CASE REPORT

Immunocompetent young man with cerebral abscess and cortical venous infarction mimicking cerebritis caused by *Gemella morbillorum*

Annette Milnik,¹ Angelos Gazis,² Ina Tammer,³ Claudius Bartels¹

¹Department of Neurology, University Medical Center Magdeburg A.ö.R, Magdeburg, Germany

²Department of Neuroradiology, University of Magdeburg, Magdeburg, Germany

³Department of Microbiology, University of Magdeburg, Magdeburg, Germany

Correspondence to

Annette Milnik,
annette.milnik@unibas.ch

SUMMARY

Gemella morbillorum is an anaerobic gram-positive diplococcus and in most cases a harmless commensal, which occasionally causes infections in the central nervous system. We report on an immunocompetent young man with focal neurological symptoms and cephalgia caused by a cerebral abscess. Although successful treatment was done with neurosurgical intervention and antibiotic therapy, he suffered from a venous infarction 5 weeks after first diagnosis, which mimicked cerebritis as an early stage of relapsing abscess. Imaging and investigation of cerebrospinal fluid was necessary for sufficient differential diagnosis and antibiotic therapy could be stopped after altogether 8 weeks of treatment. In summary, *G. morbillorum* causes not only biphasic infections, but also can be accompanied by infarction in the central nervous system despite sufficient antibiotic therapy.

BACKGROUND

Gemella morbillorum is an anaerobic gram-positive diplococcus and in most cases a harmless commensal of the mucosa of the oral cavity, the upper respiratory and the genitourinary tract. Occasionally, it causes infections of the oropharynx (eg, sinusitis, tonsillitis), endocarditis and abscesses in different locations, as well as meningitis or sepsis. A cerebral abscess is a rare but life-threatening and severe infection of the central nervous system (CNS). Clinical features are fever, cephalgia and focal neurological symptoms. Immediate antibiotic therapy should be the consequence of the tentative diagnosis of a cerebral abscess as well as discussing a possible neurosurgical intervention. Antibiotic therapy is necessary over several weeks, hence identifying the causative pathogen and adjustment of antibiotic therapy should be the aim. A rare complication of cerebral abscess can be venous infarction, which can mimic a relapse of the cerebral abscess in the form of cerebritis. Differentiation between cerebritis and venous infarction is essential for correct treatment decision.

CASE PRESENTATION

We report on a 39-year-old man with sudden focal neurological symptoms in terms of numbness and reduced fine motor skills of his left hand and arm. Neurological investigation showed a slight deficiency of fine motor skills and coordination of the left hand but no other abnormalities, also no stiffness of the neck. The patient was awake with normal consciousness and orientation. In addition,

the patient reported about a right occipital cephalgia, which started a few days before the focal neurological symptoms. Our patient did not suffer from nausea, vomiting or diarrhoea. There were no signs of fever (temperature 36.7°C) and no history of a severe infection or injury in the preceding months, but several episodes of sinusitis 1 year before. The patient reported on no further known diseases, especially no known hypertension, diabetes mellitus, seizure or neoplasm. Family history was positive for hypertension.

INVESTIGATIONS

The patient had an elevated blood pressure (200/110 mm Hg), normal heart rate (84/min) and class I obesity (body mass index 31.5). Auscultation, percussion and palpation were inconspicuous; especially, there were no abnormal heart sounds or signs of abdominal pain. Physical examination of the oral cavity, the upper respiratory tract and pharynx, the ear canals, as well as the dermis for possible infectious foci brought altogether only signs of a chronic tonsillitis but no acute infection. Owing to the signs for chronic tonsillitis, we checked for a streptococcal infection. As the antibody titre against streptolysin O was 175 units/ml (normal 100–300 U/ml) an antecedent streptococcal infection could be excluded. There were also no infectious signs or other abnormalities in the chest x-ray. Biochemical and haematological investigations were normal except for an elevated leucocyte count ($11.1 \times 10^9/l$). Especially, the C reactive protein as a second marker for an infection was in normal range. Blood cultures, sampled before administration of antibiotic agents, remained sterile. Search for an HIV infection as a cause of immunodeficiency was also negative. Cerebrospinal fluid (CSF) analysis showed a slightly elevated total protein (82.5 mg/dl) and slightly elevated white blood cells ($4/\mu l$). All other parameters of the CSF were in normal range (clear and colourless, results in normal range for glucose and lactate, gram stain negative, syphilis and borrelia serology negative, no red blood cells) and CSF cultures were sterile. EEG showed only a slight asymmetry (amplitudes over the right hemisphere lower than amplitude over the left hemisphere), but no focus or seizure patterns. EEG had an α -rhythm of 9–10 Hz. There were no abnormalities after activation with hyperventilation and photic stimulation in the EEG.

