COVENTRY UNIVERSITY
SCHOOL OF HEALTH AND SOCIAL SCIENCES

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Cohort: February 2008

Module code and title: 362CPD ACUTE RENAL CARE MANAGEMENT

Assignment Title: CASE STUDY – Rhabdomyolysis and acute renal failure

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Word Allowance: 2500 +/- 10%

Word Count: 2476

Date for Submission: 5th SEPTEMBER 2008

The purpose of this case study is to evaluate and reflect upon the care given to a patient with acute renal failure and to evaluate the outcomes of the care given using the Roper-Logan and Tierney model of learning which looks at the activities of daily living.

Robert (pseudonym), a 35 year-old engineer, presented at the hospital. He was pale, hypotensive (BP 70/40 mmHg), hypothermic (temperature 34°C), tachycardic (pulse 110) and complaining of a painful, swollen, right leg. Robert admitted to a 10 year history of heroin, cocaine and amphetamine use. He was unsure of the events of the last 24 hours, but remembered injecting himself intravenously with a "usual" dose of heroin and cocaine the previous evening. On awakening the next morning he had a painful, right leg and was unable to move his right foot. He also has a history of smoking and alcohol abuse.

Based on serum chemistry results and the clinical picture, a provisional diagnosis of rhabdomyolysis of the right leg and acute renal failure was

made. He was resuscitated with intravenous fluids and given Narcan, an antagonist to heroin.

Following a surgical consultation, the provisional diagnosis was confirmed and preparation was made for transfer to theatre for a multi-compartmental fasciotomy of the right leg that afternoon. After theatre, Robert was admitted to the Intensive Therapy Unit (ITU) for postoperative management and ventilation. His main problems were identified as: rhabdomyolysis secondary to drug overdose, acute renal failure, hyperkalaemia, metabolic acidosis and cardiac changes.

For the purpose of this case study I will look more closely at the management of rhabdomyolysis in acute renal failure. I will discuss the pathophysiology and look at the nursing interventions and outcomes for this patient.

Rhabdomyolysis literally means the breakdown of striated muscle fibres that result in the release of muscle cell contents into the blood (Harper, 1990). The breakdown in the muscle cell membrane is a consequence of hypoxic damage to the muscle. Lysis (destruction of cells), necrosis and death of the

muscle (Burr, 1991). In Robert's case, his rhabdomyolysis can be attributed to his cocaine and heroin use. Cocaine, a potent vasoconstrictor, causes rhabdomyolysis (McCrea et al, 1992) by limiting the blood supply to the muscle. Diamorphine (heroin) belongs to the class of opiates that target receptors in the central nervous system producing effects of analgesia, ventilatory depression, bradycardia and sedation (Taylor, et al, 1992). Muscle cell contents released into the bloodstream results in damage to major organs and systems, leading to acute renal failure, electrolyte imbalances, metabolic changes and cardiac arrhythmias. Extracellular fluids will also move into the damaged muscle cell, raising the intra-compartmental pressure of the enclosed muscle (Burr, 1991). This results in gross muscle swelling, weak pulses and diminished sensation and movement of the affected limb (Vucak, 1991). The influx of extracellular fluids also produces a 'third space' effect and as fluid is lost from the circulation, hypovolaemia and haemodynamic shock develop.

Acute renal failure occurs in rhabdomyolysis as a result of two processes: decreased circulating plasma volume potentiates renal hypoperfusion and release of myoglobin from the muscle causes obstruction of the renal tubules. This is particularly evident when aciduria is present (Burr, 1991).

Renal hypoperfusion and the nephrotoxic effect of myoglobin may lead to acute tubular necrosis, (Humes, 1993).

During the course of acute renal failure, waste products such as urea nitrogen and creatinine accumulate in the blood resulting in a uraemic syndrome (Hoffart, 1986). High concentrations of urea in the blood will cross the blood/brain barrier causing an osmotic effect that leads to abnormal amounts of water accumulating in the cells of the brain. As these cells swell, the patient is predisposed to seizures and neurological changes (MacGeorge & Bruno, 1986). Uraemia also decreases platelet aggregation which may lead to excessive bleeding (Uldall, 1988). Anaemia may be present as a result of this and the patient may be more susceptible to infections due to the uraemic effect.

Electrolyte imbalances result from the movement of solutes into and out of the damaged cell (Muther, 1992). Potassium and calcium, two electrolytes whose movement is quite dramatic, are potentially, lethally cardiotoxic, particularly in hypotensive patients. The high concentration of potassium normally contained intracellularly is released into the extracellular

environment when cell lysis occurs, resulting in significant hyperkalaemia (Weisberg, 1993).

