Renal profile in patients with orthopaedic trauma: A prospective study

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This prospective study was undertaken to determine the incidence of acute renal failure (ARF) and to identify factors contributing to development of ARF in orthopaedic trauma patients. A total of 55 patients who presented over a period of one year with trauma to upper and lower limbs were studied. Patients with renal injury, chest or abdominal injury, isolated fractures of the hands, feet and axial skeleton involvement were excluded. Out of these, five developed acute renal failure, three recovered and two died. The overall incidence of ARF in this study was 9.1%.

Patients with lower limb injuries are at higher risk of developing ARF. Mangled Extremity Severity Score (MESS) \( \geq 7 \), higher age, patient presenting with shock, increased myoglobin levels in urine and serum have been correlated with a greater risk of patients developing ARF and a higher mortality. This study attempts to determine the magnitude of crush injury causing renal failure and the incidence of renal failure in patients with injuries affecting the appendicular skeleton exclusively.

Key words: acute renal failure; orthopaedic trauma; MESS.

INTRODUCTION

Traumatic rhabdomyolysis may result from occupational or road traffic accidents (16). Muscles account for approximately 40% of total body mass, and fracture in a limb always leads to crushing of muscles and other soft tissues, which results in release of potentially toxic intracellular components (myoglobin, iron pigments, etc.), into the systemic circulation. The resultant myoglobinuria, intravascular volume depletion and renal hypoperfusion result in renal dysfunction. About one third patients with traumatic rhabdomyolysis develop acute renal failure (ARF) (20).

Myoglobin is readily filtered through the glomerular basement membrane and passes into tubules where water is progressively reabsorbed from tubular fluid. The uptake of water by damaged muscles leads to intravascular dehydration, renal vasoconstriction and decreased glomerular perfusion pressure. This aggravates the precipitation of myoglobin into casts (3,14).
For the diagnosis of acute renal failure secondary to rhabdomyolysis, assessment of urine and serum myoglobin is unreliable because myoglobin is metabolised in the liver quickly and in an unpredictable manner. Myoglobin may have already disappeared from the blood when the patient presents, but if it is still present, urine will show typical red-brown discoloration (port wine colour). As the patient proceeds towards renal failure, other metabolic derangements are also seen (16).

A nephrologist is not always immediately available, and it puts the responsibility on orthopaedic surgeons to manage hypovolaemia, metabolic derangements and to prevent myoglobinemia from causing acute renal failure. Massive fluid infusion and alkalinisation of the urine must be started as early as possible. The earlier the fluid replacement is established, the better the chance of avoiding renal failure. Management of acute compartment syndrome can be done by performing fasciotomy in order to achieve decompression, thereby improving both local and distal blood supply. Dead, crushed muscle bleeds abundantly, is deceptively normal in appearance, and can only be differentiated from healthy muscle by its lack of contractility on electric stimulation. The excision of the necrotic muscle is inevitably incomplete and must often be repeated.

ARF caused by a crush injury was first described by Bywaters (4) during the Battle of Britain and has been identified during most major wars and conflicts ever since. He gave the first classic detailed description of crush syndrome as a result of bombing raids of London during World War II. With this the renal complication of rhabdomyolysis became firmly entrenched in the medical literature. However, none of the studies so far have determined the magnitude of crush injury causing renal failure and incidence of renal failure exclusively in the appendicular skeleton, which the present study attempts to define.

**MATERIAL AND METHODS**

This prospective study was done from 1st Sep 2004 to 31st Aug 2005 in the Department of Orthopaedics. Patients with injury to limbs, with or without associated long bone fractures were included. Patients with renal injury, chest or abdominal injury, isolated fractures of the hands and feet and axial skeleton involvement with stab wounds were excluded. All patients admitted with crush injuries were initially managed and stabilised in the trauma center. Clinical evaluation was done to check the extent of crushing using the Mangled Extremity Severity Score (MESS).

A urine sample was sent for estimating myoglobin levels in all patients. The limb was immobilised with an appropriate splint and patients were sent for radiological examination. Serum creatinine, myoglobin, blood urea, electrolytes and complete blood counts were done at admission and repeated the next day to detect any deterioration in renal function; if renal failure was detected, further biochemical screening was performed on a daily basis. In the remaining patients, renal functions were evaluated every third day until discharge.

**RESULTS**

Out of 55 patients, five developed acute renal failure. The incidence of ARF in this study was 9.1%. The mean age of the 55 patients was 37.4 years with a range from 11 to 90 years. Majority of patients were young (34.55%), in the age group 21 to 30 years. Road traffic accidents (RTA) accounted for the highest percentage of injuries (80%); five of these patients developed ARF.

Among five patients who developed ARF, four had exclusively lower limb and one had both upper limb and lower limb injuries (table I). Ten patients presented with hypovolaemic shock, of which four developed ARF. Statistically, the relationship was significant (p < 0.01).

