Fatal Cerebellar Hemorrhage Complicating Pneumococcal Meningitis: Are Anticoagulants the Hidden Enemy?

Sir,

Bacterial meningitis is an infection of the central nervous system with an incidence of two cases per 100,000 adults. *Streptococcus pneumoniae* is the most frequent causative microorganism of community-acquired bacterial meningitis, causing 70% of adult cases.[1]

The cerebrovascular complications are associated with higher mortality and unfavorable outcome.[2] Intracranial hemorrhages (ICHs) have been described as an uncommon complication of meningitis occurring in 2%–9% of cases.[2,3] We report a case of pneumococcal meningitis complicated with fatal cerebellar hemorrhage. This case highlighted the risk of cerebral bleeding associated with anticoagulant use in these patients.

A 60-year-old woman, with a history of diabetes and hypertension, was admitted to the emergency department with altered mental status, fever, and respiratory distress. For 3 days before admission, she complained of severe occipital headache and purulent otorrhea for which amoxicillin was prescribed by an outside primary care physician. Physical examination revealed a temperature of 39°C, pulse rate 100 min^{-1}, normal blood pressure, and tachypnea with a SpO_2 of 92% on room air. Neurological examination revealed a Glasgow coma scale (GCS) score of 10 (E3, V2, and M5). There was no motor deficit and both pupils were isocoric and reactive to light. A head computed tomography (CT) scan was unremarkable. Laboratory tests showed elevated leukocytes (16.5×10^3 cells/μl) and C-reactive protein (39.7 mg/l). Cerebrospinal fluid (CSF) analysis disclosed an increased cell count (1360 cells/mm^3, 100% polymorphonuclear leukocytes), increased protein (9.15 g/l), and a low level of glucose (0.91 mmol/l). The patient was intubated and admitted to the Intensive Care Unit where she was given antibiotics (cefotaxime) and early adjunctive therapy with dexamethasone. The patient's clinical status gradually improved, and she was extubated on the 4th day. Final CSF cultures revealed *S. pneumoniae*. On the 4th day of hospitalization, lower extremity deep venous thrombosis was diagnosed by ultrasound, and intravenous infusion of unfractionated heparin was started. A day later, the patient developed a sudden severe headache associated with iterative vomiting and altered mental status (GCS 3/15) requiring reintubation. A head CT scan [Figure 1] showed a 4 cm right cerebellar hematoma compressing the 4th ventricle with hydrocephalus. Anticoagulation was discontinued. The activated partial thromboplastin time (aPTT) was 1.8. The patient developed a bilateral mydriasis. External ventricular drainage was immediately performed. Unfortunately, the patient died 8 h later.

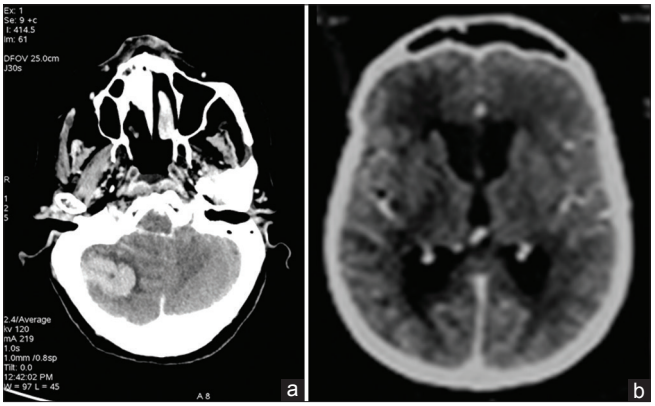

Figure 1: Brain computed tomography scan. (a) A right high-density area in the right cerebellar hemisphere measuring 4 cm in diameter corresponding to a spontaneous hematoma. (b) Acute hydrocephalus secondary to the fourth ventricle compression

There are several types of ICH in bacterial meningitis including intraparenchymal hemorrhage and microbleeds from arterial bleeding, hemorrhagic transformation of arterial or venous infarcts, and subarachnoid hemorrhage and abscess formation with subsequent hemorrhagic transformation. Parenchymal hemorrhages are the most common.[4,5] The pathogenesis of ICH in bacterial meningitis has not been explicitly documented. The most likely explanation is local hemorrhage secondary to vasculitis associated with central nervous system infection.[3]

The greatest concern regarding anticoagulation is the risk of intracranial bleeding, which may be increased further in patients with bacterial meningitis. A nationwide prospective cohort study showed a 5-fold increased risk of developing ICH if the patient with bacterial meningitis uses anticoagulant therapy.[5] Many authors attempted to answer the question, still controversial, regarding the benefits and potential risks of anticoagulant therapy in these patients. Several factors must be considered, such as the type of bacteria (*Staphylococcus aureus* is the most common organism underlying symptomatic ICH), the underlying pathology (septic emboli or ruptured mycotic aneurysm complicating infective endocarditis), comorbidities (severe uncontrolled hypertension, severe renal failure, and myeloproliferative neoplasms), congenital or acquired hemostatic disorders (thrombocytopenia and platelet dysfunction), and the type of anticoagulant.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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