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CASE REPORT

Severe Acute Liver Injury Following Repeated Supratherapeutic Paracetamol Ingestion in a Child: A Case Report from a Resource-Limited Setting

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Introduction: Paracetamol is commonly used in children and is generally considered safe when given at recommended doses. However, repeated supratherapeutic ingestion over several days can lead to acute liver injury that develops gradually and presents with nonspecific symptoms, making early recognition difficult, particularly in resource-limited settings.

Case Presentation: We describe a previously healthy two-year-old boy who presented with vomiting, abdominal pain, and lethargy after a recent febrile illness. Caregiver history revealed that paracetamol syrup had been given every two hours for three consecutive days to control persistent fever. This resulted in an estimated daily dose of 103 mg/kg and a cumulative dose of 309 mg/kg. Laboratory evaluation at presentation showed severe hepatocellular injury with alanine aminotransferase 3760 U/L, aspartate aminotransferase 3240 U/L, total bilirubin 8.9 mg/dL, direct bilirubin 4.8 mg/dL, prothrombin time 20 seconds, and international normalized ratio 1.9. Serum paracetamol concentration was unavailable. Based on the clinical history and biochemical findings, intravenous N-acetylcysteine therapy was started promptly. Liver tests worsened transiently during hospitalization, improved by discharge, and normalized by six-week follow-up.

Conclusion: This case demonstrates that repeated dosing of paracetamol beyond recommended limits can result in severe liver injury in children. It highlights the importance of obtaining a careful medication history, recognizing the limitations of serum drug levels in such situations, and starting N-acetylcysteine promptly when repeated supratherapeutic ingestion is suspected. It also reinforces the need to improve caregiver awareness of safe dosing practices.

Keywords: paracetamol toxicity, repeated supratherapeutic ingestion, acute liver injury, pediatric hepatotoxicity, N-acetylcysteine, resource-limited setting

Introduction

Paracetamol, also known as acetaminophen, is one of the most frequently used antipyretic and analgesic medications in children. When administered according to recommended weight-based dosing guidelines, it is generally considered safe and is widely used in both community and hospital settings. Despite this favorable safety profile, paracetamol overdose continues to be a leading cause of drug-induced acute liver injury worldwide and contributes significantly to cases of acute liver failure.[1]

While toxicity is commonly associated with a single large ingestion, repeated supratherapeutic ingestion can also result in significant liver injury. This occurs when doses above recommended limits are given over a period of time rather than as one large dose.[2,3] In children, this often happens when caregivers administer paracetamol more frequently than

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advised in an attempt to manage persistent fever. As a result, toxicity may develop gradually and may not be recognized at an early stage.[2]

Children with ongoing fever, reduced oral intake, dehydration, or repeated dosing by caregivers may be more vulnerable to paracetamol toxicity when the medication is given more often than recommended.[2,3] In these cases, the presentation may be subtle, and early symptoms such as nausea, vomiting, abdominal pain, and lethargy are often nonspecific, making them easy to mistake for manifestations of the underlying illness rather than drug-related toxicity.[2,3]

In addition, serum paracetamol concentrations are frequently low or undetectable at presentation, which limits the usefulness of the Rumack–Matthew nomogram since it was developed for single acute ingestion.[2,4] These challenges emphasize the need to rely on clinical evaluation and biochemical evidence of liver injury when guiding management decisions.[4–6]

Paracetamol-induced hepatotoxicity results from the formation of the toxic metabolite N-acetyl-p-benzoquinone imine, which depletes hepatic glutathione stores and causes hepatocellular injury once the liver's detoxification capacity is exceeded.[2,7] This process is characterized by oxidative stress, mitochondrial dysfunction, and hepatocyte necrosis.[7,8] Although children may have some degree of metabolic protection, repeated excessive dosing can overwhelm these protective mechanisms and lead to severe acute liver injury.[2]

Recognition of repeated supratherapeutic ingestion can be especially difficult in resource-limited settings, where access to serum drug level testing is often limited. Reports of severe liver injury caused by repeated excessive dosing in children remain scarce in East Africa, and published cases from Somalia appear to be limited. We present this case to highlight the diagnostic and management challenges of repeated supratherapeutic paracetamol ingestion in a child from a resource-limited setting and to emphasize the importance of early recognition and timely treatment.

