



Case Report

Intracranial haemorrhage in late-onset neonatal group B streptococcal disease: A case report



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Received 18 January 2021; revised 23 March 2021; accepted 1 April 2021; Available online 24 April 2021

المخلص

يهدف هذا التقرير إلى تنبيه الأطباء حول النزيف المتأخر داخل المخ باعتباره مظهر نادر لمرض المكورات العنقودية من المجموعة ب لدى حديثي الولادة، وتسلط الضوء على الحاجة إلى إرشادات علاجية فاعلة. نقدم هنا حالة مرض نزيف متأخر داخل المخ ناتج عن مرض المكورات العنقودية من المجموعة ب لدى طفلة حديثة ولادة تبلغ من العمر ١٧ يوماً، وكانت الحالة معقدة مع نزيف ثنائي تحت العنكبوتية أكدته التصوير بالرنين المغناطيسي، من خلال مراجعة الملف الطبي. تأخرت الأعراض لهذه المريضة مع أعراض الحمى والخمول والتشنجات والتأكد الميكروبيولوجي للمكورات العنقودية في مزرعة الدم، إضافة إلى وجود نزيف ثنائي تحت العنكبوتية ونقص التروية الدماغية المنتشر الذي أكدته التصوير بالرنين المغناطيسي للدماغ، يشير إلى حدوث مضاعفات شديدة، مع عدد قليل جداً من الحالات المبلغ عنها من نزيف داخل المخ. وقد أظهرت المتابعة على المدى القصير تأخراً ملحوظاً في النمو.

يعد الفحص المبكر لعدوى المكورات العنقودية من المجموعة ب بين النساء الحوامل أمراً مهماً لمنع الحالات الشديدة من مرض النزيف المتأخر داخل المخ الناتج عن مرض المكورات العنقودية من المجموعة ب. وهناك حاجة إلى مزيد من الأدلة لدعم الصلة الوثيقة بين مرض النزيف المتأخر داخل المخ الناتج عن مرض المكورات العنقودية من المجموعة ب والنزيف داخل المخ.

الكلمات المفتاحية: المكورات العنقودية من المجموعة ب؛ النزيف؛ نقص التروية؛ حديثي الولادة؛ تحت العنكبوتية

Abstract

This report aims to alert clinicians to the possibility of intracerebral haemorrhage as a rare manifestation of late-onset neonatal group B streptococcal (LOGBS) disease. This case also highlights the need for effective treatment guidelines for LOGBS disease. We report a case of LOGBS disease in a 17-day-old full-term female neonate, complicated by bilateral subarachnoid haemorrhage confirmed on magnetic resonance imaging (MRI). The patient presented with fever, lethargy, and convulsions. Microbiological examination confirmed the presence of *Streptococcus agalactiae* in the blood culture. Brain MRI showed bilateral subarachnoid haemorrhage and diffuse cerebral ischaemia, suggesting a severe complication of LOGBS disease. Short-term follow-up of the patient showed marked developmental delay. Early screening for group B streptococcus infection in pregnant women is essential to prevent severe cases of LOGBS disease. Very few cases of intracerebral haemorrhage in LOGBS disease have been reported. Further evidence is required to support a pertinent link between LOGBS disease and intracerebral haemorrhage.

Keywords: Cerebral ischaemia; Group B streptococcus; Haemorrhage; Neonatal; Subarachnoid

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Peer review under responsibility of Taibah University.



Introduction

Streptococcus agalactiae, or group B streptococcus (GBS), is the primary cause of invasive bacterial infections in human neonates, accounting for substantial neonatal mortality and morbidity worldwide, with high rates of neurological sequelae.^{1–5}

Neonatal GBS disease is divided into early-onset (EOGBS) and late-onset (LOGBS) diseases, defined by occurrence at 0–6 days and 7–89 days of life, respectively. The most important risk factor for EOGBS disease is maternal colonisation with GBS, leading to vertical transmission during labour or delivery and exposing neonates to a 1%–3% risk of developing severe GBS disease.⁶ This has led to the establishment of intrapartum antibiotic prophylaxis in GBS-positive women as the standard preventive measure, enabling up to a 90% reduction in the incidence of EOGBS in developed countries.^{6–9} Conversely, LOGBS disease is more frequently encountered among preterm infants and is associated with horizontal transmission (including mother-to-infant) as well as nosocomial and community transmission.^{5,10}

