CASE REPORT

Transient quadriceps paresis following cocaine use

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SUMMARY

A 31-year-old man presented with bilateral thigh muscle paralysis several hours after intranasal cocaine use. His blood results showed a creatine kinase (CK) level of 3447 u/L and a normal renal function. He made a marked recovery in just 2 h following the intravenous normal saline 0.9%, and was able to walk. He was discharged home the next day.

CASE PRESENTATION

A 31-year-old man presented with a 6 h history of bilateral leg weakness. He denied any pain, headache, vomiting or rashes and there was no history of any prodromal illness or injury. He recalls a similar episode in the past, which was resolved spontaneously.

The patient was not on any medications, but admitted that he occasionally uses cocaine and had used it the previous night.

On examination, his vitals were within normal limits. Cranial and peripheral neurology were normal apart from isolated decrease in power of 2/5 (movement when gravity is eliminated) of the hip flexors and knee extensors bilaterally. The thigh muscles were not tender.

INVESTIGATIONS

Haematological investigations showed a white cell count of 18.9×10^9 L (67% neutrophils) and creatine kinase (CK) 3447 u/L. His urea and electrolytes, renal and liver function tests were normal and had a negative HIV and a hepatitis screen.

TREATMENT

The patient was started on intravenous 0.9% normal saline, and, within 2 h, he reported that his power was back to normal and was able to walk. His CK decreased to 1800 u/L on day 2.

OUTCOME AND FOLLOW-UP

No further investigations and no treatment apart from intravenous fluids were required due to the quick resolution of his symptoms and a decrease in CK the next day. The patient was advised not to use cocaine again and was discharged with a follow-up appointment.

DISCUSSION

Cocaine-induced skeletal muscle myopathy is rare and it can range from just a transient myalgia to a full-blown rhabdomyolysis and acute renal failure and even death.^{1 2} The commonest muscle affected by cocaine is the myocardium and this is due to the

coronary vasospasm in combination with an increased sympathetic activity, which can precipitate myocardial infarction. In the long term, cocaine use can cause cardiomyopathy due to the increase of mechanical work as a consequence of increased intracellular calcium, increased sympathetic activity and ischaemia. The commonest prescribed drugs that can cause myopathy are corticosteroids and statins. A large spectrum of illicit drugs can also precipitate acute muscle injury including heroin, amphetamines and cocaine.

The exact aetiology as to why in our case it has affected the thigh muscles and not any other muscle group is not known. It might be due to the muscle bulk or the muscle most used after the cocaine use. Our patient had increased muscle bulk, especially in thighs, due to regular exercise for bodybuilding. He also admitted using steroids in the past, but the last time he used them was more than six months previously.

A prospective trial comparing all emergency attendances related to cocaine found that 24% (8/34 patients) of these patients had rhabdomyolysis but only one of those eight had muscle symptoms. The study also found that 3/34 people with cocaine-related muscle weakness had no evidence of high CK.³

The effects of cocaine on muscles are due to the vasoconstriction of the intramuscular arteries causing ischaemia⁴ and the interference with the sodium channels. Cocaine works by blocking the transient inward flux of sodium across the cell membrane during depolarisation, causing an increase of intracellular calcium resulting in tetany and therefore muscle breakdown and weakness.

Whether the intravenous normal saline resulted in resolution of symptoms due to the increase of plasma sodium overcoming the effects of cocaine or by washing out the toxic metabolites from the muscle vascular bed is difficult to establish without examining what happens at a cellular level and at the neuromuscular junction (NMJ).

The management of myositis is established first by the diagnosis with muscle biopsy, MRI and auto-antibodies in case of autoimmune cause.⁵ The different types of myositis are polymyositis, inclusion body myositis, necrotising myopathy and dermatomyositis, and are differentiated by biopsy. The illicit drugs mentioned above cause necrotising myopathy and the hallmark on biopsy is inflammatory cells surrounding the necrotic tissue.⁵

This case is unique, as we have not encountered in the literature a similar presentation in which there was such a quick resolution of symptoms with intravenous fluid administration.

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Learning points

- ▶ The use of illicit drugs should be kept in mind when dealing with the myositis especially in the young.
- ▶ Conservative treatment and observation is recommended if isolated rise of CK is present with normal renal function.
- ▶ If the cause is not identified, then the investigations of choice are biopsy, MRI and autoantibodies.

Competing interests None.

Patient consent Obtained.

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REFERENCES

- 1 Byard RW, Summersides G, Thompson A. Confluent muscle pallor: a macroscopic marker of cocaine-induced rhabdomyolysis. *Forensic Sci Med Pathol* 2011;7:364–6.
- 2 Raghuram K. Cocaine-induced rhabdomyolysis with secondary acute renal failure. *Applied Radiology* 2012;41(1). http://www.appliedradiology.com/uploadedFiles/Issues/2012/01/Cases/AR_01-02-12_RaghuramOLRC.pdf (accessed Oct 2013).
- 3 Welch RD, Todd K, Krause GS. Incidence of cocaine-associated rhabdomyolysis. *Ann Emerg Med* 1991;20:154–7.
- 4 Nemiroff L, Cormier S, LeBlanc C, *et al*. Don't you forget about me considering acute rhabdomyolysis in ED patients with cocaine ingestion. *Can Fam Physician* 2012;58:750–4.
- 5 Carstens PO, Schmidt J. Diagnosis, pathogenesis and treatment of myositis: recent advances. *Clin Exp Immunol*. Published Online First: 27 Aug 2013. doi:10.1111/cei.12194

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