

Acute Gastric Dilatation Causing Bacterial Cerebral Aneurysm—Case Report

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ABSTRACT

Objective: Acute gastric dilatation (AGD) is a very rare entity which can sometimes be life-threatening. We report a case of a patient presenting with a rupture of a BCA during the treatment of AGD.

Method: A 24-year-old woman, who had a history of bulimia and vomiting episodes, was transferred in shock with marked abdominal distension. A large nasogastric tube was inserted, and 9 liters of viscous gastric contents were drained out. Her circulation became stable.

Results: About 3 months after admission, she became drowsy and presented with a right hemiparesis and aphasia.

Computed tomography of the head showed a diffuse thick subarachnoid hemorrhage. Left carotid angiograms revealed an obscurely-shaped aneurysm in the left middle cerebral artery.

Conclusion: Trapping of the aneurysm was performed. Thirty-four days after admission, the patient had a residual right hemiparesis and motor aphasia, and was discharged. © 2008 by Wiley Periodicals, Inc.

Keywords: bacterial cerebral aneurysm; acute gastric dilatation; bulimia

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Introduction

Bacterial cerebral aneurysm (BCA) is very rarely seen in clinical practice. BCA occurs primarily as a result of infectious complicating cardioaortic diseases, such as rheumatic and congenital heart diseases.¹ Acute gastric dilatation (AGD), which is a very uncommon pathology, has not been previously reported as causing BCAs. We herein report a case of a patient presenting with a subarachnoid hemorrhage due to the rupture of a BCA during the treatment of AGD.

Case Report

A 24-year-old woman was transferred to our hospital in shock with marked abdominal distension. The patient had a history of bulimia and vomiting episodes and was routinely seen by a psychiatrist. She was not taking any medications or toxic substances. On September 19, 2005 she was admitted to a nearby hospital, complaining of abdominal

pain that occurred after eating an enormous amount of food. Two days after admission, she developed shock and was transferred to our department for treatment of her circulatory disturbance. On admission, she was a normally developed, but malnourished female with an extremely distended abdomen (height: 160 cm; weight: 34 Kg). Her consciousness level was normal. The patient's blood pressure was 76/38 mm Hg, and her heart rate was 93 without arrhythmia. Heart and respiratory sounds were clear. Her distended abdomen was tender and resistance to palpation was noted. An abdominal X-ray showed extreme gastric dilatation extending to the pelvis (Fig. 1). A large nasogastric tube was inserted, and 9 l of viscous gastric contents were drained out. Her abdomen became flat, and the gastric dilatation was thought to regress, though the size of the stomach did not shrink to normal. The patient's circulation became stable. Two months after her initial admission, the patient developed a low grade fever that continued for 5 days. At that time, laboratory tests showed acute inflammatory findings, including elevated white blood cells, γ -globulin, and C-reactive protein. On blood cultures, a bacteremia of methicillin-resistant *Staphylococcus aureus* was found. After antibiotics and γ -globulin were given, the fever subsided rapidly. The patient's general condition started to improve and she even tried to eat some food. However about 3 months after her initial admission, the patient became drowsy and presented with a right hemiparesis and aphasia. Her pupils were anisocoric (right: 1.5 mm; left:

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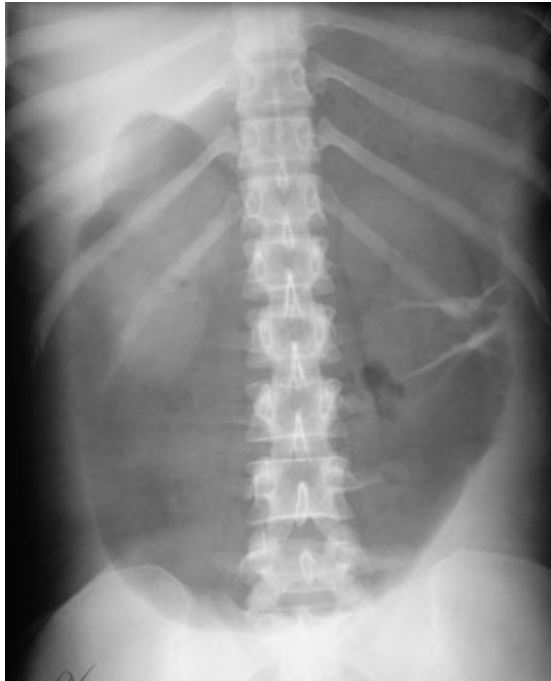
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FIGURE 1. An abdominal X-ray shows extreme gastric dilatation extending to the pelvis.