Initial cerebral MRI (cMRI) (figure 1A) revealed a round cortical lesion measuring 19 mm in the

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right parietal lobe. The lesion showed hyperintensity in T2-weighted images and in diffusion-weighted imaging (DWI) while it was hypointense in T1 and ADC (apparent diffusion coefficient). The margin of the lesion was slightly hyperintense in T1-weighted images and showed gadolinium enhancement

whereas fast low angle-shot sequence showed signal extinction. A T2-hyperintense area measuring maximally 3 cm in diameter surrounded the lesion. These findings were consistent with a haemorrhagic abscess with surrounding oedema. Initially, T2 FLASH-sequence was performed due to our standard protocol

Figure 1 Initial cMRI (A1–A3, week 0) shows a hypointensity of the abscess in the apparent diffusion coefficient (ADC) image (A1, axial image) and the T1 image (A3, coronal contrast-enhanced T1-weighted image), a gadolinium enhancement of the abscess (A3) and a surrounding oedema (A2, axial T2-weighted image; A1). Follow-up cMRI after the second (B1, axial FLAIR image) and the fourth week (C1, coronal contrast-enhanced T1-weighted image; C2, axial FLAIR image) demonstrates a stepwise remodelling of the cavern, but no signs of a new infection. Native cCT after the fifth week (D1 coronar; D2 axial), which was done because of an acute seizure, is inconspicuous. Routine follow-up cMRI after the eighth week (E) shows cortical gadolinium enhancement in a newly discovered ischaemic region (coronal, E1 and E3, and axial, E4, contrast-enhanced T1-weighted images) and the residuum of the abscess (E1, coronal contrast-enhanced T1-weighted image; E2, axial T2-weighted image). CT angiography after the eighth (F1, coronar; F2, axial) and cMRI angiography after the tenth week (G1, coronar; G2, axial) reveal a reduction of the veins in the temporoparietal area on the right side. Follow-up cMRI, 5 months after the first symptoms occurred, shows signs of the expired venous infarction (coronal, H1, and axial, H2, contrast-enhanced T1-weighted images) and a further remodelling of the abscess cavern (H3, coronal contrast-enhanced T1-weighted image; H4, axial T2-weighted image).

to exclude bleedings. This sequence was performed in follow-up measurements as well to control progression, since susceptibility artefacts were detected in the primary performed examination.

Gram stain prepared from smears of the cerebral abscess showed gram-positive cocci arranged in pairs and short chains and polymorphic neutrophils. *G. morbillorum* was identified from anaerobic cultures using API rapid 32A (Fa. Biomerieux, Marcy l'Etoile, France). Antimicrobial susceptibility testing was not performed because of the lack of standardised testing methods and interpretation criteria.

Transoesophageal echocardiography could verify a large patent foramen ovale (PFO, 15 mm) with atrial septum aneurysm and right–left shunt. The right–left shunt could be verified with contrast echocardiography and with transcranial Doppler. An accompanying finding of transoesophageal echocardiography was a slight thickening of the leaflets of the mitral valve as a possible sign for an old inflammation, but no acute signs of inflammation.

Twenty-four-hour blood pressure measurement detected a newly diagnosed hypertension stage 2 (JNC7 category) with a mean blood pressure of 165/110 mm Hg.