Case Presentation

A previously healthy two-year-old boy weighing 14 kg was brought to the emergency department by his parents with a one-day history of persistent vomiting, abdominal pain, and lethargy following a recent febrile illness.

According to the caregiver, paracetamol syrup (120 mg/5 mL) had been given at a dose of 5 mL every two hours for three consecutive days to control ongoing fever. This amounted to an estimated daily intake of approximately 103 mg/kg and a cumulative dose of about 309 mg/kg over 72 hours. The final dose was administered two hours before admission due to persistent fever.

On arrival, the child appeared tired but responsive and was visibly jaundiced. Vital signs showed a temperature of 37.5°C, heart rate of 118 beats per minute, respiratory rate of 26 breaths per minute, blood pressure of 92/56 mmHg, and oxygen saturation of 99% on room air. Capillary blood glucose was measured at 62 mg/dL. Abdominal examination revealed tenderness in the right upper quadrant along with mild hepatomegaly. Neurological examination was normal, with no signs of hepatic encephalopathy.

Laboratory investigations showed marked hepatocellular injury and impaired synthetic function. Alanine aminotransferase was 3760 U/L, aspartate aminotransferase was 3240 U/L, total bilirubin was 8.9 mg/dL, direct bilirubin was 4.8 mg/dL, prothrombin time was 20 seconds, and international normalized ratio was 1.9 (Table 1). White blood cell count was 14.2 $\times 10^9$/L, hemoglobin was 11.4 g/dL, platelet count was 295 $\times 10^9$/L, and C-reactive protein was 18 mg/L. Screening for viral hepatitis A, B, C, and E, dengue infection, malaria, and blood cultures yielded negative results. Serum paracetamol concentration could not be measured.

Based on the medication history and laboratory findings, paracetamol-induced severe acute liver injury was clinically suspected. The child was admitted and intravenous N-acetylcysteine therapy was started promptly using the standard 21-hour regimen. For this patient, the dosing included 2100 mg over one hour, followed by 700 mg over four hours and 1400 mg over the next sixteen hours.

During admission, the child was monitored with repeated clinical assessments and laboratory testing. Intravenous fluids containing glucose were given for supportive care because of low blood glucose on presentation.

During admission, liver tests worsened transiently, with alanine aminotransferase rising to 4850 U/L, aspartate aminotransferase to 3920 U/L, total bilirubin to 12.0 mg/dL, direct bilirubin to 6.8 mg/dL, prothrombin time to

Table 1 Laboratory Findings

Tests	Reference Range	On Admission	On Ward (Peak)	On Discharge	3-Week Follow-Up	6-Week Follow-Up
White cell count (WBC, $\times 10^9$/L)	5.0–15.0	14.2	13.5	9.8	NA	NA
Hemoglobin (g/dL)	11.0–13.0	11.4	11.0	11.8	NA	NA
Platelet ($\times 10^9$/L)	150–450	295	280	310	NA	NA
Neutrophil %	0.40–0.70	0.78	0.72	0.55	NA	NA
Lymphocyte %	0.20–0.50	0.18	0.22	0.35	NA	NA
C-reactive protein (mg/L)	<10	18	12	5	NA	NA
Aspartate transaminase (AST, U/L)	10–40	3240	3920	540	150	37
Alanine transaminase (ALT, U/L)	10–40	3760	4850	620	418	23
Alkaline phosphatase (U/L)	150–420	310	340	290	NA	NA
Total bilirubin (mg/dL)	0.2–1.2	8.9	12.0	3.1	1.8	0.9
Direct bilirubin (mg/dL)	0–0.3	4.8	6.8	1.2	0.8	0.2
Albumin (g/dL)	3.5–5.0	3.2	3.0	3.5	NA	NA
Prothrombin time (sec)	11–14	20	22	14	12	13
INR	0.8–1.2	1.9	2.1	1.2	1.3	1.1
Creatinine (mg/dL)	0.2–0.7	0.4	0.5	0.4	NA	NA
Blood urea (mg/dL)	5–18	13	15	12	NA	NA
Sodium (mmol/L)	135–145	136	138	140	NA	NA
Potassium (mmol/L)	3.5–5.0	4.2	4.0	4.1	NA	NA
Blood glucose (mg/dL)	70–110	62	85	92	NA	NA

Abbreviation: NA, not available.

