A woman with tetraparesis and missed beats

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DISCUSSION

This patient presented with clinical features consistent with acute cervical myelopathy or Guillain–Barré syndrome but ventricular arrhythmias on the electrocardiogram alerted the authors to a possible toxic cause. The history of drug ingestion was then elucidated, indicating aconitine poisoning from herbal medicine. Aconitine-containing plants are the most frequently encountered herbal agents that cause serious toxicity in Hong Kong (Chan et al, 1993).

Cardiac arrhythmias are the commonest presenting feature, with a mortality rate of 12% (Tai et al, 1992). Toxic effects of aconitine alkaloids result from their action on voltage-sensitive sodium channels that prolongs repolarization in the cardiac myocytes, leading to early after-depolarization and triggered activity. Onset of symptoms typically occurs within minutes of ingestion. Monomorphic and polymorphic ventricular tachycardias, and polymorphic ventricular ectopics are commonly encountered. Other arrhythmias include ventricular fibrillation, supraventricular tachycardias, sinus bradycardia and heart block. Hypotension can also be the result of muscarinic overactivity. Hyperventilation results from stimulation of the medullary respiration centre.

This case is unusual as the presenting feature is neurological rather than cardiological. Tetraplegia has been observed as a rare complication of aconitine poisoning (Chan et al, 1993, 1994). The pathogenic mechanism is unknown. This patient's clinical manifestations suggest that the neurotoxic action of aconitine alkaloids may be directed against sodium channels in both the neuronal membrane and neuromuscular junction. Opening of sodium channels in the axon terminal results in depolarization and causes sensory symptoms. With persistent activities of sodium current at the motor endplate, the postsynaptic muscle membrane will be in a sustained inexcitable state leading to paralysis.

Aconitum species are used in Chinese herbalism to treat rheumatism, ischaemic heart disease and intestinal colic. The proposed but unproven therapeutic action is enhancement of peripheral and visceral circulation (Li, 1989). Commonly used aconitine-containing medicinal herbs include 'chuanwu', 'caowu' and 'fuzi'. The recommended dose of fuzi (lateral root of *Aconitum carmichaeli*) is 1.5–4.5 g of raw material (Li, 1989), but potentially toxic doses of 7–11 g are often used (Chan and Critchley, 1996). Our patient's herbalist prescribed 110 g of fuzi, 10 times the intended quantity. Over-the-counter sale of herbs is not legally regulated in Hong Kong. This lethal dose of fuzi was dispensed straightaway despite the grossly erroneous prescription.

There is no specific antidote for aconitine poisoning. Life-threatening ventricular tachyarrhythmias have been managed anecdotally with amiodarone and flecainide (Tai et al, 1992). Refractory cases may require procainamide, mexiletine, bretylium or magnesium (Kolev et al, 1996). Toxic actions at the neuromuscular level are self-limiting, and thus expectant support seems to be the best measure. HM

CASE REPORT

A 55-year-old woman presented with acute paraesthesia affecting her limbs, which progressed rapidly to tetraparesis. Her power was grade 2/5 over the upper limbs and 1/5 over the lower limbs and she was areflexic. Sensation was also impaired but without a definite sensory level. Cranial nerves were normal. She also developed hypotension with blood pressure of 60/30 mmHg requiring fluid resuscitation. She was tachypnoeic with a respiratory rate of 28/minute. The initial differential diagnoses were acute cervical myelopathy complicated by spinal shock or Guillain–Barré syndrome with dysautonomia.

Investigations showed respiratory alkalosis, normal electrolytes and cardiac enzyme levels, and negative toxicology screen. Chest and cervical spine X-rays were unremarkable, but an electrocardiogram revealed multifocal ventricular ectopics. On further questioning, she recalled that a Chinese herbal concoction was taken 30 minutes before her symptoms appeared. The remedy was recommended by a lay herbalist as a tonic and the concoction was prepared by boiling 18 medicinal herbs, which included 110 g of 'fuzi', in water for 2 hours. Aconitine poisoning was diagnosed based on her clinical features and a history of exposure to an aconitine-containing herb.

The arrhythmia subsided spontaneously after 8 hours. Her neurological signs gradually improved and full power and tendon reflexes returned over the next day. She was in sinus bradycardia with borderline hypotension for 36 hours before becoming haemodynamically stable. Neurophysiological studies, including repetitive nerve stimulation and late responses, performed 3 days after onset and 2 weeks later, were normal.

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