In KSA, epidemiological data regarding neonatal GBS infections are scarce. Available data show an overall incidence rate of 0.51 per 1000 live births, a mortality rate of 3.6% (as shown in a Saudi study from a tertiary care centre), which is lower than the 10% overall worldwide mortality rate, and a maternal GBS colonisation rate that varies between studies, ranging from 13.4%–31.6%.^{11–14} The risk of EOGBS disease is increased with inadequate antenatal care and ruptured membranes and decreased by antibiotics during labour. The clinical picture is mainly represented by sepsis and focal disease, followed by pneumonia and meningitis, all associated with high rates of complications and extended hospital and neonatal intensive care unit stays.¹⁵

Ischaemic cerebrovascular abnormalities are commonly described as a complication of bacterial meningitis, notably in neonatal LOGBS disease^{16–18}; however, intracerebral haemorrhage is an extremely rare manifestation and has been reported in very few cases.^{19,20} This rare manifestation may result from thrombocytopenia, which frequently accompanies neonatal sepsis, and may be aggravated by intravascular thrombosis.²¹

We report a case of LOGBS complicated with bilateral subarachnoid haemorrhage suspected on lumbar puncture, and later confirmed by magnetic resonance imaging (MRI), in association with ischaemic changes.

Case report

The patient was a 17-day-old female neonate with weight of 3 kg after full-term spontaneous vaginal delivery. Birth occurred at another institution. The mother was unbooked, with unknown perinatal history, and had no reports related to prolonged labour or previous siblings with similar conditions. Her GBS status was unknown. The neonate was discharged with her mother in good condition.

She presented to our emergency department at East Jeddah Hospital with a history of fever for 1 day, associated with lethargy and decreased feeding. There was no history of

abnormal movements, and further systemic review was unremarkable.

On examination, the baby was lethargic, with weak crying, and no detectable dysmorphic features. She was febrile with a temperature of 38 °C and had a heart rate of 250 beats/min, blood pressure of 97/51 mmHg, and peripheral oxygen saturation of 96% on room air. Her random blood sugar level was 56 mg/dL. On systemic examination, we found: (1) chest: equal bilateral air entry with no additional sounds; (2) cardiovascular system: capillary refill of 3 s, normal first and second heart sounds, and no murmur; and (3) abdomen: soft and lax, with no organomegaly. Neurological examination revealed an open and flat anterior fontanelle and normal power, tone, and reflexes.

Initial laboratory findings were as follows: white blood cells, $3.8 \times 10^3/\mu\text{L}$ (normal range 6–22); neutrophils, $2 \times 10^3/\mu\text{L}$ (normal range 3–7); lymphocytes, $1.5 \times 10^3/\mu\text{L}$ (normal range 3–9); haemoglobin 18.26 g/dL (normal range 14–22); platelets, $291 \times 10^3/\mu\text{L}$ (normal range 150–350); C-reactive protein (CRP), 1.1 mg/dL (normal value < 1); and total bilirubin, 1.04 mg/dL (normal range 0.2–1.2). The other biochemical parameters were unremarkable. Blood samples were sent to the microbiology laboratory for culture.

Electrocardiography revealed supraventricular tachycardia. Adenosine was administered, and a paediatric cardiologist was consulted, who prescribed a β -blocker.

The patient was admitted for diagnostic and therapeutic management of fever, and a stat dose of ampicillin (200 mg/kg/day, q6 hours) and cefotaxime (50 mg/kg/day, q8 hours) was administered in the emergency department as per hospital protocols, which were later switched to meropenem and vancomycin.

On the second day of admission, the patient's heart rate improved. Echocardiography revealed normal heart function and structure. The patient was intubated via assisted control (A-C) mode with initial settings as pressure, 14/4 mmHg; rate, 30/min; and FIO₂, 40%, with pulse oximetry of 98%, as she had developed multiple attacks of apnoea and desaturation with bradycardia (heart rate of 75 beats/min), poor perfusion, and abnormal movements. She received a loading dose of phenobarbital, followed by maintenance therapy, and the convulsions were controlled. She was evaluated by a paediatric neurologist and started on levetiracetam. Later, her vital signs changed to: blood pressure, 51/27 mmHg (MAP 36); pulse rate, 156 beats/min; SpO₂, 97%; and capillary refill, 3–4 s. A cranial ultrasound was performed, which showed no abnormalities. Repeat laboratory investigations revealed an increase in CRP to 28 mg/dL and a reduction in platelet count to $50 \times 10^3/\mu\text{L}$. The coagulation profile was within the normal range. The infectious disease team was consulted, and lumbar puncture was proposed but not performed at that time because of the patient's critical condition. Vancomycin (15 mg/kg/dose, q8 hours) and meropenem (40 mg/kg/dose, q8 hours) were continued, and amikacin (15 mg/kg/dose, once daily) was introduced. Amphotericin B (3 mg/kg/day, once daily) was also added because of the significant drop in her platelet count.

