



A Case Report

Autopsy and Histopathological Insights in Fatal Yellow Oleander Poisoning: A Case Series with Clinicopathological Correlation

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Abstracts

Background: Yellow oleander poisoning (YOP) is a common cause of fatal plant poisoning in India due to its easy availability. While its clinical effects are well described in the literature, detailed autopsy and histopathological findings in fatal cases are less frequently reported.

Material and Methods: The cases for the study were collected after obtaining ethical permission and consent from patients admitted to the emergency department with a history of YOP and subsequently autopsied on death with ancillary histopathology and toxicology examination.

Results: This case series describes three fatal cases of YOP with emphasis on autopsy and histopathological findings to better understand the underlying pathophysiology of death. All three patients had taken crushed parts of the plant on an empty stomach and presented within 3-6 hours of ingestion, with severe

bradycardia and hypotension suggestive of marked cardiotoxicity. Clinical severity at admission was assessed using the Poisoning Severity Score (PSS), and all three cases were classified as Grade 4, indicating extreme toxicity. All patients succumbed within a short duration of admission, despite medical intervention.

On Post-mortem examination, asphyxia signs such as cyanosis and facial congestion were noted. Internally, generalised visceral congestion was found in all cases. In the heart, haemorrhages were noticed in two cases, and the mucosa of the stomach was congested with patches of submucosal haemorrhage. Histopathological examination revealed mainly non-specific changes such as vascular congestion and inflammation in various organs. However, myocardial sections revealed subepicardial and intramyocardial haemorrhages consistent with direct toxic injury.

Conclusion: The YOP autopsy and histopathology findings are not pathognomonic, but the consistent cardiac involvement, when taken in conjunction with the severe clinical grading, supports the cardiotoxic action of cardiac glycosides and points out the value of clinicopathological correlation in such cases.

Keywords- Yellow oleander; Histopathological examination; Postmortem examination; Myocardial haemorrhage; ECG.

Introduction

Yellow oleander poisoning (YOP) is a major toxicological concern in many parts of the world, especially in regions where it is widely distributed and easily accessible, resulting from ingestion of the plant and its derivatives (Fig. 1). Since ancient times, this plant has been recognized to be toxic in India, and is referred to as *Kajamaraka*, or "the

plant that kills the horse".[1] The dangerous effects of the plant are primarily attributed to potent cardiac glycosides (Oleandrin, Thevetin, neriifolin, etc.), with the highest concentrations in the seeds, the part most often ingested in intentional poisoning.[2,3] The toxic effects primarily involve the cardiovascular system, with disturbances in cardiac rhythm and function. YOP remains a significant yet underreported toxicological problem in many parts of South Asia. Because of its regional burden, limited awareness, and lack of easily available specific treatment, it has been considered a neglected tropical disease in this region.[4] Although the clinical manifestations of poisoning are well recognised, detailed pathological observations in fatal cases are comparatively scarce. Therefore, careful evaluation of autopsy and histopathological findings is important for understanding the toxic effects and for improving medico-legal diagnosis.

Material and Methods

The cases with a history of YOP of all ages and sex admitted to the emergency department of a tertiary care hospital of eastern India, and subsequently autopsied on death with ancillary histopathology and toxicology examination, were considered after obtaining the Institute's Ethical

Committee permission (IEC/AIIMS BBSR/PG Thesis/2021-22/113). Consent from the legally acceptable representatives was obtained. The relevant demographic details, the history of ingested parts, the quantity, symptoms, clinical features, admission details, hospitalisation stay, ECG findings, poison severity score at admission (PSS), and autopsy details with histopathological and toxicological report data were considered and entered in a tabular format for comparison and analysis.

Case Details

Three fatal cases of YOP were examined in the present study (Table 1). As per the history, all of them had taken crushed parts of the plant through oral ingestion on an empty stomach. ECG in all three cases demonstrated sinus bradycardia with variable nonspecific ST-T wave abnormalities, including anterior lead T-wave inversion, tall peaked T waves, mild anterior ST-segment elevation-like changes, and sinus arrhythmia (Fig.2). Their condition remained unstable throughout admission, and they were clinically categorised under severe poisoning (PSS Grade 4). Despite supportive management and emergency treatment, all three patients succumbed within a few hours of hospital admission.

