirmation of the diagnosis, revealing granulosomatous inflammation consistent with syphilitic gummata. Therefore, surgical biopsy plays a particularly notable role when imaging is inconclusive, allowing definitive histopathological confirmation and

exclusion of neoplasia. Decompression may also provide symptomatic relief when rapid deterioration occurs. Nevertheless, once a diagnosis is established, medical therapy alone is often sufficient and unnecessary extensive resections should be avoided (20,21).

The present case is noteworthy as it illustrates that CSG can occur much earlier in the course of syphilis than is typically expected. It emphasizes the importance of considering NS and its atypical manifestations, even in patients with a relatively short history of syphilis. Clinicians should maintain a high degree of suspicion for NS in patients presenting with new-onset neurological symptoms, regardless of the duration since syphilis diagnosis, especially when accompanied by risk factors or inadequate treatment history.

The occurrence of CSG in early syphilis may reflect an aggressive host response or a particularly virulent strain of *Treponema pallidum* (22). To the best of our knowledge, the pathophysiology behind the formation of gummata in such a short time frame is not well understood. Typically, gummatous lesions result from a chronic granulomatous inflammatory response to persistent infection. In the present patient, elevated *Treponema pallidum* antibody titers (signal-to-cutoff ratio, 18.84) and cerebrospinal fluid abnormalities, including increased protein and the positive CSF TP-PA test, suggested that the immune response in the present patient might have been unusually robust or dysregulated; this could be due to genetic predisposition, immune status or other unknown factors (23). As part of the present study, a literature review of reported cases of early-onset syphilitic gummas was conducted, as summarized in Table II. Among the cases, a substantial proportion of patients had comorbid conditions associated with immunosuppression, including diabetes, HIV infection or concurrent systemic infections, which may have contributed to the early development of gummatous lesions. Most lesions involved the frontal or temporal lobes, frequently presented with focal neurological deficits such as hemiparesis or speech disturbances, and were generally treated with a combination of surgical resection and antibiotic therapy. Follow-up data indicated that most patients achieved favorable neurological recovery when treated promptly. This detailed overview underscores the importance of considering underlying immunosuppressive conditions and lesion location when evaluating early-onset intracranial syphilitic gummas.

As syphilis rates continue to rise each year, it becomes increasingly more important to consider CSG, a condition that is often overlooked and occurs less frequently (24). There is no definitive standard for diagnosing CSG; although craniocerebral tumors are more common than syphilis, it is crucial to differentiate patients exhibiting nervous system symptoms and positive serological test results for syphilis from those with CSG. Imaging plays a key role in diagnosing CSG; however, when faced with a rapidly deteriorating patient, surgical intervention is often the most effective approach. For patients experiencing severe cerebral edema, decompressive craniectomy can be a viable option.

In conclusion, the present case highlights an atypical and rare presentation of CSG occurring just 6 months after the initial diagnosis of syphilis, challenging the conventional understanding that such manifestations are predominantly associated with tertiary syphilis after numerous years of

infection. Clinicians should consider NS in the differential diagnosis of unexplained neurological symptoms, regardless of the duration of syphilis, to facilitate a timely and effective intervention.

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Availability of data and materials

The data generated in the present study are included in the figures and/or tables of this article.

Authors' contributions

YJ, SL and YW analyzed clinical data, obtained medical images and advised on patient treatment. YZ performed the pathological diagnosis and contributed to the interpretation of pathological findings. ZC and BL designed the study and performed the surgery. ZC and BL confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

The present case report was prepared in accordance with the CARE guidelines.

Patient consent for publication

Written informed consent was obtained from the patient and their family for the publication of the present case report.

Competing interests

The authors declare that they have no competing interests.

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