Patients who developed ARF had a higher MESS (mean: 8.20) compared to those who did not develop ARF (mean: -5.24). Statistically, the relationship between MESS and ARF was significant (p < 0.05) (table II).

Four patients developed ARF within the first three days of hospitalisation. One patient developed septicaemia and subsequent renal failure following an arterial repair, 17 days after injury.

**DISCUSSION**

Road traffic accidents are responsible for the highest percentage of accidental deaths; they are recognised as the major cause of trauma. In this
study, 80% patients were involved in an RTA, with 34.5% of them in the 21 to 30 years age group, followed by 9% with industrial accidents. In the present study which is comparable to others, it is well documented that younger people are more prone to accidents (7). The incidence of acute renal failure in this study was seen to rise with increasing age. Trunkey (15) and Wheeler et al (18) showed that elderly patients are at a higher risk for developing acute renal failure. Vivono et al (17) found that 31% of patients developing ARF in their study were in the older age group.

In the present study hypovolaemic shock was observed in 10 patients at presentation, among whom four developed acute renal failure. Shock was a significant contributing factor in development of ARF. Similar observations were made by Lordon et al (10) who reported that 60% of renal failure patients presented with shock. Bywaters (4) documented that the majority of patients following massive trauma had low blood pressure.

A significant rise in serum creatinine level was observed in our patients who developed ARF (mean : 5 mg/dl) (Normal : 0.7-1.5 mg/dl). A rise in serum creatinine is a very important diagnostic factor in ARF. The precursor of creatinine, creatine, is one of the main constituents of muscle. It is released in large quantities from nonviable muscle cells and hydrolysed to creatinine (16). Destruction of about 200 g muscle causes a rise in creatinine levels (12).

In this study, 60% patients who developed ARF had higher serum potassium levels (mean : 5.6 mEq/L) (Normal : 3.5-5.5 mEq/L). Muscle necrosis of about 150 g will release more than 15 mmol potassium. When the integrity of muscle cell membrane is disrupted, leakage of various cellular contents into the circulation occurs; potassium is released into circulation following muscle necrosis, resulting in hyperkalaemia (10,12). Better and Stein (3) reported hyperkalaemia in all patients who developed acute renal failure. Oda et al (11) reported that 75% of patients with ARF had high serum potassium levels.

Our patients who developed ARF had significantly increased serum myoglobin levels (mean : 1646 ng/ml) (Normal : 0-65 ng/ml), this was comparable to studies by Fienfield et al (6) and Ahsan et al (1) who reported myoglobinuria in 100% patients with ARF following trauma. Ron et al (13) reported myoglobinuria in these patients but ARF was prevented by early institution of appropriate therapy. However, Wu et al (19) reported that only 30% patients with ARF had increased urine myoglobin levels following trauma. They also suggested that patients with increased levels of urine myoglobin, especially elderly patients, have a higher risk of developing acute renal failure. Vanholder et al (16) stated that serum myoglobin was not a sensitive indicator, as myoglobin is rapidly and unpredictably eliminated by hepatic metabolism. Hence, if increased serum myoglobin levels are seen in a patient, renal function should be closely monitored to diagnose ARF early.

Lordon et al (10) showed an increased incidence of sepsis with ARF. In our study 80% of the patients with ARF had wound infection. One patient with vascular injury who did not have ARF in the early days developed infection and ARF following a fasciotomy done after a vascular repair. Our findings are comparable to those of Erek et al (5) and Kazancigolu et al (9). Lordon et al (10) reported infection in 85%, with death occurring in 72% of their patients. However, they also stated that wound infections alone did not affect survival but was a probable source for most of the fatal systemic infections. Kazancigolu et al (9) in their study came to the conclusion that infections continue to be a

<table>
<thead>
<tr>
<th>Injury to Limb(s)</th>
<th>Total</th>
<th>ARF</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only Upper Limb</td>
<td>14</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Only Lower Limb</td>
<td>37</td>
<td>4</td>
<td>10.8%</td>
</tr>
<tr>
<td>Both Upper and Lower limb</td>
<td>4</td>
<td>1</td>
<td>25%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Acute Renal Failure</th>
<th>MESS (Mean ± SD) (Normal : &lt; 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes (5)</td>
<td>8.20</td>
</tr>
<tr>
<td>No (50)</td>
<td>5.24</td>
</tr>
</tbody>
</table>

Table I. — Relationship between injury to limbs and ARF

Table II. — Relationship between MESS and ARF

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major confounding factor in crush-syndrome related deaths.

Time elapsed between the injury and initiation of treatment is vital to outcome and this has been recognised for a long time. Larrey, Napoleon’s Chief Surgeon developed the concept of the “Flying Ambulance”, to reduce the time required to provide resuscitative measures to their soldiers (2). Mortality in battle casualties with ARF was as high as 91% in World War II (10). The incidence of ARF has come down over the decades and this has been attributed to early treatment provided to patients (3).

