Primary ventriculitis caused by Streptococcus intermedius – a rare case and challenge with uncertain clinical outcome. Case report

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Aims. To present a case of primary ventriculitis in a 53-year-old patient caused by Streptococcus intermedius, emphasizing the rarity of the condition and the challenges in achieving clinical improvement despite targeted therapy.
Methods. The patient underwent clinical evaluation, including CT and MRI imaging, as well as CSF analysis. Empirical antibiotic therapy was initiated with cefotaxime and metronidazole, followed by targeted therapy based on CSF culture results. External ventricular drainage was performed surgically.
Results. No predisposing factors were identified in the patient. Initial imaging showed no acute changes, but follow-up imaging revealed significant ventricular inflammation. CSF analysis confirmed the presence of Streptococcus intermedius. Despite early and targeted antibiotic therapy, and surgical intervention, the patient's clinical condition did not improve.
Conclusion. This case highlights the rarity of primary ventriculitis caused by Streptococcus intermedius and the challenges in managing it. The lack of clinical improvement despite prompt and targeted treatment underscores the need for further research to develop more effective therapeutic strategies for such infections.

Key words: ventriculitis, Streptococcus intermedius, ventricular debris, infection of central nervous system

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INTRODUCTION

Ventriculitis, most commonly either occurring as a secondary complication following inflammation or as a consequence of introduced ventricular drainage, represents a serious condition necessitating prompt diagnosis and treatment. Streptococcus intermedius, often part of the oropharyngeal microflora, is a rare pathogen associated with brain inflammation in patients without prior infectious history. Diagnosis relies on imaging studies and cerebrospinal fluid analysis. Rapid antibiotic therapy, including interventional treatment, is crucial for improving patient prognosis, although even this may not always yield expected outcomes.

CASE REPORT

Our clinic was consulted about the case of a 53-year-old patient, treated for hypertension and diabetes, who was admitted to the intensive care unit in the afternoon for headache, neck pain and cervical spine stiffness. After admission, no acute changes were detected on computed tomography (CT) of the brain and cervical spine. Aside from mild leukocytosis (12×10^9/L), laboratory results were normal. The following day the patient's clinical condition deteriorated, with qualitative alteration of consciousness including restlessness, confusion, brachial aggression and fatigue disorder. A control native CT of the brain revealed a hypodense border (edema) around the left lateral ventricle, especially the trigone (Fig. 1). The CT scan of the brain also ruled out any potential spread per continuitatem (Fig. 2). In addition, laboratory inflammatory markers progressed (leukocytosis – 22×10^9/L, CRP 111 mg/L).

The patient was admitted to our clinic for suspicion of an inflammatory etiology. At the same time, empiric antibiotic therapy with cefotaxime (2 g every 6 hours) and metronidazole (500 mg every 6 hours) was started. A magnetic resonance imaging (MRI) of the brain with contrast demonstrated thickening of the choroid plexus of the left lateral ventricle and an increase in its signal intensity in T2 and FLAIR imaging, post-contrast enhancement of the central part of the plexus and subependymal enhancement in the left lateral ventricle, the level of thickened content in the occipital horn of the right lateral ventricle (debris) showing diffusion restriction as well as left choroid plexus (Fig. 3).

The patient was already artificially comatose at first contact and was immediately transported to the operating theater for emergency surgery upon arrival. Under general anesthesia, external ventricular drainage of both lateral ventricles was performed under navigation control using antibiotic-impregnated catheters. Cloudy cerebrospinal fluid mixed with pus was derived from the left lateral ventricle. One drain was used as an inflow drain for local

