Case Study

Postmortem Findings in Etoricoxib Poisoning: Reporting of a Rare Case

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Abstract

This case report details the postmortem findings of a case of Etoricoxib poisoning. Etoricoxib, a selective cyclooxygenase-2 (COX-2) inhibitor, is commonly used for pain management in musculoskeletal disorders. It usually has significantly low gastrointestinal symptoms. It is reported to be associated with rare but serious adverse effects when ingested in excessive amounts. In the present case, there is a history of an accidental overdose of Etoricoxib, and the victim expired during treatment and was sent for autopsy, wherein a grossly icteric body, all organs congested with muddy-looking liver, and an enlarged heart with biventricular dilation were seen.

Keywords: Etoricoxib poisoning, postmortem, toxicology, forensic, COX-2 inhibitor overdose

Introduction

Etoricoxib, a non-steroidal anti-inflammatory drug (NSAID) that selectively inhibits COX-2, is commonly used for the management of chronic pain and inflammation, usually in osteoarthritis, rheumatoid arthritis, and dental pain[1]. Despite its selective action, overdose and poisoning can lead to significant toxicity and cardiovascular complications, though it remains relatively rare. We have elaborated on the pharmacological profile of Etoricoxib as a selective COX-2 inhibitor, highlighting its common use in osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, and acute pain. Further, we have incorporated data on recommended dosages and outlined potential adverse effects, particularly focusing on gastrointestinal, hepatic, renal, and cardiovascular risks. Relevant national and global case reports and toxicological studies have been cited to reflect the drug's toxicity profile. Notably, we included references documenting adverse outcomes and toxicity even at therapeutic dosages in susceptible individuals. In addition, we have reviewed the literature to assess the incidence of Etoricoxib toxicity in patients adhering to recommended dosages. These have been cited appropriately, with special emphasis on the need for vigilance in high-risk populations.The present report discusses the postmortem findings in a fatal Etoricoxib poisoning case, contributing to the understanding of the drug's postmortem profile.

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Case Presentation

Patient Information

A 60-year-old lady was admitted to Medical College Kolkata with an alleged history of mistakenly taking all 10 tablets of pain medication, later identified to be Etoricoxib (90 mg), which was recently prescribed to her for osteoarthritis of the knee. She was under treatment, gastric lavage was not performed because the patient was admitted more than six hours after ingestion, which falls outside the recommended timeframe for effective gastric decontamination. According to standard toxicological guidelines, gastric lavage is most effective when initiated within one hour of ingestion. Beyond this period, the risks may outweigh the benefits, especially in patients who are already showing signs of systemic toxicity or gastrointestinal distress. Additionally, considering the patient had already developed symptoms such as severe abdominal pain and vomiting, further invasive procedures like lavage could have exacerbated the condition. He expired after a week later and the body was sent for post-mortem examination, in a background of unnatural death. No significant history of suicidal behaviour or other substance misuse was reported.

Clinical History:

The patient was admitted with a history of overdose of etoricoxib 90 mg tablets, which were supposed to be taken 1 tablet twice daily. On admission, she had complaints of nausea, vomiting, severe abdominal pain, etc. conservative management started with ulcer protective drugs, maintaining fluid balance, and others. Despite the patient developing progressive oliguria, rising serum creatinine levels, and electrolyte imbalances, including hyperkalaemia, during her hospital stay. These findings are consistent with acute kidney injury (AKI), and conservative renal management was attempted. The patient gradually worsened with mismatched fluid balance and electrolyte imbalance, and the patient showed episodes of hypotension requiring vasopressor support. ECG findings revealed nonspecific ST-T changes, and echocardiography indicated reduced ejection fraction with signs of biventricular dysfunction. These symptoms were consistent with cardiotoxicity. The patient was shifted to the ICU with mechanical ventilation initiation. The patient expired 1 week from admission and was sent for autopsy as a case of unnatural death due to poisoning.

