LETTER TO THE EDITOR

Acute Kidney Failure Associated with Anaphylactic Shock Caused by Diclofenac Sodium

Sir,

Intramuscular Injection (IM) of Diclofenac Sodium (DS), a salt of 2-arylacetic acid (a Nonsteroidal Anti-Inflammatory Drug; NSAID), is preferred in emergency departments to control pain. Anaphylactic shock induced by diclofenac is rare, but renal functional impairment is well-known.1 NSAIDs cause renal injury by antagonising the vasodilator effects of prostaglandins.2 Dehydration in patients with chronic disease facilitates renal injury.2,3

Increase in Blood Urea Nitrogen (BUN), serum creatinine, and potassium levels, and oliguria develops in those with NSAID-associated nephrotoxicity. Renal functions return to normal 3 - 4 days following cessation of drug exposure.4

A 57 years old male patient was referred to the ophthalmology clinic with headache. Intramuscular diclofenac sodium (75 mg/3-mL) was injected to relieve pain. He lost consciousness for 5 minutes after the injection and was brought to the Emergency Department. Physical examination revealed unconsciousness, tachypnoea, tachycardia (120 beats/minute), hypotension (60/40 mmHg), oedema of the uvula and mucosa and widespread rash all over the body. Other body systems were functioning normally. His medical history included type-2 diabetes mellitus of 5 years duration. Medications taken included metformin HCL 1,000 mg p.o. twice daily and glimepiride 2 mg p.o. once daily. There was no history of drug allergy. Laboratory findings on admission were within normal ranges (Table I). A diagnosis of anaphylactic shock was made and 0.5 mg intramuscular epinephrine was administered. Intravenous 45.5 mg pheniramine maleate was given in 100 mL saline. A total of 1,000 mL of saline was given within 20 minutes. Improvements in mental status and blood pressure to 100/60 mmHg were evident during follow-up. The mucosal oedema also improved. The patient was admitted to the internal medicine unit after consultation. Follow-up in the nephrology polyclinic was recommended and the patient was discharged.

As first-line treatment of anaphylactic shock; 0.3 - 0.5 mg epinephrine i.m., 6 - 8 L/minute oxygen inhalation, rapid application of 1 - 2 L of 0.9% (w/v) saline, and overall stabilisation are required.5

Prostaglandins play active roles in maintaining the glomerular filtration rate by causing vasodilation of the renovascular bed and reducing renovascular resistance when intravascular fluid loss develops.2 If dehydration occurs in the presence of chronic disease (heart failure, hypertension, or diabetes mellitus), inhibition by NSAIDs of the beneficial effects of prostaglandins on the kidney further facilitates development of renal injury.2,3 Renal failure has been noted after 7-day use of DS.2 However, no acute renal failure caused by anaphylactic shock triggered, in turn, by a single dose of DS, has been reported in the literature. In this case, diabetes mellitus negatively affected renal function. We believe that severe intravascular fluid loss caused by DS-triggered anaphylactic shock, and antagonisation of prostaglandin function by DS, resulted in acute renal failure.

In conclusion, emergency department physicians should be aware that anaphylactic shock can develop during intramuscular administration of DS, and the resulting hypotension must be treated. In patients with underlying chronic diseases such as diabetes mellitus, physicians should note that acute renal failure might develop because of serious dehydration caused by anaphylactic shock triggered by a single dose of DS.


table 1: laboratory findings.

<table>
<thead>
<tr>
<th></th>
<th>0 hour</th>
<th>3rd hour</th>
<th>1st day</th>
<th>2nd day</th>
<th>3rd day</th>
<th>4th day</th>
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<tr>
<td>BUN (mg/dL)</td>
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<td>30</td>
<td>40</td>
<td>42</td>
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<tr>
<td>Creatinine (mg/dL)</td>
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<td>1.89</td>
<td>1.7</td>
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<td>Glucose (mg/dL)</td>
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<td>180</td>
<td>225</td>
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<td>123</td>
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<tr>
<td>AST (UL/L)</td>
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<td>17</td>
<td>17</td>
<td>16</td>
<td>15</td>
<td>17</td>
</tr>
<tr>
<td>ALT (UL/L)</td>
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<td>19</td>
<td>22</td>
<td>27</td>
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<td>34</td>
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REFERENCES


Sahin Colak1, Leyla Kutlucan2, Mehmet Ozgur Erdogan1, Mustafa Ahmet Afacan1, Ayhan Saritas3 and Hayati Kandis1
1 Department of Emergency Medicine, Haydarpasa Numune Training and Research Hospital, Istanbul, Turkey.
2 Department of Anesthesiology and Reanimation, Ataturk Government Hospital, Duzce, Turkey.
3 Department of Emergency Medicine, Duzce University School of Medicine, Duzce, Turkey.

Correspondence: Dr. Sahin Colak, Department of Emergency Medicine, Haydarpasa Numune Training and Research Hospital, Istanbul, Turkey.
E-mail: drsahincolak@hotmail.com

Received: May 26, 2014; Accepted: August 16, 2014.