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A Case Report

Fatal Myocardial Rupture Following Imidacloprid Poisoning: A Case Report

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Article Info

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Abstract:

Imidacloprid is generally considered to be relatively safer than other insecticides, such as organophosphorus, carbamates, and organochlorines. A 32-year-old male had a history of consumption of Imidacloprid insecticide and alcohol, which resulted in myocarditis, myocardial rupture and tamponade, as identified during the postmortem examination. Reported cases of Imidacloprid poisoning usually present with mild gastrointestinal and neurological symptoms. There is growing evidence that Imidacloprid poisoning may cause damage to cardiac, renal, and other organs. However, this incident underscores the rare presentation of grave cardiac features following its consumption. This case serves to highlight the uncommon manifestations of Imidacloprid poisoning, with the goal of raising awareness, encouraging better investigative and treatment strategies for this type of poisoning.

Keywords*:* Imidacloprid, Poisoning, Myocarditis, Myocardial Rupture, Cardiac tamponade.

Introduction

Imidacloprid [1-(6-chloro-3-pyridylmethyl)-N-nitroimidazolidin-2-ylideneamine] is a type of insecticide from the emerging class of chloronicotinyl neonicotinoid compounds. It is generally viewed as being safer than most other insecticides (organophosphorus, carbamates, and organochlorines).[1] Reported cases of Imidacloprid poisoning typically show mild gastrointestinal and neurological symptoms along with neurological sequelae, acute kidney injury (due to rhabdomyolysis), ischemic and metabolic encephalopathy, ventricular fibrillation, multi-organ failure, and death.[2]

Case Report

A 32-year-old male was brought to KMCRI Hospital, Hubballi, with alleged history of consumption of Imidacloprid insecticide,under the influence of alcohol.The patient was conscious and oriented upon arrival at the hospital. However, blood pressure was recorded as 80/60 mm of Hg, the patient had tachycardia and was maintaining a saturation of 97% on room air. During the hospitalization, the patient gradually deteriorated in spite of the efforts to maintain vital signs. The pupils started to constrict, the sensorium deteriorated and GCS score was recorded as E1V1M1 on the second day. The blood picture revealed neutrophilic

leucocytosis. The patient continued to have tachycardia and ended up on inotropic support. The patient also continued to have hypokalaemiasince the beginning and exhibited persistent ventricular tachycardia, along with elevated levels of CK-MB and Troponin-I. The ECG charts recorded ventricular tachycardia, absence of clear P waves and narrow QRS complexes (Fig 1 a &b). Clinically the case wasdiagnosed as "Alcohol use disorder, Imidacloprid 17.8 SL compound consumption, Toxin induced myocarditis and in Cardiogenic shock".He was treated for 2.5 days and did not respond to the treatment and passed away. The body was shifted to the mortuary and the Police registered this case under section 174 CrPC.

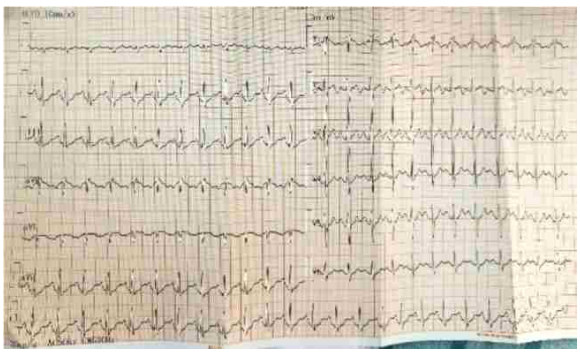

Figure 1(a)

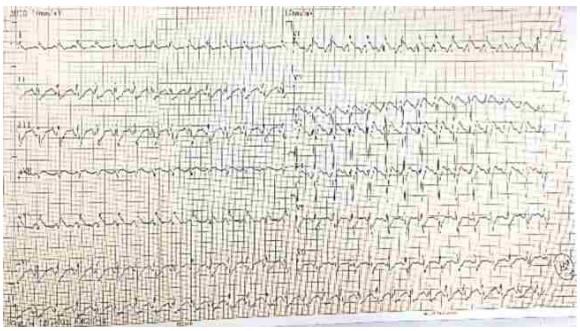

Figure 1(b)

The deceased was of moderate build and well-nourished, with fluid leaking from both nostrils. No apparent external injuries were present over the body. All internal organs were intact and congested. The stomach held 150mL of dark green fluid, and the mucosa appeared congested. The scalp and skull were intact, while the meninges and brain were both intact and congested. The pericardium contained a blood clot weighing 250 grams (Fig 2 a, b, c&d), with the right atrium showing a point of rupture (Fig 5&6).

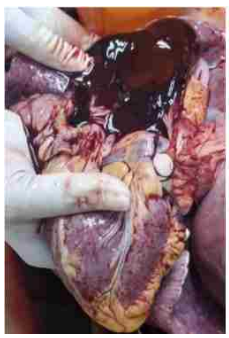

Figure 2(a)

Figure 2(b)

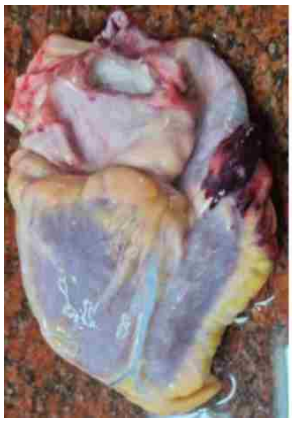

Figure 2(c)

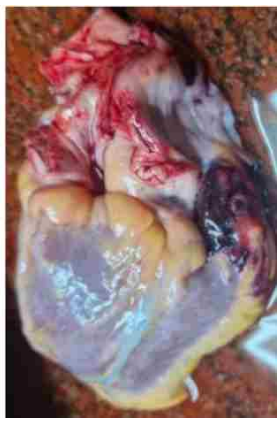

Figure 2(d)

Routine viscera and blood preserved and subjected for chemical analysis, which later turned out to be positive for Imidacloprid insecticide. The heart was subjected to histopathological examination.

