CASE REPORT

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Procalcitonin Elevation Without Infection in a Patient with Paracetamol Overdose: A Case Report

Parasetamol Zehirlenmesi Sonrası Enfeksiyon Dışı Prokalsitonin Yükselmesi: Olgu Sunumu

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Abstract

Procalcitonin (PCT) is a biomarker frequently used in the diagnosis of bacterial infections. However, elevated PCT levels have occasionally been reported in patients with paracetamol intoxication in the absence of infection. We report a case of a 31-year-old woman who presented to the emergency department four hours after ingesting 10 g of paracetamol in a suicide attempt. On admission, her PCT level was markedly elevated at 58 ng/mL, although liver function tests and other inflammatory markers were within normal limits. No infectious focus was identified and antibiotics were not initiated. The patient received standard intravenous N-acetylcysteine therapy, after which PCT levels declined rapidly, and she was discharged with complete recovery on the fourth hospital day. This case demonstrates that marked PCT elevation may occur in paracetamol toxicity even without infection, likely reflecting sterile inflammation due to hepatocellular injury and cytokine activation. In such cases, empirical antibiotic therapy may unnecessarily expose patients with paracetamol-induced liver injury to additional hepatic stress. Therefore, clinicians should interpret elevated PCT values in the context of clinical and laboratory findings rather than assuming infection.

Keywords: Case report, hepatocellular injury, non-infectious inflammation, paracetamol intoxication, procalcitonin

Öz

Prokalsitonin (PCT), bakteriyel enfeksiyonların tanısında yaygın olarak kullanılan bir biyobelirteçtir. Ancak, enfeksiyon bulunmamasına rağmen parasetamol zehirlenmesi olgularında PCT düzeylerinde artış bildirilmiştir. Bu olguda, intihar amacıyla 10 g parasetamol alan 31 yaşında bir kadında belirgin PCT yüksekliği saptanmıştır. Başvuru anında PCT düzeyi 58 ng/mL iken karaciğer fonksiyon testleri ve diğer enflamatuvar belirteçler normaldi. Enfeksiyon odağı tespit edilmedi ve antibiyotik tedavisi uygulanmadı. Standart intravenöz N-asetilsistein tedavisinin ardından PCT düzeyleri hızla geriledi ve hasta dördüncü günde tam iyileşmeyle taburcu edildi. Bu olgu, parasetamol toksisitesinde enfeksiyon olmaksızın belirgin PCT artışının görülebileceğini ve bunun muhtemelen hepatoselüler hasara bağlı steril enflamasyonu yansıttığını göstermektedir. Bu durum, parasetamol zehirlenmesine bağlı karaciğer hasarı riski bulunan hastalarda gereksiz antibiyotik kullanımının mevcut karaciğer hasarını daha da derinleştirebileceğini düşündürmektedir. Klinik pratikte, yüksek PCT düzeylerinin enfeksiyon lehine yorumlanmadan önce hastanın klinik ve laboratuvar bulgularıyla birlikte değerlendirilmesi gerektiği vurgulanmalıdır.

Anahtar kelimeler: Enfeksiyonsuz enflamasyon, hepatoselüler hasar, olgu sunumu, parasetamol zehirlenmesi, prokalsitonin

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Introduction

Procalcitonin (PCT) is a prohormone released from extra-thyroidal tissues during systemic inflammation (1,2). In clinical practice, it is most frequently used in the diagnosis of bacterial infections and in monitoring antibiotic therapy (3,4). An elevated PCT level is generally interpreted as suggestive of infection; however, transient increases may also occur in non-infectious conditions such as severe trauma, burns, surgery, cardiogenic shock, pancreatitis, and certain drug toxicities (5,6).

Paracetamol intoxication is one of the most common causes of acute liver injury (7). Hepatocellular injury due to toxic doses of paracetamol leads to oxidative stress and cytokine release, initiating a sterile inflammatory response (8,9). This reaction may cause a marked rise in PCT levels even in the absence of infection. Therefore, clinicians should be aware that PCT can be elevated for non-infectious reasons. Isolated PCT elevation following paracetamol intoxication has been rarely reported in the literature (10,11). Here, we report a case of paracetamol poisoning with preserved liver function presenting with significant PCT elevation without any evidence of infection.

Case Report

A 31-year-old woman with no known chronic disease or regular medication use, but with a history of smoking and occasional alcohol consumption, presented to the emergency department with nausea and vomiting four hours after ingesting 20 tablets of paracetamol (Minoset®, 500 mg ×20; total dose 10 g) in a suicide attempt. On admission, she was conscious, cooperative, and oriented. Vital signs were as follows: Temperature 36.7 °C, blood pressure 118/74 mmHg, heart rate 86 bpm, and oxygen saturation 97%. Physical examination was unremarkable. Initial liver and renal function tests were within normal limits. PCT was 58 ng/mL. The patient was evaluated in the emergency department and subsequently admitted to the intensive care unit (ICU) upon recommendation of the national poison consultation center for close monitoring over 72 hours.