Gunal et al (8) concluded that patients requiring dialysis had been administered I.V. fluids after a much longer period of time as compared to others not requiring dialysis. Similar observations were reported by Trunkey (15). In our study, delay in treatment did not have a significant correlation with ARF. This could be because of pre-hospital treatment administered to most patients. In the present study, 63.6% of patients received first aid measures before coming to hospital, including 43.6% patients who were administered I.V. fluids prior to coming to hospital.

In the present study, three (60%) patients with ARF underwent dialysis on the 2nd, 5th and 20th day respectively, as serum potassium and creatinine levels were high, and all of them recovered from ARF. Two patients with ARF died despite early diagnosis and prompt treatment because of their advanced age and high MESS of 10. Most patients developed ARF within two days of admission. One patient developed septicaemia and subsequent renal failure following an arterial repair. This patient developed renal failure 17 days after injury. These patients had MESS score more than seven (mean: 8.2). Table III shows the maximum rise of blood urea, serum creatinine and potassium during their course in hospital (mean serum urea 140.6 mg/dl, creatinine 5 mg/dl and potassium 5.6 mEq/L respectively).

To our knowledge this is the first study done to assess the incidence and factors responsible for acute renal failure specifically in orthopaedic trauma. In this study, the incidence was low as it was conducted on a civilian population with trauma. Associated renal injury, abdominal and chest injury, fractures of hands or feet, axial skeleton involvement, head injury, injury to chest wall stab wounds were excluded. Twenty four of our patients had received I.V. fluids and 35 had received first aid before reaching hospital. Pre-hospital treatment, early fluid replacement and continuous monitoring of renal parameters also contribute to the low incidence of ARF in this study.

Early wound and fracture management and early fluid replacement will prevent renal complications from traumatic rhabdomyolysis, hypotension and electrolyte imbalance, thus preventing ARF.

**CONCLUSION**

Acute renal failure is a much feared complication in patients sustaining orthopaedic trauma. Development of acute renal failure is multifactorial. The overall incidence of acute renal failure in our study was 9.1%. The trend observed in this study suggests that chances of acute renal failure due to orthopaedic trauma increase with age. The sooner

<table>
<thead>
<tr>
<th>Patients with ARF (Serial No.)</th>
<th>Maximum blood urea level (N: 15-45 mg/dl)</th>
<th>Maximum serum creatinine level (N: 0.7-1.5 mg/dl)</th>
<th>Maximum serum potassium level (N: 3.5-5.5 mEq/L)</th>
<th>Age (yrs)</th>
<th>MESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>247</td>
<td>5.7</td>
<td>5.8</td>
<td>62</td>
<td>9</td>
</tr>
<tr>
<td>2</td>
<td>150</td>
<td>7.9</td>
<td>7</td>
<td>28</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>174</td>
<td>5.6</td>
<td>6.6</td>
<td>45</td>
<td>5</td>
</tr>
<tr>
<td>4 (Died)</td>
<td>88</td>
<td>3</td>
<td>4.6</td>
<td>65</td>
<td>10</td>
</tr>
<tr>
<td>5 (Died)</td>
<td>44</td>
<td>2.6</td>
<td>3.9</td>
<td>60</td>
<td>10</td>
</tr>
<tr>
<td>Mean</td>
<td>140.6</td>
<td>5</td>
<td>5.6</td>
<td></td>
<td></td>
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</table>
fluid replacement is started and the patient is made haemodynamically stable, the less likely is the possibility of a patient developing acute renal failure. Based on this study, it is our opinion, that a patient with a MESS > 7 is a likely candidate to develop ARF. We also found it prudent to avoid aminoglycosides and limit use of NSAIDs in patients with severe trauma. Following injury, serum and urine myoglobin and renal profile should be monitored for all patients. Early diagnosis, prevention of development of ARF by prompt fluid resuscitation and appropriate treatment of acute renal failure is recommended.

REFERENCES


Table IV. — Blood urea level evolution during hospital stay

<table>
<thead>
<tr>
<th>Patients with ARF (Serial No.)</th>
<th>Levels started rising (time)</th>
<th>Levels started coming to normal (time)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1st day</td>
<td>1 month</td>
</tr>
<tr>
<td>2</td>
<td>2nd day</td>
<td>1 month 9 days</td>
</tr>
<tr>
<td>3</td>
<td>17th day</td>
<td>1 month 2 days</td>
</tr>
<tr>
<td>4 (Died)</td>
<td>1st day</td>
<td>–</td>
</tr>
<tr>
<td>5 (Died)</td>
<td>1st day</td>
<td>–</td>
</tr>
</tbody>
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