TREMORS AND FUNGUS

An interesting report in the Medical Journal of Australia points out some of the perils of food poisoning. An elderly couple was admitted to hospital after sudden onset of severe muscle tremors. The husband, aged 89, and wife aged 84, had shared a can of soup and some toast at about 12 noon, followed by chocolate cake, before catching a bus to the local shopping centre. They noticed the soup had a bitter taste. There had been no recent changes to their medications, and no recent exposure to garden sprays, insecticides or pesticides.

The man reported feeling slightly shaky as the bus arrived at the shops at about 13h00. He had difficulty getting off the bus, his legs felt ‘wobbly’ and he had to sit down. He developed uncontrollable shaking and felt sticky and sweaty. An ambulance was called and the initial assessment by the paramedics at about 13h10 was that he had severe muscle tremors and was unable to walk, and that it was difficult to understand his speech. He had no nausea or vomiting, and his abdomen was soft. He was sweating, with blood pressure 160/76 mmHg, pulse rate 80 beats/min, respiratory rate 32 breaths/min, and Glasgow Coma Score 15 of 15. His oxygen saturation level was 85%, improving to 97% with high-flow oxygen.

Subsequent assessment in hospital at 13h40 revealed that the patient’s Glasgow Coma Score had fallen to 11. Although his speech was incomprehensible, he was able to obey commands; he also displayed generalised weakness and intention tremor. He remained hypertensive and diaphoretic, but was afibrile. He later recounted that he had thought he was going to die. All symptoms, apart from the intention tremor, had subsided by 16h30.

The patient’s medical history included chronic airway limitation, asthma, hypertension, episodic atrial fibrillation, osteoarthritis, a left total hip replacement, and an essential tremor. He had no known allergies. His medications included verapamil, digoxin, salbutamol, tiotropium bromide, salmeterol xinafoate, quinine bisulfate prn, paracetamol and rofecoxib prn. His essential tremor remained untreated because of the contraindicated use of β-blockers in asthma.

A chest X-ray was unremarkable, but electrocardiography revealed a right bundle branch block. A computed tomography brain scan showed generalised volume loss with no intracranial haemorrhage or early changes of infarction; a small low-density focus present in the right basal ganglia region was consistent with chronic lacunar infarction. The results of haematological and biochemical tests were within normal limits, except for a raised urea. No urine screen for drugs was carried out. The patient was admitted for observation and investigation. The following day he was lethargic, exhausted, and displayed minimal tremor. He was eventually able to mobilise with the aid of his usual walking stick.

The woman presented in a similar way. She complained of feeling dizzy and shaky, but had no nausea or vomiting. The paramedics described her as shaking uncontrollably, anxious and sweaty. Her blood pressure was not palpable because of severe shaking, but she was well perfused. She was tachycardic (pulse rate 110 beats/min, taken from electrocardiographic monitoring), with a respiratory rate of 20 breaths/min, temperature 36.5°C, Glasgow Coma Score of 1.5, and blood glucose level normal. Although she was no longer tachycardic on admission to hospital, she remained anxious and diaphoretic, with a generalised tremor that increased on questioning. Her oxygen saturation level on room air remained above 95%. All symptoms, apart from the intention tremor, subsided by 16h30. The patient’s medical history included a left nephrectomy for calculi, right renal calculi and lithotripsy, chronic airway limitation, asthma, hypertension, and a left-eye cataract. Her medications included hydrochlorothiazide, frusemide, nitrofurantoin, aspirin, trimethoprim, salbutamol prn, beclomethasone dipropionate, quinine bisulfate prn, paracetamol prn, and Macu-Vision tablets [containing ascorbic acid, vitamin E, zinc oxide and cupric oxide]. A chest X-ray and electrocardiogram were normal. A computed tomography brain scan showed no abnormality apart from a polypoid density in the inferior maxillary sinus. The results of haematological and biochemical tests were normal, except for a raised urea and creatinine. The level of pseudocholinesterase was within normal limits. A urine screen conducted for drugs of abuse (including ampheta-mines, methamphetamine, benzodiazepines, cocaine, opiates and cannabis) was negative. The patient was admitted for observation, and gradually improved the following day.

Both patients were discharged 2 days after admission with a very slight residual intention tremor. When assessed by their GP 2 weeks later, the man had only a mild increase in his usual essential tremor and the woman’s tremor had completely resolved.

The damaged soup can was retrieved from the couple’s home the day they became ill and sent to Food Science Australia for analysis. There was a dent in the top adjacent to the ring-pull on the lid and black mould inside the can at the top near the damaged area, and a small amount on the underside of the lid. The mould was identified as Penicillium crustosum. This is a common foodborne fungus that causes spoilage in a wide variety of foods, including meat, cereals, nuts, cheese, eggs, fruit, and processed and refrigerated foods. Almost all P. crustosum isolates produce the mycotoxin penitrem A. Its potent neurotoxic effects have been demonstrated in various laboratory animals, and naturally acquired intoxication has been reported in sheep, cattle, horses and domestic dogs. In animals, symptoms include ataxia, tremors and severe muscle fasciculations. Larger doses may cause seizures, massive liver necrosis and death.


Bridget Farham