CASE REPORT

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Central Diabetes Insipidus Due to Parasetamol Induced Subacute Fulminant Liver Failure: Case Report

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ABSTRACT A 20-years-old male patient admitted to our hospital with nausea and vomitting after high dose paracetamol intake. With laboratory analysis and physical examination findings patient was diagnosed subacute fulminant liver failure. Urgent liver transplant was not considered according to King's College criteria and medical treatment was started. At follow up the patient's laboratory values and encephalopathy improved progressively but polyuria developed by day 7 of treatment (11 L/day). His serum sodium level increased to 152 mmol/L. His measured plasma osmolalite was 352 mOsmol/L, urine osmolalities were 171 mOsmol/L, the urinary specific gravity was 1003. The patient was diagnosed as CDI. After 10 mcg desmopressin therapy, urinary output fell under 3L/ day and patient discharged. As in our case, in liver failure patients CDI must be considered in cases that show polyuria.

Key Words: Polyurea; acetaminophen; vasopressins

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Central diabetes insipidus (CDI) is a clinic condition characterized by polyuria and antidiuretic hormone (ADH) deficiency. Autoimmunity is mostly responsible for the etiology but rarely travma, encephalopathy, surgery and genetic disorders can play role in the etiology.[1] Paracetamol intoxication is the most common cause of acute liver failure and the disease associated with encephalopathy.[2] We present a case of neurogenic diabetes insipidus that developed after paracetamol induced subacute fulminant liver failure.

CASE REPORT

A 20-years-old male patient admitted to emergency room with nausea and vomitting. In history 20 grams of paracetamol intake due to severe headache associated with migraine was present. In laboratory analysis international normalized ratio (INR): 2.65, alanine transferase (ALT):8400 U/L, aspartate transaminase (AST): 4900 U/L, total biluribin: 3 mg/dl, direct biluribin: 1.5 mg/dl, albumine: 3.4 g/dL, sodium: 141 mmol/L, creatinin: 0.8 mg/dl, pH: 7.4, HCO_3:24 mmol/L were detected. Serum lactate levels were normal. Autoimmune and viral screen results were negative. Physical examination re-vealed grade 2 encephalopathy. An ultrasound scan showed a normal liver. Treat-

ment began with the diagnosis of suba-cute fulminant hepatic failure. Antidotal treatment with N-acetylcysteine (NAC) was started. We administered an initial loading dose of 150 mg/kg iv over to 60 minutes; next, a 4 hour infusion at 12.5 mg/kg per hour iv (ie, total of 50 mg/kg over 4 hours) and finally, a 16 hour infusion at 6.25 mg/kg per hour IV (ie, total of 100 mg/kg over 16 hours). Urgent liver transplant was not considered according to King's College criteria. At follow up the patient's laboratory values and encephalopathy improved progressively but polyuria developed by day 7 of treatment (11 L/day). His serum sodium level was increased to 152 mmol/L. His measured plasma osmolalite was 352 mOsmol/L, urine osmolalities were 171 mOsmol/L, the urinary specific gravity was 1003. Due to acute liver failure history, dehydration test was not applied to patient. With theese findings and the resolution of polyuria in response to desmopressin therapy, the patient was diagnosed as CDI. After 10 mcg des-mopressin therapy, urinary output fell under 3L/day and patient discharged.

DISCUSSION

Acetaminophen (paracetamol) is the most widely used analgesic-antipyretic in the world. Although the drug is remarkably safe when taken at usual therapeutic doses, overdose of acetaminophen cause fatal and nonfatal hepatic necrosis.[3] Because of liver failure encephalopaty and consciousness changes can be seen. But in most cases polyuria is not a common finding. Although CDI is generally associated with surgery and encephalopathy, cases related to drug intoxication are also reported in the liteature. Especially CDI cases due to ADH cell destruction in lithium overdose are reported.[4] Similarly astrocyte swelling and ADH cell destruction due to brain oedema can be seen in fulminant liver failure. In literature only one case is present about CDI due to liver failure but there is not any case about CDI in paracetamol induced liver failure.[5] In our case our patient has taken a high dose of paracetamol. We can predict that this high dose may provoke CDI. But we don't have evidence about that this CDI manifestation is dose dependent. Another randomized controled studies must be done. As a result, in liver failure patients CDI must be thinked in cases that polyuria seen.

Conflict of Interest

Authors declared no conflict of interest or financial support.

Authorship Contributions

Study conception and design; acquisition of data and analysis-interpretation of data: *Mustafa Kaplan, Erkin Öztaş, Mahmut Yüksel, Volkan Gökbulut, Muhammet Yener Akpınar, Adem Aksoy, Orhan Coşkun, Ertuğrul Kayaçetin;* ***Drafting of manuscript:*** *Mustafa Kaplan, Erkin Öztaş, Mahmut Yüksel;* ***Critical revision:*** *Orhan Coşkun, Ertuğrul Kayaçetin.*

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