DIFFERENTIAL DIAGNOSIS

From a clinical point of view at the beginning, the most likely differential diagnosis were a cerebrovascular event or a focal seizure. Our patient had an increased risk for cerebrovascular events since he had an elevated blood pressure and a class I obesity. In addition, he had a positive family history for elevated blood pressure. After taking the cMRI findings into account, possible differential diagnoses were beneath a cerebral abscess, a tumour or metastasis. The abscess-typical pattern in diffusion imaging of the lesion made the differential diagnosis cerebral abscess from the radiologist's point of view most likely (DWI hyperintense and ADC hypointense as a sign of restricted diffusion due to cellularity and viscosity of purulence; for an overview of the typical MRI sequence patterns of a cerebral abscess see table 1). In general, epidermoid show a similar suppression of diffusion, but morphology and localisation are not typical for an epidermoid. Considering all investigations, the most likely diagnosis was a cerebral abscess.

Typically, the infectious pathway of a cerebral abscess is, for example, from an acute or chronic sinusitis or mastoiditis by continuity to the frontal lobe or cerebellum.¹ In the present case, a haematogenous spread of micro-organisms into the brain was possible because of the large patent foramen ovale with right–left shunt. The parietal lobe is not a typical location for a

Table 2 Typical abscess location depending on the pathogenesis

Pathogenesis	Typical abscess location
Per continuity (50%)	
Frontal sinus, ethmoid sinus, sphenoid sinus, dentogenic	Frontal lobe
Otitis media, mastoiditis, sphenoid sinus	Temporal lobe
Mastoiditis	Cerebellum
Traumatic brain injury, neurosurgery (10%)	Depending on location of the trauma
Haematogenous (25%, eg, endocarditis)	Multiple locations
Cryptic (15%)	None

cerebral abscess, but can be explained in the present case by this unusual infectious pathway (for an overview, see table 2). *G. morbillorum* is able to cause varying infections as, for example, infections of the oropharynx (eg, sinusitis, tonsillitis), endocarditis and abscesses in different locations. However, it is also a harmless commensal, for example, of the mucosa of the oral cavity. Therefore, a possible origin of the infection in the case of our patient could be a trivial mucosa lesion or chronic tonsillitis or sinusitis, which caused bacteraemia.

TREATMENT

Immediately, when the differential diagnosis cerebral abscess was most likely, we started an intravenous broadband triple antibiotic therapy with metronidazole, a third-generation cephalosporin (cefotiam) and an antibiotic potent against *Staphylococcus* (vancomycin), as it is recommended for community-acquired intracranial abscess. Because the location of the abscess was close to the brain surface, neurosurgery with complete resection was possible 1 week after diagnosis. Since the culture of the abscess pus was negative for the *Staphylococcus* species, but identified *G. morbillorum*, we stopped vancomycin but continued intravenous cefotiam and metronidazole. As recommended, we initially did MRI follow-up controls after 1–2 weeks (figure 1B,C), which demonstrated a stepwise remodelling of the former abscess cavern but no signs for a relapse of the infection; therefore, after 4 weeks of intravenous antibiotic treatment, we changed to oral therapy for a further 4 weeks.

To treat the newly diagnosed hypertension, we started a medication with an ACE inhibitor (ramipril). Owing to an insufficient response, we had to add a calcium-channel blocker (amlodipine) later on.

OUTCOME AND FOLLOW-UP

Five weeks after the first diagnosis, the patient suffered from an event with severe sweating, disturbance of consciousness, prolonged hemiparesis of left side and perioral convulsions. Biochemical and haematological investigations of the blood were in normal range. Our patient had no fever, no headache and no other abnormal medical conditions. Cerebral CT (cCT; figure 1D) brought no new aspects. EEG verified a new right parietal-occipital focus. Seizure is the most often seen complication of a cerebral abscess and happens in 30–70% of the cases. Therefore, we immediately started an anticonvulsive therapy with carbamazepine.

Routine cMRI (figure 1E) 3 weeks later demonstrated a further remodelling of the former abscess cavern. However, there was now a new cortical dull hyperintensity in DWI and T2-weighted images in the temporoparietal area with cortical gadolinium enhancement, as is typical for a venous infarction.