On the third day of admission, she underwent a brain CT, which showed subdural and subarachnoid haemorrhage (Figure 1); accordingly, the infectious disease team added acyclovir and requested HSV PCR test of the CSF. Blood culture tests on the fourth day showed *Streptococcus*

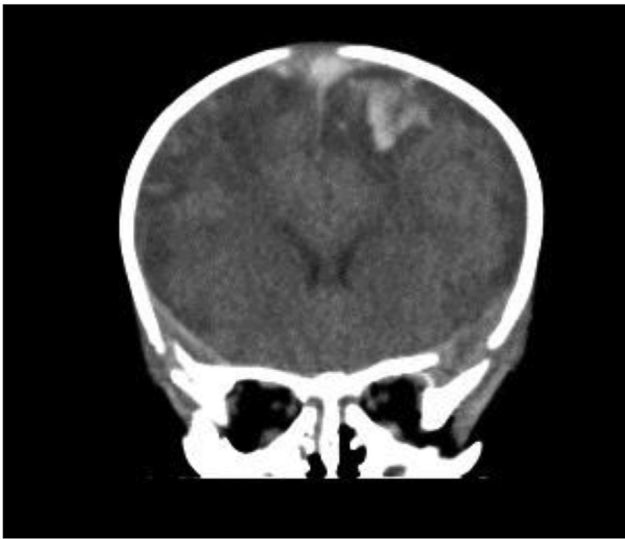


Figure 1: Brain computed tomography (CT scan) shows acute diffuse bilateral extra axial bleeding mostly subarachnoid and to lesser extent subdural, with suspicious of small left parietal lobe intraparenchymal hemorrhage.

agalactiae, which is sensitive to ampicillin. Ampicillin (300 mg/kg/day, q4 hours) was resumed, and vancomycin, meropenem, and amphotericin B were discontinued. The neurosurgeon and the haematology team stated that no surgical intervention was required.

From day 3 to day 7 after hospitalisation, the patient's condition was critical; she required the maximum settings on conventional mechanical ventilation and had respiratory acidosis with pH, 6.83; pCO₂, 144; HCO₃⁻, 12.6 mEq/L; base excess, -10.3 mEq/L; blood pressure, 73/46 mmHg; heart rate, 150 beats/min, ventilator setting pressure, 16/5 mmHg; and ventilator rate (V-R), 60/min. She did not require any inotropes.

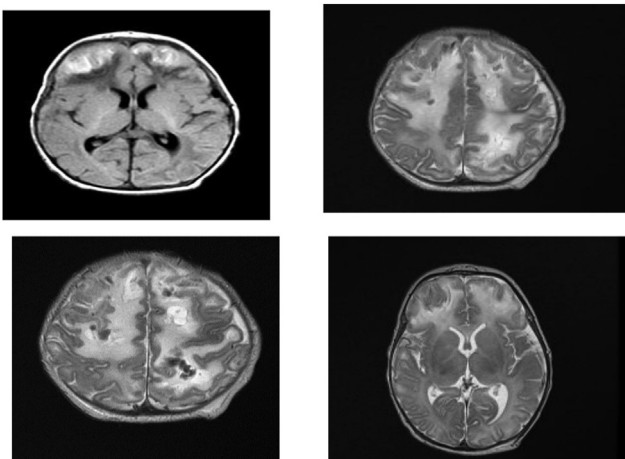


Figure 2: Brain magnetic resonance imaging (MRI) showing moderate diffuse bilateral acute subarachnoid hemorrhage, suggesting acute ischemic changes involving the diffuse brain cortical gray matter, corpus callosum, and bilateral internal capsules.