Table 1 : Details of the three cases studied

Sr. no	Age (years)	Sex	Part ingested	Amount ingested	Time between exposure & presentation (hours)	Clinical profile at time of presentation	Hospital stays	PSS
1	60	F	Seeds & Fruits	8 & 4	3-6	Unconscious, severe bradycardia and hypotension	<1 day	4
2	18	F	Seeds	11	3-6	Nausea, Vomiting, severe bradycardia and hypotension	<1 day	4
3	36	M	Fruits	5	3-6	Nausea, Vomiting, severe bradycardia and hypotension	<1 day	4

Post-mortem findings

Gross examination findings

External examination revealed features of asphyxia, such as facial congestion and cyanosis of the nail beds. No external injuries were observed. In two cases, there were petechial haemorrhages over the heart, particularly over the left ventricular apical region and the posteroinferior wall of the left ventricle (Figs 3 and 4). The gastric mucosa was congested, with some focal areas of submucosal haemorrhage (Fig 5). There was also congestion to varying degrees in the lungs, liver, kidneys, and brain, but no distinct gross pathological lesion was noted (Fig 6). Viscera samples were collected for toxicological analysis, and representative tissue sections of major organs were taken for microscopic examination.

Toxicological reports

The toxicological analysis report came out to be positive for cardiac glycosides on Thin-Layer Chromatography (TLC) analysis, which was consistent with YOP.

Histopathological findings

On histopathological examination, the heart showed haemorrhage beneath the epicardium (subepicardial haemorrhage) with vascular congestion and adjacent dense fibrocollagenous tissue (Fig. 7a) and wavy myocardial fibres with haemorrhage within the muscle fibres (intramyocardial haemorrhage) and focal separation of muscle bundles (Fig. 7b). Pulmonary sections revealed congestion of alveolar capillaries with focal interstitial oedema. Renal sections demonstrated marked vascular congestion with mild tubular epithelial degenerative changes and dilatation of glomerular capillaries (Fig 8a). In the stomach, congested mucosal and submucosal blood vessels with focal haemorrhagic areas and mild mononuclear inflammatory cell infiltrate were noted (Fig 8b). Hepatic sections demonstrated sinusoidal congestion without significant hepatocellular necrosis, and the brain also showed a variable degree of vascular congestion and mild perivascular oedema, but no characteristic microscopic findings (Fig. 9).

Discussion

YOP continues to be encountered frequently

in tropical countries like India, particularly in cases of deliberate self-poisoning. The principal cardiac glycosides, including thevetin and nerifolin, inhibit the Na^+/K^+ ATPase, leading to disturbances in cardiac conduction and contractility.[5,6] The severity and onset of poisoning depend on several factors, such as the amount consumed, the form in which it is ingested and the individual's susceptibility. Diagnosis is primarily based on history, clinical profile, and toxicological investigations, including fluorescence polarisation immunoassay (FPIA), digoxin immunoassay, and liquid chromatography-mass spectrometry (LC-MS/MS), which are capable of detecting cardiac glycosides in biological specimens.[7]

In the present study, all fatal cases had a history of ingestion of crushed plant material (fruits and seeds) on an empty stomach. This form of intake is known to accelerate toxin absorption and has been associated with more serious clinical outcomes in previous studies[5,6]. The amount consumed in these cases was also within or above the reported fatal range, as ingestion of 8-10 seeds has been documented to cause death.[8] Clinically, all patients presented with severe bradycardia and hypotension, and similar findings have been widely reported, where bradyarrhythmias and varying degrees of atrioventricular block are the most common life-threatening manifestations[9,10]. In this study, all fatal cases were classified as PSS Grade 4, indicating extreme toxicity and poor prognosis. Previous studies have also demonstrated a significant association between higher PSS grades and mortality, highlighting its usefulness in early prognostic assessment.[9]

The gross findings observed in the present study were largely non-specific and comprised generalised visceral congestion with features of terminal asphyxia, similar to previous autopsy reports in YOP.[10] The associated cardiac haemorrhages suggest myocardial involvement, while the gastric changes are likely related to the local irritant effect of the ingested toxin on the gastric mucosa. [10]

Histopathological examination demonstrated predominantly non-specific vascular congestion across multiple organs. The myocardial alterations observed in the present study are

suggestive of direct cardiotoxic injury, compounded by terminal hypoxic damage, and are comparable with findings described in previous autopsy studies [11]. The remaining organs showed mainly congestive and mild inflammatory changes, which are consistent with systemic hypoxia and circulatory failure.

