Case Study

Paraquat Poisoning with Negative Chemical Analysis: Forensic Value of Gross Autopsy Findings in the Pancreas

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How to cite this article: Elakkiyan S, B. Suresh Kumar Shetty, Mayur Sudhir Balbudhe et. al. Paraquat Poisoning with Negative Chemical Analysis: Forensic Value of Gross Autopsy Findings in the Pancreas. Indian Journal of Forensic Medicine and Toxicology/Volume 20 No. 2, April-June 2026.

Abstract

Introduction: Paraquat is a highly toxic herbicide commonly implicated in fatal poisonings, particularly in agricultural communities. Diagnosis of paraquat poisoning poses a challenge in forensic practice due to rapid tissue distribution, metabolism, and elimination, often resulting in negative toxicological findings. In such cases, autopsy and histopathological findings become crucial in establishing the cause of death. This study highlights the forensic importance of pancreatic haemorrhage as a potential indicator in paraquat poisoning with negative chemical analysis.

Background: Paraquat toxicity primarily affects the lungs, kidneys, and gastrointestinal tract, leading to progressive multiorgan failure. However, pancreatic involvement is rarely reported. Additionally, instability of paraquat in biological samples often leads to negative viscera analysis, complicating medicolegal interpretation. Hence, reliance on clinical history and pathological findings becomes essential for accurate diagnosis.

Methods: A medicolegal autopsy was performed on a middle-aged male farmer with alleged ingestion of paraquat. Clinical history, laboratory investigations, gross autopsy findings, toxicological analysis, and histopathological examination were reviewed. Viscera, including stomach, intestine, liver, kidney, and blood, were preserved and sent for chemical analysis. Histopathological examination of major organs was conducted to identify microscopic changes.

Results: The patient developed severe vomiting, respiratory distress, metabolic acidosis, and acute kidney injury, leading to death on the third day following ingestion. Autopsy revealed congested lungs, haemorrhagic gastric mucosa, subcapsular hepatic haemorrhhages, fatty liver changes, and significant pancreatic haemorrhage. Toxicological analysis of viscera was negative for paraquat and other common poisons. Histopathology confirmed hepatic steatosis, acute tubular necrosis of kidneys, and pancreatic haemorrhage. Differential diagnoses including

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Submission date: January 23, 2026 **Acceptance date:** March 30 , 2026 **Published date: April 23, 2026**

organophosphate poisoning, septic shock, heavy metal poisoning, and primary pancreatitis were excluded based on clinical and pathological findings.

Conclusion: Paraquat poisoning should be suspected in cases of rapid multiorgan failure following herbicide ingestion, even when toxicological analysis is negative. Pancreatic haemorrhage may represent a rare but important forensic indicator of systemic paraquat toxicity. This case underscores the importance of integrating clinical history, autopsy findings, and histopathological examination in establishing the cause of death when chemical analysis is inconclusive.

Keywords: Paraquat poisoning, Negative toxicological analysis, Pancreatic haemorrhage, Forensic autopsy, Multiorgan failure, Herbicide poisoning, Histopathological findings

Background

Paraquat is a very toxic herbicide that is one of the major reasons of fatal poisoning in the agricultural society, especially in third world countries. Its high rate of absorption and tissue affinity particularly in the lungs, kidney and gastrointestinal tract causes multi-organ failure and progressive pulmonary fibrosis being the characteristic one. In forensic samples paraquat is instable and is rapidly metabolized thus making it difficult to detect. The adverse toxicological outcomes tend to make the medicolegal investigations rather difficult, which requires relying on the clinical history, autopsy, and histopathological data. By highlighting the diagnostic complexity of paraquat poisoning and pancreatic haemorrhage as an unexplored manifestation of systemic toxicity, the case suggests research value to the forensic literature by informing the integrative approach to diagnosis in failure of chemical analyses.

Case Presentation

Middle aged man farmer reported to the emergency department with acute respiratory distress and severe vomiting. According to family members, he had deliberately ingested a herbicide which was suspected to be paraquat several hours before. His past medical history did not record any significant illnesses and neither was the social or family history remarkable in contributory factors. On observation, he was found to have yellowish scleral discolouration, bluish nail beds and reddish brown, parchmentized lips. No extrinsic traumas. His condition could not be supported; despite the intensive supportive care, fluids, and monitoring, his health condition deteriorated quickly, and his respiratory distress and the metabolic acidosis worsened. He suffered acute kidney disease and died on day three after taking it. A medicolegal autopsy of the case was requested because of the suspicious character of the ingestion.