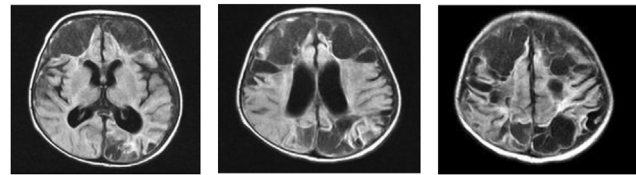


Figure 3: Brain magnetic resonance imaging (MRI) show severe bilateral cerebral hemisphere encephalomalacia.

On day 8 of hospitalisation, at 24 days of age, the patient became more stable and was weaned from mechanical ventilation and shifted to nasal continuous positive airway pressure (NCPAP), positive end expiratory pressure (PEEP) of 4 mmHg, FiO₂ to maintain SpO₂>95%, and a high-flow nasal cannula (HFNC) with 4 L/min oxygen. Her convulsions were controlled. A lumbar puncture was then performed. The cerebrospinal fluid (CSF) had a clear appearance, with a protein level of 175 mg/dL (normal range, 20–80 mg/dL), glucose level of 17 mg/dL (normal range, 60–80 mg/dL), white blood cell count of 53 cells/μL (normal range, 0–5 cells/μL), red blood cell count of 2000 cells/μL, CSF polymorphonuclear leukocyte (PMNL) count of 19 cells/μL, and CSF mononuclear cell count of 81 cells/μL. CSF culture showed no microbial growth at 48 h or 5 days. HSV PCR was not detected in the CSF; accordingly, acyclovir was stopped after a total of 8 days. Amikacin was continued for 7 days and then discontinued. MRI showed moderate-to-diffuse bilateral acute subarachnoid haemorrhage, with no midline shift, as well as possible acute ischaemic changes with bilateral diffuse topography involving the cortical grey matter, corpus callosum, and internal capsules (Figure 2).

Follow-up lumbar puncture was performed at 43 days of age. The CSF was clear in appearance, with a protein level of 71 mg/dL, glucose level of 37 mg/dL, white blood cell count of 59 cells/μL, red blood cell count of 3000 cells/μL, CSF PMNL count of 11 cells/μL, and CSF mononuclear cell count of 89 cells/μL. The CSF culture showed no microbial growth at 48 h or 5 days.

She was on total parenteral nutrition (TPN) and then slowly shifted to oral gastric tube feeding (OGT). Direct breastfeeding was then slowly allowed, as tolerated.

Follow-up brain MRI was performed at 57 days of age and showed severe bilateral hemisphere encephalomalacia and subarachnoid calcification (Figure 3).

The patient was discharged after completing the course of antibiotics for the ventriculitis. She had been administered 6 weeks of ampicillin and 7 days of amikacin, with a total of 49 days of stay in the neonatal intensive care unit (NICU). At the age of 6 months, she was seen at the outpatient department for follow-up. She displayed global developmental delay, and convulsions were controlled on levetiracetam. Currently, she is one and a half years old and able to stand on furniture, creep but with left hemiparesis of the upper and lower limb, and say single words.

Discussion

This paper presents a rare complication of LOGBS meningitis, i.e., subarachnoid haemorrhage combined with

diffuse cerebral ischaemia, which appeared 2 days after the onset of infection symptoms. Neurological symptomatology was predominated by convulsions, which led to further investigations and multidisciplinary diagnostic and therapeutic management. In addition to the uncommon clinical picture, the therapeutic management of the present case was challenging because of the combination of haemorrhagic and ischaemic lesions. Short-term follow-up showed marked developmental delay, and the long-term prognosis was shadowed by a high risk of severe neurological sequelae.

