The term rhabdomyolysis refers to disintegration of striated muscle, which results in the release of the key compounds released is myoglobin, an 18,800-Dalton oxygen carrier. Normally, myoglobin is loosely bound to plasma globulins and only small amounts reach the urine. When massive amounts of myoglobin are released, the binding capacity of the plasma protein is exceeded. Myoglobin is then filtered by the glomeruli and reaches the tubules, where it may cause obstruction and renal dysfunction. If the energy-dependent transcellular pump systems fail in the traumatized tissue, the muscle cells swell. Prolonged pressure may provoke irreversible paralytic damage to the peripheral nerves.

- Release of constituents of necrotic muscle results in altered plasma concentrations of several anorganic and organic compounds, which are responsible for toxic and sometimes life threatening complications. The accumulation of these compounds is aggravated by the simultaneous development of renal failure.

- Necrosis of the muscles, together with inflammation, results in the accumulation of substantial amounts of fluid in the affected limbs (up to 10 L per limb). Unless large amounts of volume are administered, shock, hypokalemia, and deterioration of renal function will supervene.

- Release of organic acids from dying muscle cells provokes high anion gap acidosis. In particular, hypoxic muscles release lactic acid into the circulation. The lower urinary pH and intracellular acidosis will facilitate intracellular precipitation of myoglobin and uric acid.

- During the early stages of rhabdomyolysis, calcium accumulates in the muscles. Sometimes massive calcification of necrotic muscles or even heterotopic ossification is seen. In the presence of hyperkalemia, severe hypocalcemia may lead to cardiac arrhythmia, muscular contraction, or seizures.

- During later stages of the disease, the accumulated calcium is released from the storage sites. This is often associated with hyperparathyroidism and hypervitaminosis D, and over hypercalcaemia. However, the hyperparathyroidism and hypervitaminosis D are not seen in all cases.

- In patients with massive breakdown of muscles, substantial amounts of potassium are released into the blood. Elimination via the kidneys fails if patients have ARF. Frequently, hyperkalaemia in patients with rhabdomyolysis is life-threatening, requiring immediate treatment. In nontraumatic rhabdomyolysis, hyperkalaemia is not consistently present at the time of admission.

- Nucleosides are released from disintegrating cell nuclei into the blood and metabolized in the liver to purines such as xanthine, hypoxanthine, and uric acid, among which the latter may contribute to tubular obstruction.

- Myoglobinuria does not occur without rhabdomyolysis, but rhabdomyolysis not necessarily results in visible myoglobinuria.

- During rhabdomyolysis, extreme quantities of CKMM are released and peak concentrations of 100,000 IU/ml or more are not unusual. Because overall degradation and removal are slow, the concentration of CK remains elevated much longer and in a more consistent manner than that of myoglobin.

- Consequently, CK is more reliable than myoglobin in assessing the presence and intensity of damage to the muscles.

- Once overt renal failure has developed, the only reliable therapeutic modality is extracorporeal blood purification.

Changes in Cellular Metabolism
- Stretching or exhaustive work of muscle cells increases sarcoplasmic influx of sodium, chloride, and water, which results in cell swelling and autodestruction. Calcium entersthe cell, in exchange for intracellular sodium. If the energy-dependent transcellular pump systems fail in the traumatized tissue, the muscle cells swell. If the energy-dependent transcellular pump systems fail in the traumatized tissue, the muscle cells swell. If the energy-dependent transcellular pump systems fail in the traumatized tissue, the muscle cells swell. Prolonged pressure may provoke irreversible paralytic damage to the peripheral nerves.

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- The primary therapeutic goal is to prevent the factors that cause ARF, i.e. volume depletion, tubular obstruction, aciduria, and free radical release.

- Once overt renal failure has developed, the only reliable therapeutic modality is extracorporeal blood purification.

(i) iv fluids
- Hyponatraemia may result from sequestration of water by muscles and must be prevented by the aggressive administration of intravenous fluids. To obtain volume equilibrium, the amount of fluid required is as high as 10 L or more per day.

(ii) sodium bicarbonate
- Sodium bicarbonate helps to correct the acidosis induced by the release of protons from damaged muscles, to prevent precipitation of myoglobin in the tubules, and to reduce the risk of hyperkalaemia.

(iii) mannitol
- Mannitol may be added to the fluid regime serves several potential purposes:
  (1) mannitol increases renal blood flow and GFR;
  (2) mannitol is an osmotic agent that affects fluid from the interstitial compartment, thus countering hypercalcemia and reducing muscular swelling and nerve compression;
  (3) mannitol is an osmotic diuretic that increases urinary flow and prevents obstructive myoglobin casts; and
  (4) mannitol scavenges free radicals.

(iv) Loop diuretics (furosamide and bumetanide)
- Increase tubular flow and decrease the risk of precipitation of myoglobin, while simultaneously acidifying urine and increasing calcium losses.

(v) Adsorbents
- May be useful because it reduces the production of uric acid and also acts as a free radical scavenger

(vi) Hyperkalaemia
- An important therapeutic goal is control of hyperkalaemia.

(vii) Renal replacement therapy
- Once overt renal failure has been established, or severe hyperkalaemia and acidosis are present, the patient requires dialysis. Fluid overload is a rare indication to start dialysis, because patients tend to be dehydrated due to massive fluid accumulation in the damaged muscle. Removal of myoglobin by plasma exchange has no demonstrated benefit

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- The degree of rhabdomyolysis that can manifest ranges from a subclinical rise of creatinine kinase (CK) to a medical emergency comprising interstitial and muscle cell edema, contraction of intravascular volume, and pigment-induced acute renal failure.