Hyperkalaemia is defined as a serum potassium greater than 5.0 mmol/L. Cardiac changes occur above this level due to the role of potassium in determining resting membrane potential. Normal ECGs may occur despite extreme hyperkalaemia and changes in the ECG may first be seen as ventricular fibrillation or asystole (Weisberg, 1993). Serum potassium levels above 6.5 mmol/L must be treated as an emergency (Weisberg, 1993).

Hypocalcaemia, defined as a serum calcium less than 2.25 mmol/L, results from the deposition of calcium salts into injured or necrotic muscle (Farmer, 1992).

From the hypoxic effects of a drug overdose to the metabolic consequence of muscle breakdown, each aspect of rhabdomyolysis has the potential to cause cardiac problems (Hojs et al 1999).

The management of Robert was based on a combination of physical assessment, biochemical analysis and close monitoring of all body systems. In the intensive care unit, medical, nursing and staff from other disciplines

worked together to manage each of Robert's individual problems simultaneously.

The instigation of early large volume replacement in rhabdomyolysis has been shown by Better (1990) to produce a forced solute diuresis that may protect the kidneys against acute renal failure. In Robert's case, fluid replacement was delayed and consequently renal failure resulted. Vasoconstriction of the renal vessels may also have been a contributing factor due to cocaine intake (Taylor et al, 1992).

Mannitol, an osmotic diuretic, has the potential for reducing the level of toxin in the renal tubules by excretion of water and sodium (Humes, 1993). Frusemide was also used to produce a diuresis as recommended (Humes, 1993) but failed in this case. Definitive studies validating the use of mannitol or frusemide are lacking (Farmer, 1992). Renal dose dopamine was commenced for renal vasodilation (Lee & Branch, 1991). Despite this aggressive therapy, urine output failed to rise above oliguric levels.

Haemodialysis, via a subclavian vascular catheter, was commenced when serum urea peaked at 20.8 mmol/L. It was successful in reducing the serum

urea to 14.4 mmol/L and removing 2000 mL of fluid. On haemodialysis the blood flows through a dialysis machine that filters away the waste products. This artificial kidney or dialyser is of various sizes and contains thousands of hollow fibres. These fibres act like a semi-permeable membrane, that is, they allow wastes to pass through but retain important substances like proteins. The blood circulates on one side of the membrane and the dialysate (a solution of water& electrolytes) on the other side. The toxic products and excess fluids pass through the dialyser and are carried away in the dialysate and cleansed blood, flows back into the body. The process of haemodialysis takes from 2-5 hours depending on the amount of time the consultant orders and it has to be done two to three times a week. Dialysis continued after discharge from intensive care every two days until urine output resumed As the central line, urinary catheter and AV fistula were all potential ports for infection, aseptic technique was adhered to when attending to each site (Thomas 2004). The central line and AV fistula were dressed according to unit protocol. IV lines were changed second daily and new IV fluids commenced every 24 hours. As Robert's condition improved, unnecessary lines were removed.

The fasciotomy wound was dressed daily as per the surgeon's orders and remained free from infection.

In patients at high risk for rhabdomyolysis, prevention and early recognition of the abnormality are the first steps in treatment (Russell 2000). Although prompt intervention is important, both muscle and renal cells are fairly resilient (Haskins 1998). This resiliency allows time to first address any immediate airway, breathing, or circulatory needs.

The next step in treatment is to enhance clearance of toxic intracellular contents from the circulation and from the kidneys. Although investigators agree that volume expansion, alkalinisation, and mannitol infusion are each important interventions, no consensus exists on volume, amount, and timing (Curry et al, 1989). Experts agree, however, that the single most important intervention to prevent acute renal failure in rhabdomyolysis is restoring intravascular volume and inducing a solute diuresis (Visweswaran & Guntupalli 1999, Russell 2000). Expanding the intravascular volume maximizes renal excretion by flushing out tubular debris and limiting the time nephrotoxins are in contact with renal tissues (Haskins 1998). In adult patients, the goal of isotonic crystalloid volume replacement therapy is an

hourly urine output of 150 to 300 mL(Russell 2000, Abassi et al 1998, Cheney 1995). Maintaining an output this high may require intravenous infusions of fluid of 500 to 1000 mL/h,[7] and all patients should have a urinary catheter placed in order to adequately monitor fluid output (Walsworth & Kessler 2001).

Ongoing nursing care of patients with rhabdomyolysis includes sequential monitoring of urine output (ie, checking volume and ^{colour},) to guide further fluid resuscitation. In order to prevent fluid overload and the development of pulmonary edema and congestive heart failure (Visweswaran & Guntupalli 1999, patients should be monitored closely for the development of oliguric renal failure (daily urine output <400 mL) (Cheney1994). Patients with rhabdomyolysis may benefit from invasive arterial and pulmonary artery pressure monitoring to assist with assessment of volume status (Haskins 1998). Urine pH must be serially tracked to ensure that it remains high, and arterial pH is monitored on a regular basis to prevent potential overalkalinisation (pH >7.5) due to aggressive administration of sodium bicarbonate (Slater & Mullins 1998). Other interventions include limiting the use of nephrotoxic antibiotics to minimize further kidney damage (Slater & Mullins 1998).