Investigations

Medicolegal autopsy was carried out. On the external inspection, there was some yellowish scleral discoloration, bluish nail-beds, and parchmentized lip lesions. The thorax contained 150 mL of a straw-coloured pleural fluid, and filled lungs (right: 810 g, left: 410 g). The abdomen showed a reddish gastric fluid of abnormal odour, bleeding gastric mucosa, and subcapsular hepatic haemorrhages and changes in fatty liver. On cut section, significant haemorrhage of the pancreas was found and it looked soft and lobulated (Figures B, C). Viscera (stomach, small intestine, liver, gall bladder, kidneys and blood) was stored to be used in toxicological studies and this was found negative of common toxins such as paraquat. Histopathological changes proved the presence of hepatic steatosis, acute tubular necrosis in the kidney and pancreatic haemorrhage. Figures A, B and C depict subcapsular liver haemorrhages, pancreatic haemorrhage respectively. Figure D is a suspected bottle of paraquat (original, created by author).

Differential Diagnosis

Other toxic ingestions (e.g. organophosphates, heavy metals) and non-toxic causes of multiorgan failure were part of the differential diagnosis (e.g. septic shock or acute pancreatitis). Toxicological screening was negative, excluding organophosphate poisoning as the patient did not have cholinergic symptoms. The clinical presentation was less likely to cause heavy metal poisoning since Mees lines were not found. Septic shock was eliminated because of the lack of fever or foci. Acute pancreatitis was also taken into consideration but ruled as

secondary because pancreatic haemorrhage was in line with systemic paraquat toxicity, not with primary pancreatic disease. The blood investigations at admission were Haemoglobin 14.7g/dl, Total leucocyte count 27200mm^3, Creatinine 4.81mg/dl, Potassium 4.12mmol/L, APTT 35.7. Clinical history, acute progressive deterioration, and autopsy (haemorrhagic pancreas, congested lungs, and acute tubular necrosis) were highly pointing toward paraquat poisoning.

Treatment

The patient was provided with supportive care in the intensive care unit which included intravenous fluid, oxygen therapy and observation of metabolic acidosis and renal function. No particular antidote to paraquat was used, since there is none. Haemodialysis was taken into consideration and not started because of the rapid clinical deterioration. The treatment was aimed at stabilizing the patient, but the multiorgan failure went on and the patient died.

Outcome and Follow-Up

On the third day after the ingestion, the patient died, which was a result of metabolic acidosis and acute renal failure, which is characteristic of paraquat toxicity. Follow up data were not available because the patient had passed away. The autopsy and histopathological results proved to be important in proving the cause of death which was backed by family witness that he ingested herbicides. The patient did not survive making it impossible to apply any form of surveillance or long-term monitoring. It was reported to the legal authorities, and the findings indicated paraquat poisoning despite negative.

Figure A

Figure B

Figure C

Figure D

Discussion

Paraquat poisoning poses a forensic problem because it rapidly accumulates and breaks down in the tissues which in most cases leads to negative toxicological results as the case is. The mechanism of Paraquat is that this drug causes reactive oxygen species, oxidative stress, cell necrosis, and multiorgan failure especially in lungs, kidney, and gastrointestinal tract [1,7]. In this case, pancreatic haemorrhage, which was a seldom-reported observation, was probably caused by vascular injury at the systemic level by oxidative stress [6]. Other similar cases have documented gastrointestinal erosions, acute tubular necrosis, and lung congestion whereas pancreatic involvement is less frequently reported [4,6]. The recent guidelines are focused on the importance of early decontamination and supportive care but there is still a low survival rate [2,10]. The negative toxicological results indicates that the usage of specialized tests (e.g., HPLC-MS or ELISA) and adequate storage of the sample is necessary to identify paraquat [5,12]. Under circumstances where chemical analysis is not able to confirm diagnosis, forensic pathologists should incorporate clinical and circumstantial evidence with pathological evidence.

Learning points/take home messages

Poisoning with paraquat should be considered in a situation where there is a rapid multiorgan failure after ingesting a herbicide that is negative in toxicological studies.

Pancreatic haemorrhage can be a new forensic indicator of systemic paraquat toxicity.

The extensive autopsy and histopathological analysis are essential to diagnosis where chemical analysis is not conclusive.

Specialized toxicological techniques (e.g., HPLC-MS, ELISA) and storage of the sample in favour of the detection of paraquat are required.

Medicolegal integration by means of a synthesis of clinical history, circumstantial evidence and autopsy findings are essential to accurate diagnosis.

Conflict of interest: authors declared no conflict of interest.

Source of funding: self-funded.

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