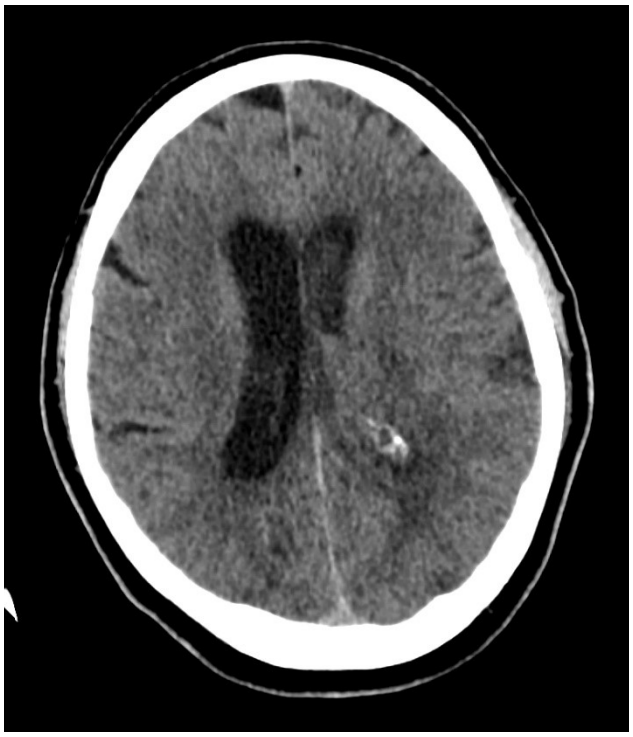

Fig. 1. Computed tomography of the brain – hypodense border around the left lateral ventricle.

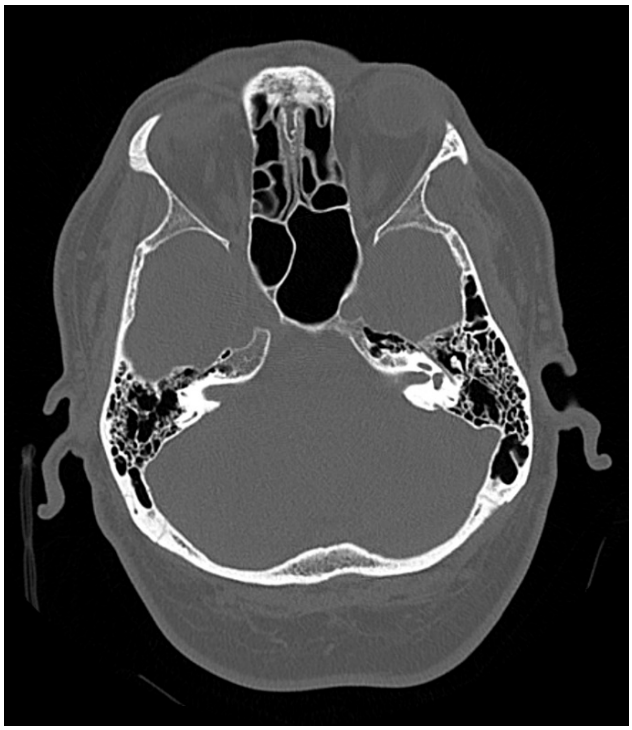

Fig. 2. Computed tomography of the brain – displaying the petrous bone and mastoids, all intact with no signs of inflammation.

antibiotic instillation and the other as an outflow drain for ventricular drainage. The patient was left on artificial lung ventilation after the procedure. Through ventricular drainage, antibiotic therapy was supplemented with vancomycin and gentamicin locally. We repeatedly isolated Streptococcus intermedius from the cerebrospinal fluid sensitive to the already empirically established cefotaxime. The initial examination of the cerebrospinal fluid showed a picture typical of inflammation, a negative coefficient of energy balance (KEB) (−1360; lactate 15.54 mmol/L, glucose below 0.20 mmol/L). CSF parameters gradually normalized with continued therapy. After six days, the ventricular drainage was extracted with a negative culture finding in the cerebrospinal fluid. Attempts to wean him from artificial lung ventilation were unsuccessful, and a tracheostomy was performed for borderline respiratory activity. Other control cultures of the cerebrospinal fluid were also negative. The patient underwent chest and lung X-rays, abdominal ultrasound, as well as cardiological, dental, and ENT (Ear Nose Throat specialist) examinations to exclude an infectious focus, all with negative results.

The patient was transferred to a subsequent intensive care unit to continue antibiotic therapy. After a month from the initiation of therapy, no improvement in the patient's neurological condition was observed. The patient was still dependent on artificial lung ventilation, without useful contact, he reacted to algic stimulus with facial grimacing and untargeted movement of the right lower limb. The control CT scans were satisfactory. The anamnesis was supplemented by the patient's family, where the patient did not experience any infection or symptoms before the illness.