Intravenous N-acetylcysteine (NAC) therapy was initiated according to the standard 21-hour protocol: The first step 150 mg/kg (8.25 g) in 200 mL of 5% dextrose infused over 1 hour (in the emergency department); the second step 50 mg/kg (2.75 g) in 500 mL of 5% dextrose over 4 hours; and the third step 100 mg/kg (5.5 g) in 1000 mL of 5% dextrose over 16 hours (in the ICU). A total of 16.5 g of NAC was administered. On the second day, the PCT level decreased to 44 ng/mL. The patient remained afebrile and hemodynamically stable without leukocytosis or clinical signs of infection. On day three, PCT decreased to 17 ng/mL, and on day four to 6 ng/mL. Throughout hospitalization, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and international normalized ratio (INR) levels showed no significant elevation. Blood, urine, and sputum cultures were negative. Abdominal ultrasonography revealed a normal-sized liver with increased parenchymal echogenicity consistent with grade 1 hepatic steatosis. Sepsis, viral hepatitis, and other drug-related toxicities were excluded based on clinical and laboratory findings. Arterial blood gas analysis showed a normal pH and lactate <2 mmol/L. The paracetamol concentration measured approximately 4 hours after ingestion was 148 µg/mL, consistent with a potentially toxic exposure requiring antidotal therapy according to standard assessment tools. Laboratory trends over time are summarized in Table 1.

Table 1. Laboratory parameters during hospitalization

Parameter	Day 1	Day 2	Day 3	Day 4	Normal range
AST (U/L)	22	23	20	27	0-35
ALT (U/L)	17	18	18	28	0-45
INR	0.97	1.81	1.07	0.93	0.8-1.2
Direct bilirubin (mg/dL)	0.13	0.22	0.19	0.11	<0.3
Creatinine (mg/dL)	0.85	0.72	0.70	0.69	0.6-1.1
Urea (mg/dL)	16	11	6	12	10-50
CRP (mg/L)	<2	<2	<2	<2	<5
Leukocytes (×10^9/L)	8.98	8.46	8.19	6.47	4-10
Procalcitonin (ng/mL)	58	44	17	6	<0.5
Acetaminophen (µg/mL)	148				

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, INR: ınternational normalized ratio, CRP: C-reactive protein

The patient remained hemodynamically stable throughout her ICU stay, and no antibiotics were administered. Throughout hospitalization, the patient remained afebrile, hemodynamically stable, with no leukocytosis. Blood, urine, and sputum cultures were negative, and no clinical or laboratory findings suggested infection. She was discharged uneventfully on day four with complete clinical and laboratory recovery. At follow-up, liver function tests were within normal limits.

The participant has consented to the submission of the case report to the journal. Patients signed informed consent regarding publishing their data.

Discussion

In this case, a marked elevation in PCT was observed in the absence of any evidence of infection. Similar findings have been reported in the literature describing paracetamol intoxication with elevated PCT levels unrelated to infection (10-12). In the present case, despite normal AST, ALT, and INR values, the PCT level was significantly elevated, suggesting that the increase resulted from a sterile inflammatory response rather than hepatocellular necrosis. Oxidative stress caused by toxic paracetamol metabolites leads to the release of damage-associated molecular patterns from hepatocytes (8,9,12). These molecules activate monocytes and macrophages, promoting the production of interleukin-6 and tumor necrosis factor-alpha. These cytokines stimulate extra-thyroidal synthesis of PCT, primarily in the liver and lungs (2,5). Additionally, reactive oxygen species generated during paracetamol metabolism may amplify this inflammatory response. Paracetamol-induced oxidative stress leads to hepatocellular release of damage-associated molecular patterns, triggering sterile inflammation and cytokine-mediated extra-thyroidal PCT production despite the absence of infection (2,5,8,9,12).

The marked PCT elevation in this patient can be attributed to aseptic inflammation rather than infection. This case underscores that high PCT levels can occur independently of infection and should not be used alone to justify antibiotic therapy. The gradual decline in PCT levels during follow-up further supports a non-infectious etiology. Recognizing this phenomenon is particularly important in patients with paracetamol intoxication who are at risk of hepatic dysfunction, as unnecessary antibiotic administration may aggravate liver injury.

Conclusion

Marked PCT elevation may occur after paracetamol poisoning even in the absence of infection, likely reflecting sterile inflammation secondary to oxidative stress. Clinicians should be aware of this phenomenon and interpret PCT levels alongside clinical and laboratory findings. Recognizing non-infectious PCT elevation is particularly important in paracetamol intoxication, where unnecessary antibiotic therapy could further compromise hepatic function. Importantly, isolated PCT elevation should not, by itself, prompt initiation of antibiotic therapy in the absence of clinical or microbiological evidence of infection.

Ethics

Informed Consent: The participant has consented to the submission of the case report to the journal. Patients signed informed consent regarding publishing their data.

Footnotes

Authorship Contributions

Surgical and Medical Practices: R.O.K., S.R., M.S.S., Concept: R.O.K., İ.K., Design: R.O.K., S.R., M.S.S., Data Collection or Processing: R.O.K., İ.K., M.S.S., Analysis or Interpretation: R.O.K., S.R., M.S.S., Literature Search: R.O.K., S.R., Writing: R.O.K., S.R., M.S.S.

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