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OPIS PRZYPADKU / *CASE REPORT*

Intravenous lipid emulsion in wide complex arrhythmia with alternating bundle branch block pattern from cocaine overdose

Dożylna emulsja tłuszczowa u pacjenta z arytmią z szerokimi zespołami QRS z naprzemiennym blokiem odnóg pęczka Hisa po przedawkowaniu kokainy

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Abstract

We describe the management of a young patient who had experienced a cocaine overdose. The patient presented with altered mental status and seizures and subsequently developed a wide complex arrhythmia with a rare alternating bundle branch block pattern. Intravenous lipid emulsion was administered following initial resuscitation and endotracheal intubation, because conservative methods of treating the persistent cardiac arrhythmias failed.

Key words: cocaine, wide complex rhythm, intravenous lipid emulsion

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INTRODUCTION

Cocaine overdose is associated with a variety of arrhythmias which are considered the main cause of mortality in this population. Wide complex arrhythmias have been reported with cocaine overdose, but the management of these has been a matter of debate. Intravenous bicarbonate and lidocaine have been used, with varying results [1]. Recently some authors have reported the successful management of these arrhythmias with intravenous lipids in patients with cocaine toxicity [2]. We report an interesting case of a patient with significant wide complex arrhythmias with an alternating right (RBBB) and left bundle branch block (LBBB) pattern from cocaine overdose, who failed to respond to intravenous lipids therapy.

CASE REPORT

A 30-year-old man was brought to the emergency department after being found at home in an altered mental state after a suspected drug overdose. As per his family members, he had no significant past medical history. Vital signs included a blood pressure of 170/90 mm Hg, heart rate of 120 bpm and respiratory rate of 20/min. He responded to painful stimuli with no purposeful movements of all extremities, had brisk reflexes in both upper and lower extremities, and his pupils were bilaterally dilated with intact light reflex. The Glasgow coma scale was calculated as 9 out of 15. Urine toxicology screen was positive for cocaine, cannabis and phencyclidine. Computed tomography (CT) scan of the brain showed no acute intracranial abnormalities. Laboratory studies found him to be in acute renal failure with rhabdomyolysis and he had a positive anion gap metabolic acidosis. Aggressive hydration with intravenous (*i.v.*) fluids including sodium bicarbonate was started. About an hour after arrival, the patient had a generalised tonic-clonic seizure of two minutes' duration. He was given *i.v.* lorazepam after which the seizure subsided; however, he continued to have recurrent tonic-clonic seizures which needed repeated doses of lorazepam and a fosphenytoin infusion as well. A decision was made to intubate the patient for airway protection and to start propofol. The blood pressure and heart rate stabilised to 110/70 mm Hg and 110 bpm, respectively. Although the patient was haemodynamically stable, the heart rhythm started going back and forth between sinus tachycardia and a wide complex rhythm that alternated between a LBBB type of wide complex tachy-

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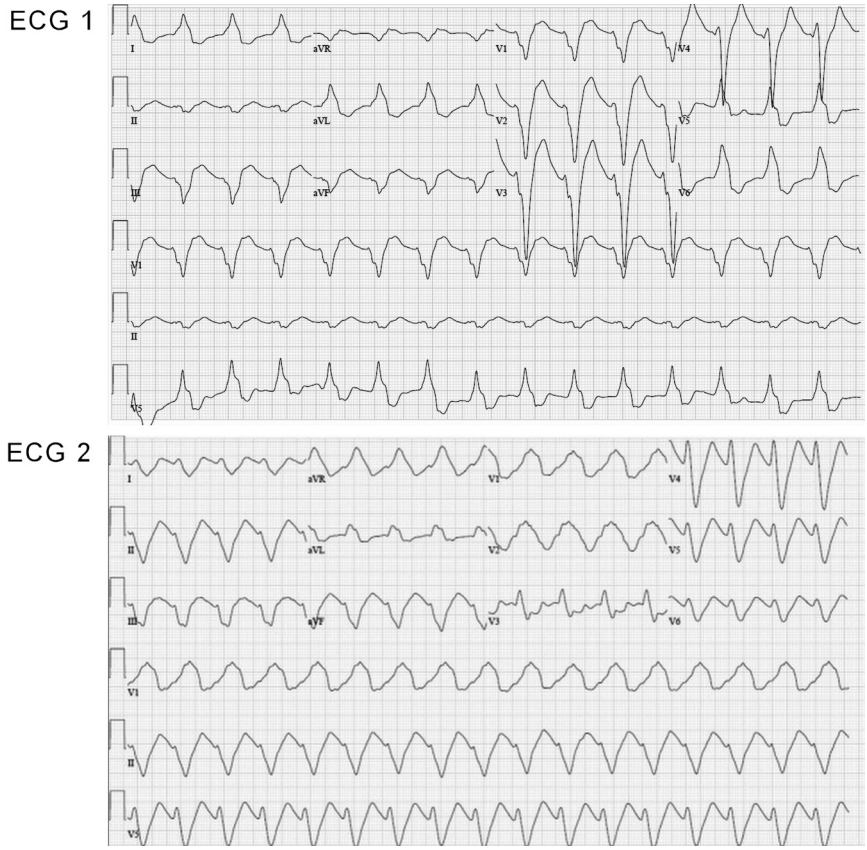