The gross and microscopic findings by themselves were non-specific to YOP, as similar pathological findings may also be seen in several conditions, such as asphyxial deaths, myocardial infarction, myocarditis, septicaemia, shock, electrical fatalities, blunt cardiac trauma, and other cardiotoxic poisonings.[12] However, toxicological analysis of the preserved viscera samples was positive for oleander poison. When correlated with the clinical profile and severity of poisoning, they provided supportive evidence regarding the cause of death. In the present cases, cardiac involvement was a consistent observation, suggesting that death was most likely related to fatal disturbances in cardiac conduction and rhythm. Hence, pathological findings should always be interpreted along with clinical and toxicological evidence in suspected cases of YOP.

Conclusion

Yellow oleander is a cardiotoxic plant, and its poisoning causes severe conduction abnormalities and death. The study highlights the importance of clinicopathological correlation in medicolegal investigations of fatal YOP. While gross and histopathological findings are generally non-specific, their association with history, clinical profile and positive toxicological analysis supports the diagnosis and the cause of death in suspected cases of YOP.

Limitations

The study was conducted on a limited number of cases and lacked a quantitative estimation of cardiac glycosides in postmortem specimens. Advanced investigations, such as immunohistochemical and ultrastructural studies, were also not performed.

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Ethical approval

Approval was granted by the Ethics Committee of AIIMS Bhubaneswar, vide ref no IEC/AIIMS BBSR/PG Thesis/2021-22/113.

Conflict of interest

The authors declare no conflict of interest.

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Financial interests

The authors declare that they have no financial interests.

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Fig. 1 : *Thevetia peruviana* bearing green fruits and leaves

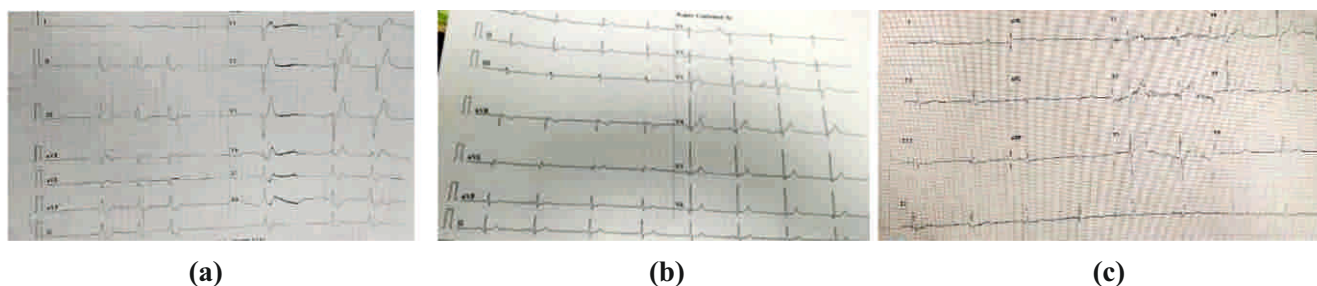


Figure 2 : demonstrates marked sinus bradycardia with mild 1st degree AV block and intraventricular conduction delay with T-wave abnormalities (a). Sinus bradycardia with T-wave inversion and mild nonspecific ST-T changes were noted (b &c), with sinus arrhythmia(b)



Figure 3 : Gross examination of the heart showing congestion and multiple petechiae haemorrhage over the posteroinferior wall of the left ventricle and left ventricular apical region (a & b) (black arrow)



Figure 4 : Examination of formalin fixed heart showing subepicardial and intramyocardial haemorrhage (a&b) respectively (black arrow).



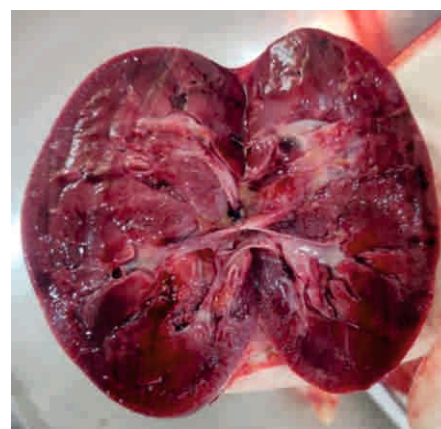
Figure 5 : Gross examination of stomach showing congested mucosa and submucosal hemorrhage



(a)



(b)



(c)

Figure 6 : Gross examination of the brain, liver and kidney showing congestion (a, b and c).