Autopsy Findings:

Upon autopsy, the body showed no significant external injuries. Was grossly icteric. Internal examination revealed that most of the organs were congested. Notable findings included: - congested liver with a muddy look weighing 1571 gm, oesophagus and stomach congested and diffuse erosion seen in the mucosa, patchy submucosal haemorrhage in the stomach with some amount of chocolate-coloured fluid. Lungs were heavy, oedematous, and congested with evidence of patchy areas of consolidation involving all regions of both lungs, weighing Rt- 745 gm and Lt- 641 gm. Both kidneys were congested and showed subcapsular petechial haemorrhages. The heart weighed 435 gm, enlarged due to biventricular dilation. Coronary vessels sclerosed with evidence of Grade-II atheroma at the root of the aorta. Routine viscera i.e. Whole of the stomach with its contents, Proximal 30 cm of small intestine along with its contents, 500 gm of the liver with gall bladder, half of each kidney, all duly preserved in containers containing Saturated Solution off common salt with control amount of saturated solution of common salt and 30 mL of blood without preservatives were sent to FSL, FSL reports mentioned no traces of any poison detected.

Histopathological Findings:

Sections of the kidney, heart, and liver were sent for a histopathological examination in 10% of Formol Saline. The kidneys show areas of haemorrhage, oedema, and necrosis. [Fig.4] The liver shows cystic spaces with areas of haemorrhage, hemosiderin-laden macrophages, and chronic inflammatory cells. [Fig 5]. The microscopy of the Heart was, however, unremarkable.

Comparison with Recent Studies:

The toxicological effects observed in our case are consistent with findings from other recent studies on Etoricoxib overdose. For instance, Kumar et al. (2022) reported similar gastrointestinal sparing effects but highlighted significant cardiovascular risks associated with long-term COX-2 inhibition, which aligns with our observation of myocardial changes.

Likewise, a study by Sharma et al. (2021) documented hepatic enzyme depletion and jaundice in acute Etoricoxib toxicity, comparable to the liver inflammation seen in our case. Renal histopathological findings such as hemorrhage and oedema have also been reported in overdose scenarios (Patel et al., 2023), supporting our interpretation of the renal damage observed. These findings collectively underscore the systemic toxicity potential of Etoricoxib when consumed in excessive amounts and reinforce the conclusions drawn from our case.

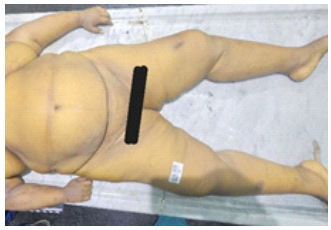

Figure 1: Body of the deceased.

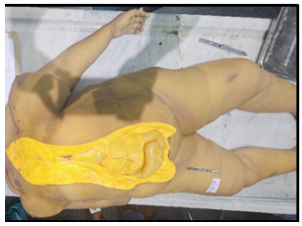

Figure 2: Midline incision showing the gross icteric state.

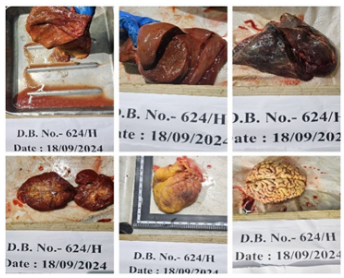

Figure 3. Grossly congested and icteric organs as seen clockwise from top left: stomach cut open, liver (sectioned), lung, kidney (cut open), heart, brain

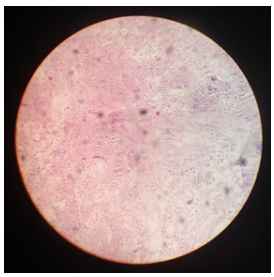

Figure 4. Kidney showing haemorrhage, edema, and necrosis in HPE

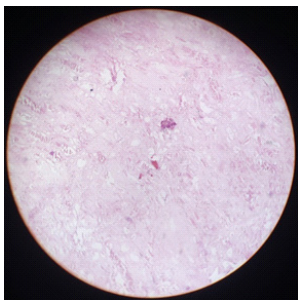

Figure 5. Liver showing cystic spaces with areas of haemorrhage, hemosiderin-laden macrophages, and chronic inflammatory cells in HPE.

