Mysterious dysphagia

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In a visitor from Eastern Europe, dysphagia has special connotations.

CASE HISTORY

A man of 30 was referred to our service from the accident department complaining of something wrong with his throat. He was from Poland and had entered the UK three months ago to find work. His command of spoken English was limited, but with the assistance of a friend he indicated that three days earlier during a chicken meal he had experienced 'blockage in the throat', since when he had taken very little by mouth, either solid or liquid. There was no voice change, and he had experienced no swelling or tenderness of the neck. He had vomited initially but not subsequently. Now he felt tired, weak and unwell.

On examination he was afebrile. There was no obvious cranial nerve or other neurological dysfunction, and the usual vital signs and observations were within the normal range. On inspection of the oral cavity, pharynx and hypopharynx there were no masses. The pharyngeal mucosa was slightly reddened and showed white lacy exudates suggestive of candidiasis. The faucial tonsils were asymmetrical and slightly inflamed but not unduly enlarged and there was no peritonsillar swelling.

The findings on flexible laryngopharyngoscopy were all normal—full and equal movement of the vocal cords; no evidence of foreign body or recent trauma; no pooling of saliva in the hypopharynx. The patient was admitted, placed on intravenous fluids to counteract his dehydration and started on intravenous benzylpenicillin and metronidazole for the provisional clinical diagnosis of pharyngitis. Later, nystatin pastilles were added. Blood test results, including the erythrocyte sedimentation rate and the Paul–Bunnell test (for infectious mononucleosis), were essentially normal. The white cell count was $15.9 \times 10^9/L$, with a differential cell count showing only neutrophilia. Plain radiographic evaluation of the soft tissues of the neck and a chest X-ray were likewise unrevealing. The day after admission a barium swallow was contemplated, but this was not feasible since the patient could not or would not

swallow a single sip of water. After 48 hours his ability to swallow had not improved. No other systemic symptoms were offered and no additional signs were evident. On flexible endoscopy of the oesophagus and stomach the white mucosal exudates of the pharynx were seen not to extend into the oesophagus, thereby excluding oesophageal candidiasis; no other abnormality was seen. In view of the suspected pharyngeal candidiasis, immunodeficiency was a consideration. The patient readily consented to an HIV test, which was negative. He continued to be alert and cooperative but expressed a feeling of general malaise. The supportive measures were continued.

On the evening of his eighth day after admission he was observed walking to the toilet, talking and responding in a manner no different from usual. Forty-five minutes later, a nurse found him lying on his bed motionless, pulseless and not breathing. The cardiac arrest team succeeded in reestablishing a circulation but in the intensive therapy unit he was unresponsive to painful stimuli and was noted to exhibit episodic, spontaneous twitching of the upper and lower limbs. CT of the head showed no intracranial lesion.

About 24 hours after the cardiac arrest another Polish friend informed staff of something that had happened to an acquaintance of the patient. This was a heavy-goods-vehicle driver who had been in the UK a few days before our patient became ill and had shared with him a dish known as *bigos*, or 'hunter's stew', purchased commercially in Poland. *Bigos* includes smoked sausage, ham, mushrooms, cabbage and numerous other vegetables. The driver had then returned to Poland and fallen ill, where he was taken to hospital and promptly treated for botulism. On the ninth day after admission, we began treating our patient with botulinum antitoxin, and fourteen days later type B botulinum neurotoxin was identified in his serum. After four weeks in which there was no improvement in the patient's cerebral function, the family elected to take him back to Poland for continued ventilatory and supportive care.

COMMENT

Botulism is the name given to the paralytic disease caused by the neurotoxin released by *Clostridium botulinum*. It is very rare in the UK, only 58 cases having been reported between 1922 and 1998.¹ In Poland, however, several hundred cases are seen every year, attributable to the widespread practice of preserving foods in the home.² After ingestion or subsequent production in the gut the neurotoxin irreversibly blocks transmission of nerve impulses at neuromuscular junctions and in the cholinergic autonomic nervous system. It binds most readily at locations where there is rapid cycling of depolarization and repolarization; cranial nerves are affected primarily, and common initial symptoms are diplopia, blurred vision, dry mouth and

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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 botulinal toxin have emerged. Intravenous and nasal routes from recreational drugs 'cut' with contaminated soil particles and 'inadvertent' botulism in patients being treated with botulinal 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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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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