Complete heart block following intentional carbamate ingestion

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Organophosphates and carbamate compounds are acetylcholinesterase inhibitors used as agricultural insecticides and represent a common cause of cholinergic toxicity. Cardiac manifestations of organophosphate and carbamate toxicity are described primarily from reports of organophosphate exposure and include sinus bradycardia, prolonged PR interval, sinus tachycardia, prolonged corrected QT interval and ventricular arrhythmias. Complete atrioventricular block has rarely been reported with insecticide poisonings. A case of complete heart block following carbamate ingestion is described and the importance of extended cardiac monitoring in these patients is emphasized.

Key Words: Carbamate; Carbaryl; Complete heart block; Overdose; Poisoning

Organophosphates and carbamate compounds are agricultural insecticides that inhibit acetylcholinesterase (AChE) and can cause cholinergic toxicity following all routes of exposure (1). Primary manifestations are due to effects on the neuromuscular junction, and the autonomic and central nervous systems. The predominant clinical features of acute cholinergic toxicity include salivation, lacrimation, nausea, vomiting, diarrhea, diaphoresis, miosis and bradycardia (1).

Each year, more than 6000 cases of pesticide poisoning are reported in Canada (2). The predominant cardiac effect is sinus bradycardia, which occurs less frequently following carbamate poisoning than organophosphate poisoning (1,3). Other cardiac manifestations include sinus tachycardia, hypertension, hypotension and noncardiogenic pulmonary edema (1,4). Electrocardiogram (ECG) abnormalities include prolonged corrected QT (QTc) interval, ST-T changes, prolonged PR interval, atrial fibrillation, ventricular tachycardia and fibrillation (3-5). High-grade atrioventricular (AV) block has rarely been described.

CASE PRESENTATION

A 33-year-old man presented to the emergency department with dizziness, myalgia, vomiting, increased salivation and diaphoresis. Two hours earlier, he intentionally ingested 30 mL to 45 mL of a pesticide containing 22.5% carbaryl (1-naphthyl methylcarbamate) in a suicide attempt. No additional medications, drugs or alcohol were consumed at the time. The patient denied any significant medical history and was on no regular medications. At presentation, he appeared pale and diaphoretic. His blood pressure was 158/97 mmHg and his heart rate was 95 beats/min and regular, with an oxygen saturation of 98%. His pupils were 3 mm to 4 mm in diameter and reacted appropriately to light. The initial ECG showed sinus tachycardia at 105 beats/min, a normal axis, a PR interval of 172 ms, a QRS duration of 116 ms (incomplete right bundle branch block) and a QTc interval of 494 ms

Un blocage cardiaque complet après l'ingestion intentionnelle de carbamate

Les organophosphates et les composés du carbamate sont des inhibiteurs de l'acétylcholinestérase utilisés comme insecticides en agriculture. Ils constituent une cause courante de toxicité cholinergique. Les manifestations cardiaques de la toxicité de l'organophosphate et du carbamate sont surtout décrites dans des rapports d'exposition aux organophosphates et incluent la bradycardie sinusale, un espace auriculoventriculaire prolongé, la tachycardie sinusale, un intervalle Q-T corrigé prolongé et des arythmies ventriculaires. Il est rare qu'on rende compte d'un blocage auriculoventriculaire complet causé par un empoisonnement aux insecticides. On décrit un cas de blocage cardiaque complet après l'ingestion de carbamate et on souligne l'importance d'une surveillance cardiaque prolongée chez ces patients.

(Figure 1). Bloodwork demonstrated mild hypokalemia (3.3 mmol/L), hyperglycemia (13.3 mmol/L) and leukocytosis ($26.1 \times 10^9/L$). Toxicological screens for acetaminophen and salicylates were negative. Telemetry and pulse oximetry were initiated and intravenous access was established.

Vomiting and diaphoresis resolved during the first few hours of observation. However, 11 h following exposure, the patient experienced two episodes of symptomatic bradycardia due to complete heart block. No QRS or QT interval prolongation was noted. The rhythm strip showed complete heart block with a pause longer than 10 s (Figure 2). Atropine (0.5 mg intravenously) was administered and conducted sinus rhythm was restored. The patient was transferred to the cardiac care unit for further monitoring. There were no further episodes of bradycardia. All laboratory abnormalities normalized before discharge.

DISCUSSION

Organophosphate and carbamate exposure produces clinical manifestations of cholinergic excess. Cardiac complications commonly occur in association with poisoning and are described primarily as a result of organophosphate exposure (1,3-5). Bradycardia develops secondary to augmented vagal influence that shortens the effective refractory period of atrial myocytes and increases the refractory period and conduction time of the sinoatrial and AV nodes (1). Carbamate insecticides cause a reversible inhibition of the AChE enzyme compared with the organophosphate insecticides. With organophosphate insecticide poisonings, generation of endogenous AChE enzyme is often required to overcome the effects of the poisoning. As a result, carbamate poisonings tend to resolve sooner and are associated with less morbidity and mortality. As well, blood cholinesterase activity is not useful in the diagnosis of carbamate poisoning (1).

The current case is an unusual presentation of complete AV block due to carbamate poisoning, which underscores the importance of

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Figure 1) A 12-lead electrocardiogram on admission showing sinus tachycardia at 105 beats/min, a normal axis, a PR interval of 172 ms, a QRS duration of 116 ms (incomplete right bundle branch block) and a corrected QT interval of 494 ms

Figure 2) Cardiac monitor rhythm strip showing cardiac activity before (A) and during (B and C) complete heart block with a pause longer than 10 s

extended cardiac monitoring in all cases of suspected or confirmed carbamate exposure. Rarely described in the literature, complete heart block was reported in three of 168 patients exposed to organophosphates or carbamates (3). Large doses are associated with more marked bradycardia and this may underlie the severity of conduction abnormality observed in our case. Our patient ingested approximately 10 g of carbaryl (approximately 140 mg/kg).

The patient's initial ECG revealed a prolonged QTc interval, which resolved spontaneously. Prolongation of the QT interval is known to be associated with carbamate and organophosphate toxicity, and poses a significant risk of arrhythmia (1-5). It is the most frequent and dangerous ECG change resulting from exposure to drugs.

In the present case, significant nausea and vomiting occurred within 2 h of carbamate ingestion. Complete heart block occurred approximately 11 h postexposure, following resolution of noncardiac cholinergic manifestations. This suggests that the presence of other systemic effects may not predict the subsequent development of cardiac complications including complete heart block. In addition, the delayed development of complete heart block suggests that cardiac monitoring should be continued for at least 24 h following exposure.

Leukocytosis, hyperglycemia and hypokalemia have been reported following carbamate and/or organophosphate poisoning (1,4).

Management of oral cholinergic toxicity includes maintaining the patient's airway and improving oxygenation (1). Activated charcoal or gastric lavage may be considered in patients presenting within 1 h of

ingestion. Significant vomiting and loss of consciousness preclude the use of activated charcoal and gastric lavage.

In the current case, atropine was administered during complete heart block, after which, conducted sinus rhythm was established.

CONCLUSION

Carbamate ingestion can produce significant cardiac conduction abnormalities including complete heart block. These abnormalities can occur after other systemic manifestations of cholinergic excess have resolved. Patients with suspected carbamate exposure should be observed closely for at least 24 h in an acute care setting with cardiac monitoring and access to atropine and/or external pacing.

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