22 seconds, and international normalized ratio to 2.1 (Table 1). Despite these biochemical changes, the child remained clinically stable and did not develop hepatic encephalopathy.

An abdominal ultrasound performed during hospitalization to evaluate for structural or biliary abnormalities showed normal liver morphology with no evidence of biliary obstruction or ascites (Figure 1).

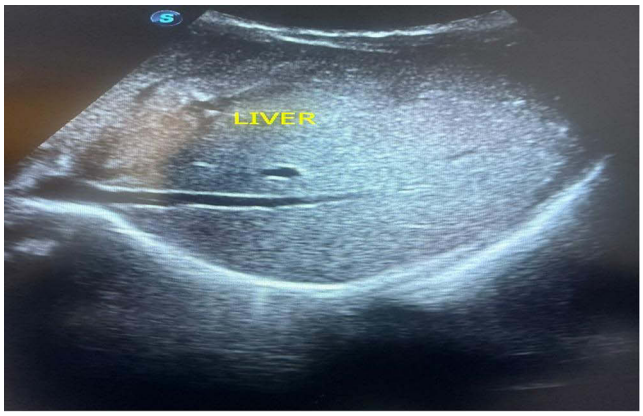

Figure 1 Abdominal ultrasound showing normal liver morphology without biliary obstruction or ascites.

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Table 2 Clinical Timeline

Time Point	Clinical Events
3 days before admission	Child developed febrile illness. Caregiver administered paracetamol syrup 120 mg/5 mL at 5 mL every 2 hours.
Day of admission	Presented with vomiting, abdominal pain, lethargy, jaundice, right upper quadrant tenderness, and mild hepatomegaly. Initial laboratory investigations showed severe acute liver injury and coagulopathy.
Early hospitalization	Intravenous N-acetylcysteine was started promptly using the standard 21-hour regimen. Intravenous glucose-containing fluids were given for supportive care.
Peak during hospitalization	Liver tests worsened transiently, with peak ALT 4850 U/L, AST 3920 U/L, total bilirubin 12.0 mg/dL, and INR 2.1. No hepatic encephalopathy developed.
During hospitalization	Abdominal ultrasound showed normal liver morphology with no biliary obstruction or ascites.
Day 6 / discharge	Clinical condition improved. Liver tests declined substantially but remained abnormal at discharge.
3-week follow-up	Continued biochemical improvement, although ALT, AST, bilirubin, and INR remained mildly abnormal.
6-week follow-up	Liver tests normalized clinically and biochemically.

With ongoing N-acetylcysteine therapy and supportive care, the child improved clinically. Liver tests improved substantially by discharge, although some values remained abnormal. At discharge on day 6, alanine aminotransferase was 620 U/L, aspartate aminotransferase was 540 U/L, total bilirubin was 3.1 mg/dL, direct bilirubin was 1.2 mg/dL, prothrombin time was 14 seconds, and international normalized ratio was 1.2. The child was discharged after six days in stable condition.

At three-week follow-up, liver enzyme levels had improved substantially but remained mildly elevated, with alanine aminotransferase 418 U/L, aspartate aminotransferase 150 U/L, total bilirubin 1.8 mg/dL, direct bilirubin 0.8 mg/dL, prothrombin time 12 seconds, and international normalized ratio 1.3. By six weeks, liver tests had normalized, with alanine aminotransferase 23 U/L, aspartate aminotransferase 37 U/L, total bilirubin 0.9 mg/dL, direct bilirubin 0.2 mg/dL, prothrombin time 13 seconds, and international normalized ratio 1.1 The sequence of key clinical events is summarized in (Table 2).

