

slurred speech. Pyrexia is absent.³ The cranial nerve symptoms are frequently, but not always, preceded by nausea, vomiting and abdominal cramps, especially after ingestion of contaminated foods. Incubation periods of between 3 hours and 54 days have been reported⁴ but 12–72 hours is often quoted. The clinical diagnosis of botulism is confirmed by injection of the patient's serum into a laboratory animal, which then shows signs of neurotoxicity.

Seven serotypes of the toxin are known, but only types A, B and E have been consistently responsible for botulism in man and trivalent antitoxin directed at these is the treatment of choice. Prompt treatment is crucial since, once the toxin has blocked neurotransmission at a synapse, antitoxin will have no effect on that synapse. Even a suspicion of botulism demands treatment with antitoxin, without waiting for a laboratory test. Recovery from the neurotoxic injury depends on the regeneration of neuromuscular junctions, which may take many months and is often incomplete. The implementation of ventilatory support has reduced the mortality from 60–70% in the early twentieth century to around 20% more recently.⁵

Even with the diagnosis of botulism, our patient's progression within 45 minutes from an alert ambulatory state to respiratory arrest is hard to explain. We suspect that his earlier symptoms of weakness and somnolence reflected increasing compromise of his tidal exchange, culminating in the hypoxia/hypercardia that precipitated the arrest.

Previously unseen and unpredicted routes of infection with botulinum toxin have emerged. Intravenous and nasal routes from recreational drugs 'cut' with contaminated soil particles and 'inadvertent' botulism in patients being treated with botulinum toxin for dystonias and cosmetic procedures⁸ have been reported. The possibility of the use of *C. botulinum* and its toxin as a weapon for bio-terrorism is considered both 'real and frightening'.^{2,9}

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An aneurysm is born

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J R Soc Med 2003;96:550–551

When a *de novo* intracranial saccular aneurysm is detected at a site where angiography was previously negative, the interval between angiographies is usually a matter of years^{1,2} rather than days.

CASE HISTORY

A woman of 76 with a history of hypertension developed headache with mild disturbance of consciousness. There was no personal or family history of inflammatory, collagen or cerebrovascular disease. On neurological examination there were no localizing features and laboratory data were all within the normal range. Urgent cranial CT revealed diffuse subarachnoid haemorrhage in the interpeduncular and ambient cisterns, and a magnetic resonance angiogram disclosed a single aneurysm, 12 mm in diameter, at the bifurcation of the basilar artery (Figure 1). Eight days later, the patient suddenly vomited and became comatose with neck rigidity. 30 minutes after this, CT revealed a dense subarachnoid haemorrhage localized in the left sylvian fissure. Next day the basilar artery aneurysm, which was then thought to be responsible for both vascular events, was successfully occluded by endovascular coiling. At that time a new aneurysm (Figure 2), 10 mm in diameter, was observed at the bifurcation of the left middle cerebral artery; the artery itself was not clearly visualized owing to vasospasm. Because of the patient's clinical state (semi-comatose and dyspnoeic) the new aneurysm was left untreated. 7 days later, magnetic resonance angiography showed the left middle cerebral artery to be completely occluded proximal to the aneurysm, which was no longer visible (Figure 3). Thereafter the patient remained stuporose with a right hemiplegia.

COMMENT

The shortest reported interval between an angiography and the demonstration of a *de novo* aneurysm at further angiography is 47 days.³ In the present case the interval was 9 days. The arteriographic appearance was suggestive of a true saccular aneurysm rather than a mycotic, dissecting or arteriosclerotic aneurysm.