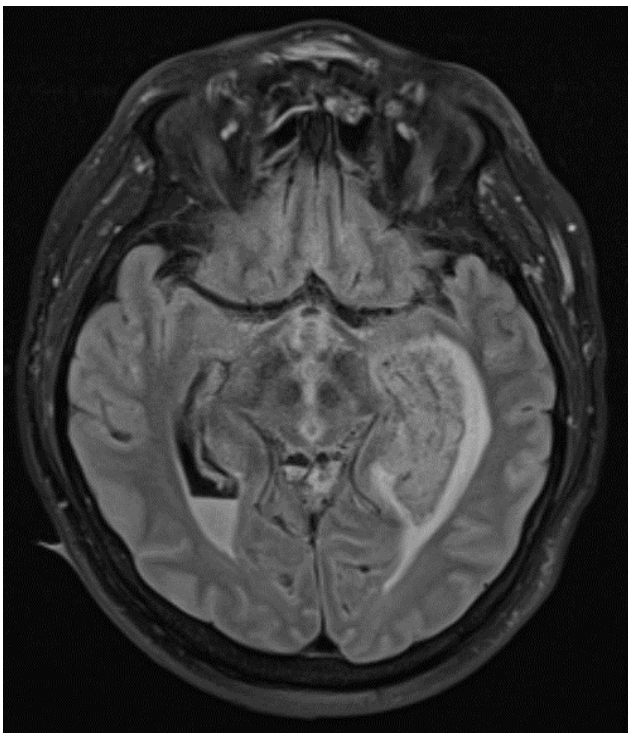

Fig. 3. Magnetic resonance imaging of the brain, FLAIR modality – level of debris in the occipital corner of the right lateral ventricle, enlargement of the choroid plexus of the left lateral ventricle with an increase in its signal intensity and periventricular edema rim.

DISCUSSION

Ventriculitis is a serious condition that requires prompt diagnosis and adequate treatment due to its potentially fatal course. This term has many synonyms including inflammation of the ependymal lining of the cerebral ventricles, ependymitis, ventricular empyema, pyocephalus and pyogenic ventriculitis[1]. In the professional public, its emergence is primarily connected with the insertion of external ventricular drainage. There are also other causes of its occurrence. These are secondary complications after trauma, after neurosurgery, conditions with leakage of cerebrospinal fluid, after intrathecal application of chemotherapy, after brain abscess or, last but not least, the result of bacterial or viral meningitis[2]. Hematogenous seeding and spread to the choroid plexus as a possible mechanism of secondary origin is reported by Harris et al. in his review[3]. An unforgettable factor that can contribute to its development is immunosuppression. However, the diagnostic puzzle that leads to this diagnosis is often not complete.

Identification of the exact pathogen is key to initiating specific antimicrobial therapy. The most common agents are gram-negative bacteria and staphylococci[1]. Primary ventriculitis without predisposing factors caused by Streptococcus intermedius is reported rather rarely[4]. In addition to cerebrospinal fluid cultivation, evaluation of the increase in markers such as lactate, procalcitonin and lysozyme helps to distinguish a viral from a bacterial agent[5].

Typical clinical manifestations are fever and meningismus[3]. On a CT scan of the brain we can see various non-specific signs, as like in meningitis. An MRI of the brain shows restriction of diffusion as like in an abscess and other inflammatory signs[3]. Fukui et al. prove that the most common MRI sign is ventricular debris, which was also imaged in our case. Graphically expressed ependymal hypersignality or complications in the form of hydrocephalus may not always be expressed[6]. Although not occurring in our case, the feared complication described is multiocular hydrocephalus, which is one of the reasons for the frequent graphic control recommended in these patients.

The basis of treatment is administration of antibiotics. In the case of ventriculitis, which arose as a result of ventricular drainage, their extraction is recommended due to the possibility of microorganisms adhering to the catheter[3]. An alternative to establishing an optimally effective level of antibiotics is their intraventricular application. Their choice depends on local customs, but vancomycin (5 to 20 mg/day) or gentamicin (1 to 8 mg/day) are most often recommended[2]. Unrecognized and untreated ventriculitis is burdened by the development of hydrocephalus or the development of a severe neurological condition and even death[7].

CONCLUSION

Primary ventriculitis caused by Streptococcus intermedius represents a rare and challenging case with an uncertain clinical outcome, underscoring the need for further research in this area despite prompt targeted therapy failing to improve the patient's condition.

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