

Location: Emergency Room

Vitals: BP: 100/60 mm Hg; HR: 104/min; RR: 30/min; Temp: 100.4F

C.C: Generalized bodyache and weakness

HPI:

A 80 years old white male is brought to the ER by his son. His son found him lying in the woods on a hot sunny day. It seemed that the patient had gone for a stroll last evening and fell down. He was unable to get up, shouted for help but could not get any. He had been lying on the ground for the last 24 hours till his son found him. The patient complained of severe bodyache. He felt very weak and was thirsty. He denied having lost consciousness. He did not pass urine for the past 24 hours. There was no history of head injury or seizures. He has no allergies and is not taking any medications. The patient does not smoke and denies any alcohol use. Family history is non-contributory. Rest of the review of systems is unremarkable.

How would you approach this patient?

This is an 80 years old man who had a fall and had been lying on the ground for more than 24 hours on a hot sunny day with no help. He is hemodynamically stable. The generalized bodyache is a hint towards possible muscle injury and should be a guide for ordering further diagnostic tests. Remember you always need a thorough physical examination to rule out serious injuries and decide which body parts to image.

Order No. 1:

IV access, stat
Pulse oximetry, stat

Results for order No 1:

Oxygen Saturation is 95% on room air

Order examination:

Complete

Results of the exam:

General appearance: Well-built, in dirt laden clothes, appears extremely dry and weak. HEENT-normal; Neck- no JVD; Respiratory - Clear to auscultation bilaterally; Cardiovascular- Tachycardia, S1 S2 normal, no murmur, rub or gallop; Abdomen-soft, non-distended, non-tender, normal bowel sounds, no organomegaly; Extremities- no edema, clubbing or cyanosis, no calf tenderness, peripheral pulses feeble; Neurological- awake, alert, oriented, no focal neurological deficit

Order No 2:

Start IV fluids: Normal saline, bolus
Insert Foley's catheter, stat
CBC with differential, stat
BMP, stat
EKG, 12 lead, stat
Urinalysis

Results for order No 2:

The nurse reports that the patient could give her only 5 cc of dark brown urine
CBC: Hgb -13.0 g/dl, Hct - 39% WBC - 13,200/uL, Platelet - 250,000/mm³, normal differential count
BMP: BUN - 45mg%, Creatinine-2.6 mg%, Sodium -134 meq/L, Potassium - 5.5 meq/L, Chloride - 92 meq/L, and bicarbonate - 17 meq/L. Calcium- 8.0 mg%
EKG shows sinus tachycardia
Urine dipstick- positive for blood; Urine microscopic- no RBC, no WBC, reddish-gold pigmented casts

Order No 3:

CPK, stat
Ionized calcium, stat
Serum magnesium, stat
Serum phosphorus, stat
Serum uric acid, stat
Urine myoglobin, stat
PT/INR, stat

APTT, stat
Admit in floor
Vitals Q 2 hours
Urine output, hourly
Activity as tolerated
IV NS, continuous

Results of Order No 3:

CPK- 10,500 IU/L
10 cc urine in Urobag
Ionized calcium- 0.99 mmol/L
Serum magnesium- 1.8 meq/L
Serum phosphorus-5.5 mg/dl
Serum uric acid- 8.5 mg/dl
Urine myoglobin- positive
PT- 14.2 sec, INR- 1.40; APTT- 35 sec

Order No 4:

Inform in 4 hours

Result of Order No 4:

BP-110/80 mmHg, HR-104/min
Urine output- 75 ml/hr

Order No. 5:

Stop 0.9% Saline
Start 0.45% Saline (with mannitol and Soda bicarbonate added to it)
Monitor urine pH every 1 hour
Titrate the mannitol - bicarbonate drip for urine pH > 6.5 and Urine output of >300 mL/hr
Check CPK in 4 Hours
Check BMP in 4 Hours
Check Magnesium and phosphorus in 4 Hours

Result of Order No 5:

CPK- 9000 IU/L
BMP: BUN-38mg%, Creatinine-2.1 mg%, Sodium -138 meq/L, Potassium -5.0 meq/L, Chloride -101 meq/L, and bicarbonate - 21 meq/L. Calcium- 8.2 mg%
Serum Magnesium- 1.4 meq/L
Serum Phosphorus- 5.0 mg/dl
BP- 130/80 mm Hg; HR-96/min
Urine pH-7.2
Urine output- 1300 cc in last 4 hours

Nurse says that the patient is feeling better**Order No. 6:**

Stop mannitol-bicarbonate diuresis
Start 0.45% saline, continuous
Check BMP, every six hours
Check serum magnesium every 6 hours
Check serum phosphorus every 6 hours
Check CPK, every 12 hours

Discussion

This is a case of rhabdomyolysis. Prolonged immobilization and compression of muscles lead to ischemic muscle damage. The hot climate and dehydration contributed to the myoglobin induced acute tubular necrosis. This is in acute renal failure with anion gap metabolic acidosis and the electrolyte abnormalities seen with rhabdomyolysis.