Figure 1. ECG 1 showing accelerated idioventricular rhythm with left bundle branch block morphology; ECG 2 showing accelerated idioventricular rhythm with right bundle branch block morphology

cardia to a RBBB type (Fig. 1). During periods of sinus rhythm, the QTc interval was prolonged at 527 ms. The addition of amiodarone infusion to bicarbonate seemed to have no effect on this abnormal rhythm. Due to the failure of all conservative measures to control the arrhythmias, it was decided to treat the patient with lipid emulsion because the literature had shown evidence of the usefulness of this therapy in arrhythmias from cocaine overdose [1]. 20% lipid emulsion was administered as a 150 mL (1.5 mL/kg) bolus *i.v.* over 2–3 min followed by 250 mL (0.25 mL/kg/min) over 10 min. So the patient received a total of 500 mL on the first day. However, the patient failed to respond to treatment and continued to have wide complex arrhythmias on electrocardiogram (ECG) and continuous seizure activity on electroencephalogram as well. Investigations for underlying causes including electrolyte imbalances and cardiac ischaemia were negative. The lipid emulsion therapy was repeated again the next day with a 20% lipid emulsion bolus of 150 mL (1.5 mL/kg) *i.v.*, followed by continuous infusion of 750 mL (0.25/kg/min) for 30 min. Unfortunately, the patient remained critically ill. He needed continuous veno-venous haemodialysis for intractable oliguric acute kidney injury. He subsequently developed severe sepis secondary to MRSA pneumonia with multiple organ dysfunction. Serial neurological assessments revealed the absence of cortical function with preservation of brainstem function. On the eighth day of his hospital stay, the patient again developed wide complex tachycardia after which he became haemodynamically unstable and died after 35 min of unsuccessful resuscitation.

DISCUSSION

Clinical studies have shown wide QRS complex arrhythmias to occur in 0–6% of patients with cocaine overdose [3, 4]. There are also a few case reports on wide complex arrhythmias from cocaine toxicity [1, 5, 6]. The commonest abnormalities in all the above studies were RBBB, non-specific intraventricular conduction delay, a new Brugada pattern, and accelerated idioventricular rhythm. On analysis of the wide complex rhythm seen in the ECG of our patient, using the Brugada criteria, the most likely explanation was accelerated idioventricular rhythm or slow ventricular tachycardia [7]. In ECG 1 with a LBBB morphology lead V_1 showed a notched downstroke of the S wave and a duration from the onset of the QRS complex to the nadir of the S wave of > 60 ms [8]. Similarly in EKG 2 with RBBB morphology a monophasic R wave was present in lead V_1 [9, 10].

Cocaine has a direct effect on cardiac ion channels including inhibition of voltage gated sodium channels thus slowing impulse conduction and prolonging the QRS interval in a manner similar to class I antiarrhythmics [11, 12]. This forms the basis of treatment with sodium bicarbonate similar to patients with tricyclic antidepressant or Class Ia antiarrhythmic overdose. Cocaine also exerts a blockade on the potassium channels thus impairing repolarisation leading to a prolonged QTc interval on surface ECG which was seen in our patient at presentation when he was in sinus rhythm with a narrow QRS. Concomitant intoxication with phencyclidine and cannabis may have contributed to the changes seen in our patient; however, to the best of our knowledge, there is little literature on reported ventricular arrhythmias caused by these agents.

Treatment options for wide complex dysrhythmias in cocaine overdose are largely supportive and symptomatic in nature. No specific antidote has yet been described. Sodium bicarbonate and lidocaine have been used in the past to suppress these arrhythmias, with variable success [1]. The use of amiodarone in this clinical scenario is a matter of debate with no conclusive evidence. A recent animal study failed to demonstrate any mortality benefit of amiodarone pretreatment before cocaine exposure in mice [13]. Many case reports have shown the successful use of lipid emulsion as an antidote for cardiovascular toxicities from local anaesthetic overdose [14, 15]. A few case reports have also showed the efficacy of lipid emulsion as an antidote for some antidepressants, lamotrigine, calcium channel blockers and beta-blockers [16, 17]. The common denominator of all these substances is that they are lipid soluble.

Recently there have been reports describing intravenous lipid as an effective treatment for cardiovascular complications associated with cocaine toxicity [2]. However, we did not have a successful outcome with the use of intravenous lipids: there could be many reasons for this. The profound metabolic and toxic abnormalities present in our patient could be a confounding factor for therapeutic effect of the lipid emulsion. There are limited studies on the usefulness of intravenous lipids in cocaine-associated arrhythmias and hence there is a lack of data on pharmacodynamics like effective dose or toxicity. Thus more data on the use of lipid emulsion in cocaine-induced arrhythmias is needed before any guidelines can be formulated; this is why we would like to report our case in the literature, despite a negative outcome.

CONCLUSIONS

Physicians should be aware of wide QRS complex arrhythmias with cocaine toxicity, and the need to differentiate rapid idioventricular rhythms or supraventricular tachycardias with alternating bundle branch block aberrancy. Management of these arrhythmias can be challenging. There is some evidence on the role of intravenous lipids, but more studies are necessary before this therapy can be endorsed.

Conflict of interest: *none declared*

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