Discussion

Repeated supratherapeutic ingestion of paracetamol is an important but often overlooked cause of acute liver injury in children.[1] In contrast to toxicity following a single large ingestion, excessive dosing over several days may lead to gradual accumulation and delayed onset of symptoms.[2–4]

In this case, paracetamol was administered every two hours for three consecutive days to control persistent fever. This reflects a common situation in which dosing intervals may be shortened in response to ongoing symptoms. However, repeated shortening of the dosing interval can result in intake that exceeds recommended limits and may lead to cumulative toxicity.[2,3]

The clinical presentation in such situations is usually nonspecific. Symptoms such as vomiting, abdominal pain, lethargy, and poor oral intake may initially be attributed to the underlying illness rather than medication-related injury.[2,3] This may delay recognition and medical evaluation. In this patient, jaundice, marked transaminase elevation, hyperbilirubinemia, and coagulopathy supported the diagnosis of severe acute liver injury.

Diagnosis may be further complicated by the limited usefulness of serum paracetamol concentrations in this setting. Drug levels are often low or undetectable at presentation, which reduces the applicability of the Rumack–Matthew nomogram developed for single acute ingestion.[2,4] In this patient, the absence of measurable serum levels meant that management decisions were guided by the medication history and laboratory evidence of liver injury.

The mechanism of injury is well established. Excessive paracetamol exposure increases production of N-acetyl-p-benzoquinone imine, a reactive metabolite normally detoxified by glutathione. When glutathione is depleted, N-acetyl-p-benzoquinone imine binds to cellular proteins, promotes oxidative stress, impairs mitochondrial function, and leads to hepatocellular necrosis.[2,7,8] Although children may have some degree of metabolic protection, repeated supratherapeutic dosing can still overwhelm protective pathways and produce severe liver injury.[2]

Early initiation of N-acetylcysteine likely contributed to recovery. N-acetylcysteine helps restore glutathione stores, supports detoxification of toxic metabolites, and may reduce oxidative injury.[4–6] Current guidance supports starting treatment when repeated supratherapeutic ingestion is suspected and biochemical evidence of liver injury is present, even when serum drug levels are unavailable.[4–6]

This case also illustrates associated metabolic consequences. The child had hypoglycemia on presentation, which can occur in severe hepatic injury because of impaired gluconeogenesis and reduced hepatic reserve. No hepatic encephalopathy developed in this case, but encephalopathy remains an important marker of severe toxicity and acute liver failure in advanced presentations.[1,2]

The novelty of this report lies in the description of severe acute liver injury following repeated supratherapeutic paracetamol ingestion in a child from Somalia, where published reports of such cases appear limited. It also highlights the diagnostic and therapeutic realities of a resource-limited setting, where serum drug level testing may be unavailable and clinical judgment becomes central to management.

This report has limitations. Serum paracetamol concentration was not available, and detailed caregiver knowledge regarding dosing was not systematically assessed. As a single case report, the findings are not generalizable. However, the clear temporal relationship between repeated excessive paracetamol administration, characteristic biochemical abnormalities, exclusion of major alternative infectious causes, and response to treatment strongly supports the diagnosis.

Overall, this case emphasizes the importance of recognizing repeated supratherapeutic ingestion as a potential cause of acute liver injury in children and highlights the need to improve caregiver awareness of appropriate dosing practices to reduce the risk of unintentional toxicity.

Conclusion

This case demonstrates that repeated supratherapeutic paracetamol use can cause severe acute liver injury in children, even when there is no history of a single large overdose. It underscores the importance of obtaining a careful medication history and maintaining clinical suspicion when children present with nonspecific symptoms after a febrile illness. Early initiation of N-acetylcysteine based on clinical assessment may lead to favorable outcomes, especially in settings where serum drug level testing is not available. Improving caregiver understanding of appropriate weight-based dosing and proper dosing intervals is essential to reduce the risk of unintentional toxicity.

AI Statement

The authors used ChatGPT (OpenAI) during manuscript preparation to assist with language editing and improve clarity and readability. The tool was used only for refining grammar and sentence structure. All clinical content, interpretation, and conclusions were developed by the authors, who take full responsibility for the accuracy of the manuscript.

Abbreviations

ALT, alanine aminotransferase, AST, aspartate aminotransferase, INR; international normalized ratio, NAC, N-acetylcysteine, RSTI, repeated supratherapeutic ingestion.

Ethics and Consent

Written informed consent for publication of this case report and accompanying images was obtained from the patient's parent. Ethical approval was not required for this case report in accordance with institutional guidelines.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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