Perinatal infections are major risk factors for acquired prothrombotic disorders in paediatric patients, frequently involving cerebral locations and with arterial vascular locations often presenting as ischaemic stroke.²² Although ischaemic complications of the central nervous system are frequently reported in neonatal LOGBS disease,^{16–18} it has not yet been established whether GBS meningitis exposes patients to a higher risk of cerebrovascular stroke, compared to other GBS diseases. However, arterial vascular occlusions are reportedly the most frequent cerebrovascular complication associated with GBS meningitis. In a series of 14 neonates with cerebrovascular complications of LOGBS meningitis, arterial ischaemic stroke was identified in 10 patients, with a pattern varying from territorial infarction to diffuse ischaemic images.¹⁹ Another paper described intraparenchymal focal infarctions in 10 neonates with LOGBS meningitis and reported two patterns of infarcts in the territory of the deep perforators of the carotid system and superficial cortical lesions, with potential for both localisations to coexist in the same patient.¹⁷ Comparably, in our patient, although no infarct images were evidenced in the acute phase, we observed diffuse and bilateral ischaemic changes involving both superficial cortical regions and subcortical white matter, including the corpus callosum and internal capsules. These images progressed to severe encephalomalacia. Such a pattern of hypoxic-ischaemic lesions is known to be associated with unfavourable outcomes, notably neurodevelopmental impairments.^{23,24} Although the study assessed 26 infants with hypoxic-ischaemic injury (HIE), 6 infants died before completing the assessment at the age of 2 years, 5 infants had moderate to severe cerebral palsy in addition to severe cognitive impairment, and the remaining 15 infants had favourable outcomes.²⁴ Of note, and correlating with the present case, the usefulness of cranial ultrasonography in detecting such lesions is questionable, and normal ultrasound findings should imperatively be completed with MRI.²³

Intracerebral haemorrhagic manifestations of GBS disease are extremely rare in the literature. A similar case was reported in an 8-day-old baby, who presented with febrile respiratory arrest and, subsequently, GBS-positive blood and cerebrospinal fluid cultures. Subarachnoid haemorrhage was observed on computed tomography, which was associated with cerebral swelling. The patient was injected with cryoprecipitate and human immunoglobulin and was also managed with haemopurification and electrolyte balance treatment. The case was further complicated by multiple organ dysfunction and disseminated intravascular coagulation, among other major complications, with repeated cardiac arrest and cerebral prolapse, leading to treatment

discontinuation.²⁰ The other case was meningitis with *Streptococcus pneumoniae* complicated by intracerebral haemorrhage.¹⁹ Consequently, data regarding the appropriate management of such cases are scarce, compromising our ability to establish efficacious treatment guidelines.

The association of stroke injury with haemorrhage in the present case made treatment more complicated as anticoagulants could not be used to treat the thrombosis. Hypothetically, the co-presence of the two complications, brain haemorrhage and ischaemia, may result from the thrombocytopenic state induced by neonatal sepsis, combined with intravascular thrombosis.²¹ In addition, thrombocytopenic complications may be aggravated by disseminated intravascular coagulation.²⁰ In the present case, profound thrombocytopenia developed on the second day of hospitalisation (i.e., 2 days after symptom onset), although the platelet count was normal on the first day of presentation. This indicates the importance of repeated investigations, especially when the patient's state worsens or does not improve.

Study limitations include the utilisation of several antimicrobial therapies because of the critical situation the neonate presented in, as well as the use of ultrasound of the brain being inconclusive and misleading to the physician in the beginning, until CT scan and MRI brain could be performed and blood culture results were found to be positive.

Conclusion

In conclusion, intracranial haemorrhage is a severe but uncommon complication of GBS disease that presents a challenging therapeutic dilemma, especially when associated with cerebrovascular ischaemic disorders. There is insufficient evidence to support a pertinent link between GBS infection and the presence of subarachnoid haemorrhage; other possibilities such as cerebral vascular malformation should be excluded.

Recommendations

Further research is warranted to classify GBS diseases and establish management guidelines for the severe forms—notably, central nervous system localisations with cerebrovascular complications.

Physicians should promote screening for GBS among pregnant women and provide intrapartum antibiotic prophylaxis to prevent severe cases of GBS disease and the consequent impact on the neurodevelopment of the infant.

Source of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interest

The authors have no conflicts of interest to declare.

Ethical approval

Informed consent was signed by the mother of the child for publication of this manuscript, and we obtained approval from the Institutional Review Board (IRB)-Jeddah on 01/12/2020, and registration with KACST: KSA: H-02-J-002/ approval no: A01037.

Authors' contribution

NB, the corresponding author, conceived and provided the data for the case report and submitted the revised manuscript. EF, the first author, collected and organised the data, wrote the initial draft of the article, and performed the initial submission. AB, a co-author, provided logistic support as well as references collections, and MH, a co-author, provided logistic and reference collections. All authors have critically reviewed the case report and approved the final draft and rebuttal response letter and are responsible for the content and the manuscript.

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How to cite this article: Fallata EM, Bokhary NA, Bugshan AS, Hakami MH. Intracranial haemorrhage in late-onset neonatal group B streptococcal disease: A case report. *J Taibah Univ Med Sc* 2021;16(5):771–775.