Creatinine kinase (CK) levels should be determined every 6 to 12 hours. The level most likely will peak dramatically in the first 12 to 36 hours and then steadily decrease during the next several days (Cheney 1994, Walsworth & Kessler 2001). Importantly, eventual renal outcome is largely dependent on the speed and efficacy of treatment and not on the CK level itself (Visweswaran & Guntupalli 1999). Patients with CK values greater than 800000 U/L who receive early and aggressive treatment may experience no subsequent renal failure (Gronert 2001). Conversely, even patients with CK levels less than 10000 U/L can have permanent impairment if care is delayed or inadequate.

Serum electrolytes must be monitored closely, and life-threatening abnormalities must be addressed promptly. Potassium levels generally peak 12 to 36 hours after injury (Dayer- Berenson 1994), and elevations are treated with standard hyperkalemic therapy (Russell 2000, Visweswaran & Guntupalli 1999). Unless patients are symptomatic, administration of exogenous calcium to correct hypocalcemia is not recommended. With hydration, calcium remobilizes from the soft tissues and can cause hypercalcaemia (Vanholder et al 2000). All patients with rhabdomyolysis

require continuous electrocardiographic monitoring for signs of hyperkalemia or cardiac irritability (Russell 2000, Haskins 1998). Compartment pressures may be measured in patients at risk for rhabdomyolysis due to extremity trauma (Slater & Mullins 1998).

Unfortunately Robert had a poor social network and had few visitors while he was in intensive care. Over the years he had lost contact with his family, and the majority of his close friends were connected with his fast-paced lifestyle.

There were many challenges for Robert concerning his upcoming recovery. He faced the possibility of not regaining the use of his leg and consequently it was uncertain if he would be able to return to his profession as an Engineer. This placed his financial stability and lifestyle at risk. Physiotherapy was commenced in ITU by way of passive range of motion exercises and a leg splint to prevent foot-drop. On the ward he was encouraged to keep his leg elevated and rested as requested by the surgeon to promote healing and later skin grafting. He was uncompliant with these orders and insisted on mobilising to go outside for cigarettes. The social workers attempted to assist with his financial and social problems. As he

was still receiving sick pay from his employer he did not see his financial situation as an immediate threat. Robert was not interested in discussing his social situation or the possibility of work retraining.

While Robert was unable to care for himself his needs were attended by the nursing staff. Initially while he was intubated and sedated, mouth and eye care were performed every two hours. Pressure area care was attended second hourly and he was nursed side-to-side to promote lung drainage and maintain skin integrity. As Robert's condition improved and he regained his independence he was encouraged to attend to his own hygiene needs. For his own safety, bedrails were kept in place. A nurse observed Robert at all times. He was continually orientated to his environment and all procedures explained prior to commencing. Environmental stressors such as excessive noise and light were kept to a minimum. These measures assisted to avoid unnecessary anxiety and consequently Robert was quite compliant with his personal care while in ITU.

Robert was offered drug rehabilitation services by the medical and nursing staff. Despite his long history of drug use, he failed to identify that he had a problem. During his hospital admission he did not show signs of drug

withdrawal. The risk of infectious diseases such as HIV and hepatitis from Robert's IV drug use was also discussed with him. He assured staff that he adhered to 'safe' needle practices; therefore, he did not see this as a problem. He did not recognise that by continuing his present lifestyle he was placing his life at risk.

The combined medical and nursing management of Robert's problems assisted him to achieve a relatively complication free admission. His ABG's returned to normal indicating a resolution of the metabolic acidosis, blood electrolyte results were within normal range, renal function returned and no permanent cardiac problems ensued. He remained normotensive for the majority of his stay.

Considering Robert's long history of drug abuse and his refusal of drug rehabilitation, there is a risk that he will return to his previous lifestyle when he is discharged from hospital. The rehabilitation process to regain the use of his leg will be extensive and it is still uncertain if he will be able to return to his previous employment. These factors combined with his poor social support network and noncompliant nature unfortunately jeopardise his long term prognosis.

Rhabdomyolysis is a clinical syndrome in which the contents of injured muscle cells leak into the circulation. This leakage results in electrolyte abnormalities, acidosis, clotting disorders, hypovolemia, and acute renal failure. More than 100 conditions, both traumatic and non-traumatic, can lead to rhabdomyolysis. Intervention consists of early detection, treatment of the underlying cause, volume replacement, urinary alkalinization, and aggressive diuresis or hemodialysis. Patients with rhabdomyolysis often require intensive care, and nurses are instrumental in both the early detection and the ongoing management of this life-threatening syndrome.

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