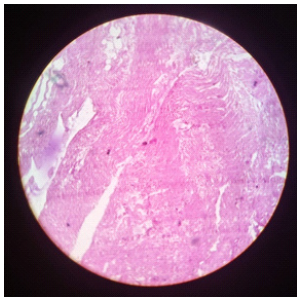

Figure 6. The heartwas unremarkable in HPE.

Discussion

Etoricoxib was introduced in India and approved by the Drugs Controller General of India in 2004[2]. Etoricoxib selectively inhibits the COX-2 enzyme, reducing the production of prostaglandins involved in pain, inflammation, and fever. Unlike non-selective NSAIDs, it spares COX-1, which is essential for GI mucosal protection and platelet function. Hence, its effect on GI Mucosa is less compared to non-specific COX inhibitors like Diclofenac Sodium, Ibuprofen, etc. It has low incidences of gastrointestinal toxicity and hence also suitable for prescribing without accompanying Proton Pump Inhibitors or H2 Receptor blockers[3]. The long half-life (~22 hrs) of Etoricoxib allows for once daily dosages without significant loss in efficacy[4]. Hence, there is a very low chance of overdose unless voluntarily/accidentally taken. However, there is an increased chance of cardiovascular events as it has been hypothesized that selective COX-2 inhibition will lead to decreased levels of endothelial prostacyclin (PGI2), a prostaglandin with vasodilatory and antithrombotic properties, and increased levels of thromboxane (TXA2), a platelet-derived prothrombotic vasoconstrictor. This shift in the myocardial prostaglandin milieu is thought to lead to increased coronary thrombosis and MI. Similar to Non-selective COX inhibitors in case of acute overdose, the significant load of metabolization of the drug causes acute depletion of the liver enzymes and causes jaundice due to inflammation of the liver seen in this case. Also, as seen in any poisoning, there is acute kidney injury seen consistent with the subcapsular petechial haemorrhages observed in this case[5]. Also, histopathology suggested haemorrhage, oedema, and necrosis in the kidneys, which further supports this finding. Here we can see that the heart is enlarged and the ventricles are dilated. But this dilation of the heart may be due to pre-existing heart conditions, details of which are not accurately available. We consider the finding of Grade II atheroma seen at the root of the aorta, and its contribution to causing the death of the individual cannot be neglected. Since treatment was done, and patient survived for some duration before expiring. We can say that death was due to the sequelae of toxicity due to etoricoxib overdose.

Conclusion

Despite its selective COX-2 inhibition and lower gastrointestinal risk compared to non-selective NSAIDs, there has always been a need to highlight the potential toxicity and fatal sequelae associated with Etoricoxib overdose. While accidental overdose of Etoricoxib is uncommon, its long half-life and metabolic burden on the liver can lead to severe systemic complications, including hepatic dysfunction, acute kidney injury, and cardiovascular effects. The postmortem findings, including hepatic congestion, renal haemorrhages, and ventricular dilation, suggest multi-organ involvement in the pathophysiology of toxicity. Although the presence of pre-existing cardiovascular conditions cannot be ruled out as a contributing factor in this case, the findings emphasize the need for cautious prescription practices, patient education on dosage adherence, and early recognition of overdose symptoms to prevent fatal outcomes. Grade II classification indicates minimal luminal narrowing and is unlikely to have caused significant ischemia or directly led to cardiac dysfunction. Instead, the observed biventricular dilation is more plausibly attributed to the toxic effects of Etoricoxib on myocardial tissue or fluid overload secondary to renal dysfunction. This distinction helps to avoid over-attributing the cardiac findings to pre-existing vascular pathology.

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Consent: Written informed consent for publication of clinical details, post-mortem findings, and histopathological images was obtained from the autopsy surgeon, investigating police personnel, and the relative of the deceased.

Ethical Clearance: Date:17/06/2025 Referenceno.:MC/KOL/IEC/2790/06/2025 Name: Institutional Ethics Committee, Medical College, Kolkata

Conflict of interest: The authors have no conflict of interest in this case report.

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