Table 1 Typical MRI patterns of a cerebral bacterial abscess

MRI sequence	Feature
T1	Hypointense centre
+Gadolinium	Ring enhancement of the abscess wall
T2	Hyperintense centre
	Hypointense capsule (mature abscess)
DWI (diffusion weighted)	Hyperintense centre (restricted diffusion)
ADC (apparent diffusion coefficient)	Reduced signal (restricted diffusion)
rCBV/PWI (relative cerebral blood volume/perfusion weighted)	Reduced signal

Table 3 Typical causes of infectious venous thrombosis due to venous drainage

Venous thrombosis	Origin and venous drainage
Cavernous sinus	Infectious origin in the face→superior/inferior ophthalmic veins→cavernous sinus Infectious of sphenoid or ethmoid sinuses→small emissary veins→cavernous sinus
Transverse (lateral) sinus/ Sigmoid sinus	Otitis media, mastoiditis→emissary veins→transverse sinuses→sigmoid sinus
Superior sagittal sinus	Bacterial meningitis→meningeal veins→diploic veins→superior sagittal sinus

The formation was also compatible with cerebritis as a hint for relapse of infection. Again, our patient had no fever, no headache and no other abnormal medical conditions including biochemical and haematological investigations of the blood. Owing to the cortical location contiguous to the subarachnoid space, we expected an elevated cell count in the CSF in the case of an infection. This diagnostic finding could not be verified in the CSF (again white blood cell count 4/ μ L, total protein slightly elevated with 63.6 mg/dL, clear and colourless, normal range for glucose and lactate, gram stain negative, no red blood cells, CSF cultures sterile). We added a CT angiography (arterial and venous system), which additionally revealed reduction of veins in the temporoparietal area on the right side supporting the diagnosis of venous infarction (figure 1F). This diagnostic finding was confirmed 2 weeks later by an MRI venography (figure 1G). Before surgery a high-resolution MRI had been performed to reduce the chance of harming vessels during surgery, but nevertheless the reduction of veins could, in principle, also be caused by the surgery.

Taking into account the clinical presentation and the CSF, both with no signs of an ongoing infection, and the imaging results, we concluded that a relapse of the infection was unlikely. Consequently, we stopped antibiotics after 8 weeks of treatment. From a retrospective point of view we draw the conclusion, that the seizure symptoms 3 weeks before were most likely in the wake of a subacute venous infarction. Table 3 summarises the typical venous pathways at risk of thrombosis in the case of an infectious origin due to venous drainage.

Three months after stopping antibiotic therapy, the patient underwent a percutaneous atrial septal defect closure, as it is recommended for a symptomatic PFO. Cerebral imaging outcome control, carried out 2 weeks later, demonstrated further remodelling of the lesion and the expired venous infarction (figure 1H).

DISCUSSION

G. morbillorum very seldom causes cerebral infections. Up to now, eight cases of cerebral abscess caused by *G. morbillorum*

are described; additionally, there are three cases described with meningitis and two cases with subdural empyema, which all were caused by *G. morbillorum*.^{2–4} In two of the cases, a biphasic clinical course is described.^{3 5} On the other hand, one case is described, where meningitis caused by *G. morbillorum* was followed by a cerebral infarction.⁶

In the present case, the most probable late complication was a venous infarction. Since a relapse of the cerebral abscess would have been again a life-threatening event, the exclusion of a relapse in terms of an early stage cerebritis was essential for correct treatment decision. Especially in the present constellation, the analysis of the CSF, which is not necessarily recommended in the case of cerebral abscess, gave important information to disprove an infectious focus.

Learning points

- ▶ The parietal lobe is an uncommon location for a cerebral abscess and makes a haematogenous infectious pathway likely.
- ▶ A typical sign for a cerebral abscess in the MRI is the restricted diffusion (diffusion-weighted imaging hyperintense and apparent diffusion coefficient hypointense) due to cellularity and viscosity of purulence.
- ▶ *Gemella morbillorum*, although a harmless commensal, can cause biphasic infections and consecutive infarction in immunocompetent humans.
- ▶ Venous infarction can be a complication of cerebral abscess even under successful treatment.
- ▶ Investigation of the CSF can give essential hints for correct differentiation between venous infarction and cerebritis.

Competing interests None.

Patient consent Obtained.

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