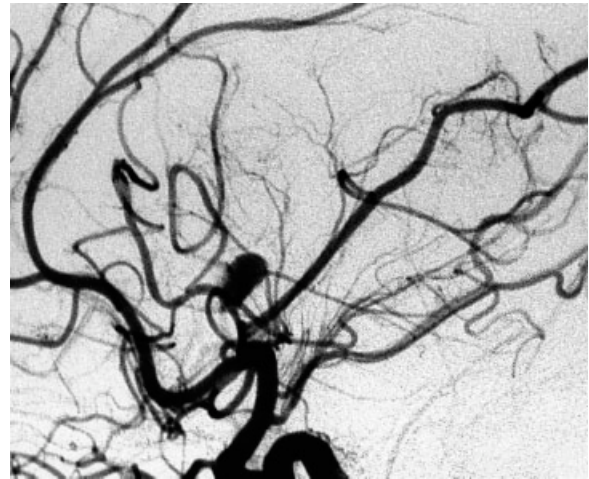


3.0 mm). Computed tomography of the head showed a diffuse thick subarachnoid hemorrhage at the basal cistern and an intracerebral hemorrhage in the left temporal lobe. On laboratory testing, no abnormal findings that would have indicated infection were found. No cardiac murmurs were audible. Cardiac ultrasound, which was frequently done to obtain information about infectious endocarditis (IE), showed no vegetations on the valves. On the left carotid angiograms, an obscurely shaped aneurysm involving several branches of the sylvian portion of the left middle cerebral artery were noted (Fig. 2). Surgical intervention was performed to prevent rebleeding from the aneurysm and to remove the intracerebral hematoma. Trapping of the aneurysm was performed. On visual inspection, the aneurysm was partially thrombosed and very fragile. The specimen of the excised aneurysm revealed that the aneurysmal wall was infiltrated with a large amount of polymorphonuclear neutrophils, indicating a bacterial aneurysm. Thirty-four days after admission, the patient had a residual right hemiparesis and motor aphasia, and was discharged to the rehabilitation program.

Conclusion

BCA is rare, and its incidence is decreasing because of proper use of effective antibiotics. BCA occurs in

FIGURE 2. Left carotid angiograms shows an obscurely shaped aneurysm involving several branches of the sylvian portion of the left middle cerebral artery, and severe narrowing, as well as obstruction of the distal arteries exiting from the aneurysm.



0.4% of all patients with intracranial aneurysms and in 3% of all patients with IE.¹ The main etiology of BCA is IE associated with rheumatic or congenital heart diseases. The mortality rate of patients with an unruptured BCA is 30%, but in patients with a ruptured BCA, the mortality rate is 80%.² The pathogenesis of BCA is caused by the lodging of septic emboli at distal sites within the circulation. Bacterial translocation via the vasa vasorum to the adventitia, and inflammatory necrosis extends from the adventitia to the medial layer, which leads to aneurysmal formation.^{1,2} BCA is treated with antibiotics as well as surgical intervention. Should antibiotic therapy not control the infection or the aneurysm enlarges, then surgical intervention is required. Clipping is preferred, but the involvement of the vessel wall by the infectious process may make it difficult to do so.

AGD is a very rare entity which can sometimes be life-threatening due to complications including gastric necrosis, perforation, ischemic bowel injury by compression of mesenteric vessels, and shock.³ AGD is caused by various factors, such as: excessive eating, pylorospasm after scarring secondary to peptic ulcer, posttrauma, pregnancy, ketoacidosis, or postsurgery.⁴ The symptoms include restlessness, vomiting, dyspnea, and abdominal pain.³ Aspiration of gastric contents is a frequent and dreaded complaint. If the syndromes are not diagnosed early and treated, then circulatory collapse and cardiac arrest can occur.^{3,4} Initial therapy must include nasogastric decompression and fluid resuscitation. Most patients respond to nasogastric tube

drainage, but some require gastrectomy with esophagojejunostomy. Early recognition is essential, since 80% mortality rate has been reported with delayed treatment.⁴ Even if the patients' stomach shrinks and their circulation is stabilized, they will have gastric mucosal damage.⁵

There has been no report of BCA following AGD. In our case, a cardiac murmur was not heard and on the echocardiography done before the BCA ruptured, no vegetations on the valves and no valvular dysfunction, suggesting IE, were noted. Therefore, the aneurysm may have been formed by bacteremia or infectious emboli originating in the necrotic gastric mucosa, which persisted even though gastric decompression was successful. Thus, even in stabilized patients with AGD that is well controlled and whose infection is well controlled by proper antibiotics, special attention must be given to gastric lesions, which could serve as infectious foci

and lead to systemic bacterial infections such as IE or BCA.

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