Histopathology specimen of Liver

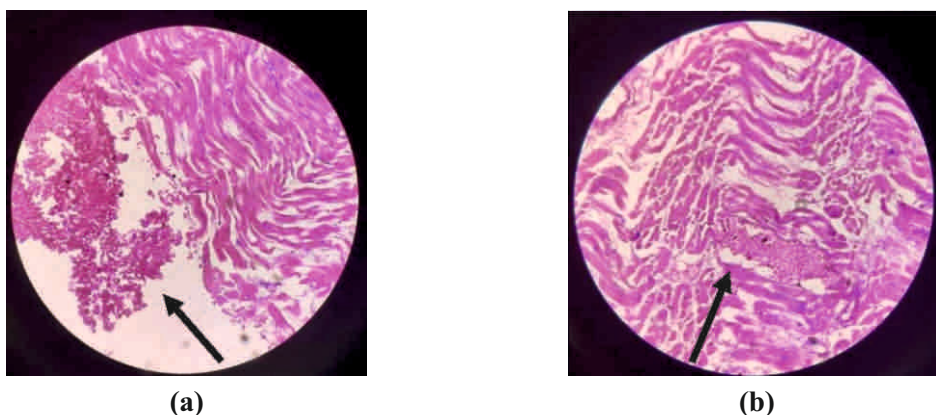


Figure 7 : Section from heart showing subepicardial haemorrhage (black arrow) with vascular congestion and adjacent dense fibrocollagenous tissue(a)and intramyocardial haemorrhage (black arrow) with wavy myocardial fibresand focal separation of muscle bundles (haematoxylin-eosin stain, original magnification x10).

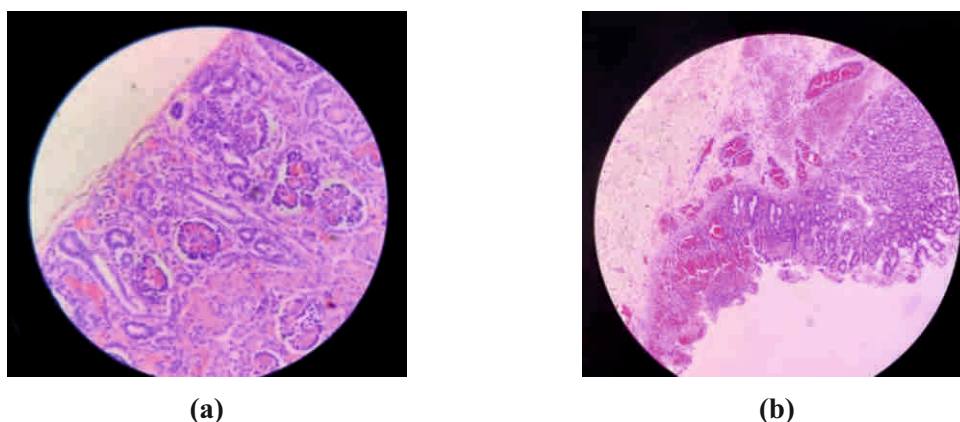


Figure 8 : Section fromKidney showing vascular congestion with mild tubular epithelial degenerative changes and dilatation of glomerular capillaries(a)and section from stomachshowingcongested mucosal and submucosal blood vessels with focal hemorrhagic areas and mild inflammatory cell infiltrate(b) (haematoxylin-eosin stain, original magnification x10).

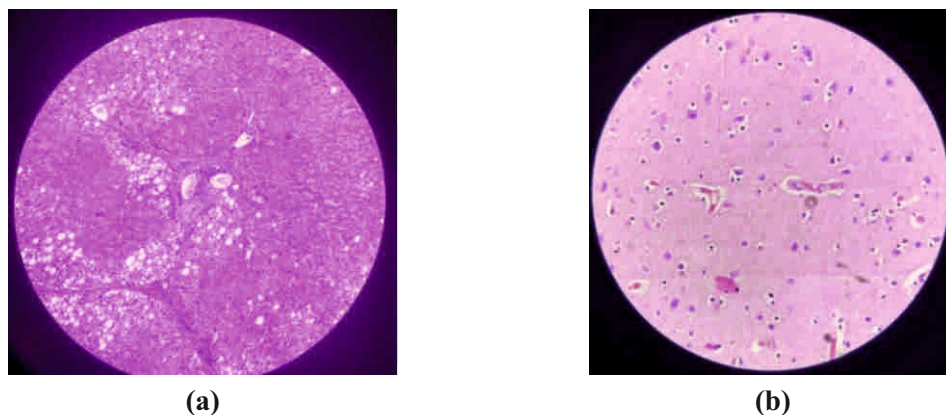


Figure 9 : Section from liver showinginflammation around the Porta hepatis and oedema along with fatty cells with vascular congestion (a) and section from brain showing vascular congestion and mild perivascular oedema (b) (Hematoxylin-eosin stain, original magnification x 20).
(haematoxylin-eosin stain, original magnification x10)

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