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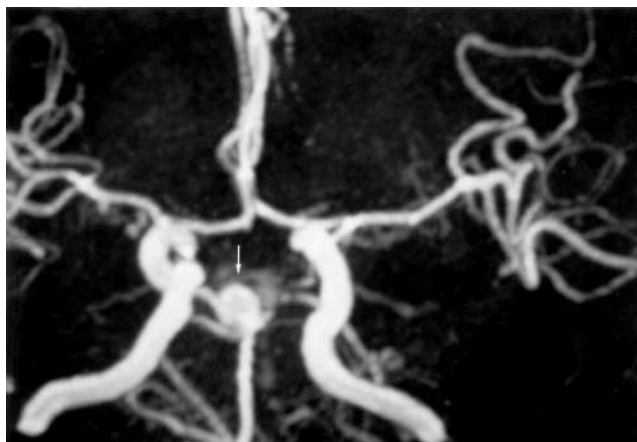


Figure 1. Initial MR angiogram. A single aneurysm is seen at the bifurcation of the basilar artery (arrow)

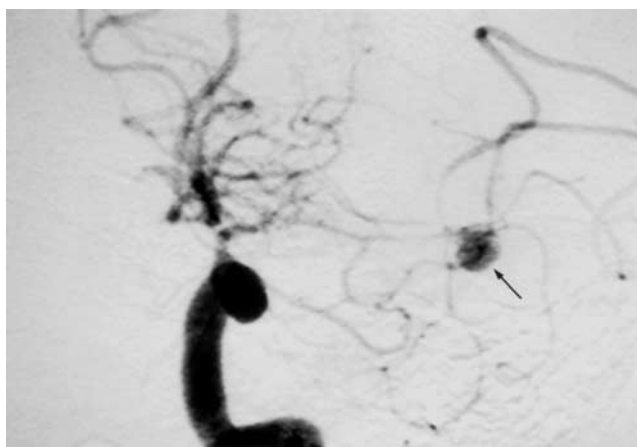


Figure 2. Left internal carotid digital subtraction arteriogram 9 days later. A *de novo* saccular aneurysm is seen at the bifurcation of the left middle cerebral artery (arrow)

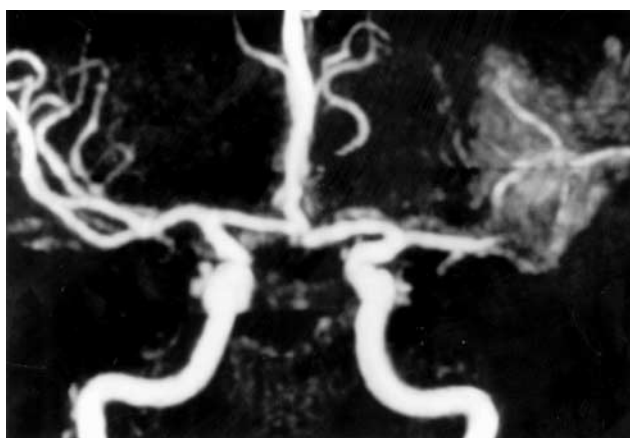


Figure 3. MR angiogram 16 days after initial subarachnoid haemorrhage. The left middle cerebral artery is completely occluded proximal to the aneurysm, which is no longer visible

Could the middle cerebral artery aneurysm have been missed at the original magnetic resonance angiography? Digital subtraction arteriography is certainly more sensitive; however, a basilar bifurcation aneurysm and the first and

second portion of the left middle cerebral artery are clearly seen in the first magnetic resonance angiogram. Moreover, on examination of the published work⁴⁻⁶ I could find no report of false-negative magnetic resonance angiography for an aneurysm greater than 10 mm in diameter. On this evidence I conclude that this aneurysm did not exist at the time of initial study 9 days earlier.

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Aortic root replacement in pregnancy

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J R Soc Med 2003;**96**:551-552

When complex cardiac operations become necessary during pregnancy, timing is crucial to survival of the fetus.

CASE HISTORY

A woman of 31, in the 10th week of her first pregnancy, reported palpitations. She was tall, with fingers and toes unusually long for her family, and had 'double-jointed' thumbs. She was also short-sighted and her hearing was poor. There was no medical or family history of note. The pregnancy had followed 3 years of infertility treatment. Aortic regurgitation was diagnosed clinically, and she met

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