Rhabdomyolysis is a syndrome resulting from skeletal muscle injury with release of myoglobin and creatine phosphokinase (CPK) into the plasma. The myoglobinuria, acid urine pH and renal hypoperfusion resulting from hypovolemia leads to precipitation of heme proteins and acute tubular necrosis.

Etiology:

1. Traumatic causes: Crush syndrome, burns, electrocution,
2. Non-traumatic causes:

- Muscle hyperactivity- strenuous physical exercise, seizures, delirium tremens
- Muscle compression- prolonged immobilization, coma
- Muscle ischemia- acute arterial occlusion
- Malignant hyperthermia, neuroleptic malignant syndrome, hypothermia
- Infections- Viral including HIV, bacterial, etc.
- Drugs - alcohol, heroin, cocaine, amphetamines, zidovudine, statins
- Metabolic disorders- hypocalcaemia, hypokalemia, hypophosphatemia, hypothyroidism, hyperthyroidism, diabetic ketoacidosis
- Metabolic myopathies- e.g. Carnitine palmitoyltransferase deficiency. These should be suspected in patients with recurrent episodes of rhabdomyolysis after exertion.
- Others- carbon monoxide, snake bite

Remember that inflammatory myopathies like polymyositis and dermatomyositis very rarely give rise to rhabdomyolysis and acute renal failure.

Diagnosis:

The most common complaint is muscular pain, which is very non-specific. Moreover, a comatose patient will not complain. Dark brown urine may be the only visible sign. Suspect rhabdomyolysis in a patient with renal failure, who has blood present on urine dipstick but no RBC on microscopic examination. This is because the myoglobin in the urine causes the urine dipstick to be falsely positive for blood. Plasma creatinine concentration rises more rapidly with rhabdomyolysis (up to 2.5 mg/dL per day) than with other causes of acute renal failure. In contrast to other forms of acute tubular necrosis, FENa is less than 1 percent.

The diagnosis of rhabdomyolysis is made by measurement of CPK. It begins to raise 2 to 12 hrs after the injury and reaches its peak value 1 to 3 days after injury. The peak may range from several hundred IU/L to over 200,000 IU/L in a full blown crush syndrome. Therefore, CPK should be measured daily for at least 3 days to follow extent of muscle damage. If the serum CPK remains elevated despite treatment, ongoing muscle injury, necrosis and/or compartment syndrome should be sought.

Myoglobin is also released from the injured muscle. It increases before CPK and decreases more rapidly owing to its clearance by kidneys and metabolism to bilirubin. Therefore, remember that a normal serum myoglobin and absence of myoglobinuria does not exclude the diagnosis of rhabdomyolysis.

Various electrolyte abnormalities result from rhabdomyolysis. These can be better understood by grouping them into two categories

1. Influx from Extracellular compartment into muscle cells- water, sodium, chloride (hypovolemic shock), calcium(hypocalcemia)
2. Efflux from injured muscle cells- potassium(hyperkalemia), purines (hyperuricemia), phosphate (hyperphosphatemia), lactic acid (metabolic acidosis), myoglobin(myoglobinuria, nephrotoxicity), thromboplastin (DIC), creatine kinase, creatinine (increased serum creatinine-to-urea ratio)

Management:

1. Fluid replacement is the mainstay of therapy. Use normal saline and initiate at 1.5 L/hr. The aim is to wash off the myoglobin from the renal tubules, establish a good urine output and prevent or limit acute tubular necrosis. While on one hand many electrolyte abnormalities can precipitate rhabdomyolysis, the syndrome itself can lead to various metabolic derangements. Hence one needs to monitor the BMP and electrolytes very closely for the initial 2 days.
2. Forced alkaline diuresis using mannitol and bicarbonate is recommended by some. Alkalinization of urine prevents precipitation of myoglobin in the tubules. However, this should be used once the BP is stable and a urine output is established using isotonic saline.

One has to be careful during such large volume fluid replacement as there is always a risk of fluid overload.

Final Diagnosis:

Rhabdomyolysis due to prolonged immobilization

This copy of the material is licensed to HAFIZ MUHAMMAD ALI RAZA.
Copyright © 2014. All Rights Reserved www.usmleworld.com