Epicardium shows large areas of haemorrhage and adjacent myocardium shows hypertrophic changes (Fig 3a). Coronaries and aorta show atherosclerotic changes (Fig 3b). Blackish area over right atrium and right atrioventricular junction shows large areas of haemorrhage with hypertrophic changes (Fig 3c) and thrombosed microvessels.

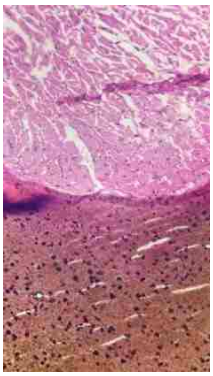

Figure 3(a)

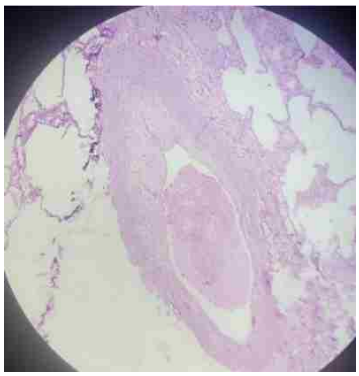

Figure 3(b)

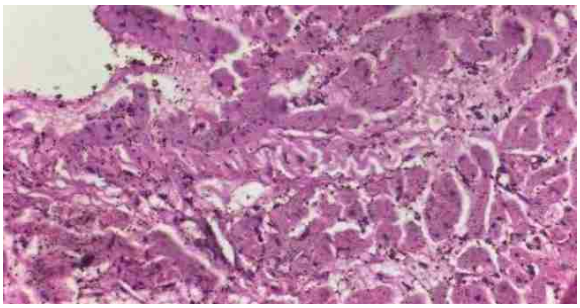

Figure 3(c)

Accordingly, the cause of death was finalized as "Death due to cardiac rupture due to myocarditis as a consequence of consumption of substance containing Imidacloprid".

Discussion

Imidacloprid, a neonicotinoid insecticide, is widely used due to its relative safety profile compared to older classes such as organophosphates and carbamates.[3] It acts as an agonist at the nicotinic acetylcholine receptors (nAChRs) in the central nervous system of insects, leading to paralysis and death. While its affinity for mammalian receptors is lower, systemic toxicity has been documented in humans, especially following intentional oral ingestion.[4] Reported cases have documented ventricular fibrillation and tachycardia within approximately one hour of acute imidacloprid ingestion, suggestive of its potential to induce severe cardiotoxic effects.[5] In this case, a 32-year-old male presented with intentional ingestion of Imidacloprid (17.8% SL), reportedly under the influence of alcohol. In spiteof supportive treatment over 2.5 days, the patient succumbed to complications arising from persistent ventricular tachycardia, elevated cardiac biomarkers (CK-MB, Troponin-I), and ultimately cardiogenic shock. While most reported cases of Imidacloprid poisoning involve mild gastrointestinal or neurological symptoms.[6] rare but serious cardiovascular effects—including arrhythmias, myocarditis, and myocardial infarction—have also been reported.[7] The persistent ventricular tachycardia and elevated cardiac enzymes in this case strongly suggest acute myocardial injury.

Histopathological findings revealed epicardial haemorrhages, hypertrophic changes, thrombosed microvessels, and an insignificant obstruction of the coronaries by atherosclerosmatous plaques. These are consistent with toxin-induced myocarditis. Myocarditis can result in weakening of myocardial walls, which, under stress (e.g., tachyarrhythmias or hypertensive crises), may lead to cardiac rupture—a rare but well-documented complication.[8,9] Notably, a rupture of the right atrium was identified on autopsy, accompanied by a 250g pericardial clot, indicating haemopericardium and tamponade as the terminal event. This is highly unusual in Imidacloprid poisoning and represents a potentially novel fatal manifestation of the toxin's cardiotoxic profile.

Concurrent alcohol intoxication may have potentiated the toxic effects.[9] Ethanol can independently cause arrhythmias, depress myocardial function, and increase gut absorption of co-ingested toxins.[10] The combination of Imidacloprid and alcohol likely contributed synergistically to the severity of the presentation. Detection of Imidacloprid in both blood and viscera confirms systemic exposure. The concentration, although not quantified in the provided report, was sufficient to cause significant systemic and cardiac toxicity.

Conclusion

This case highlights the evolving clinical significance of Imidacloprid poisoning, which was previously regarded as trivial due to its mild manifestations. A novel observation from this case report is the occurrence of Imidacloprid (poison)-induced myocarditis, resulting in cardiac rupture and fatality. This case underscores

the potential for severe and fatal cardiotoxicity following Imidacloprid ingestion, especially when combined with alcohol. Here, close observation and monitoring should be prioritized in patients with a large intake or exhibiting warning signs such as cardiovascular and central nervous system effects. Clinicians should be aware of rare but life-threatening complications such as toxin-induced myocarditis and cardiac rupture. Early recognition, cardiac monitoring, and aggressive supportive therapy—including management of arrhythmias and cardiac dysfunction—are essential.

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