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Case Report

Acute kidney injury secondary to rhabdomylosis and automated peritoneal dialysis

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We report a case of 30 year old Saudi male who was admitted with status epilepticus. He then developed acute kidney injury (AKI) secondary to rhabdomyolysis which was treated with automated peritoneal dialysis (APD) with full recovery. To our knowledge, few reports have discussed the use of peritoneal dialysis in AKI.

Keyword: Rhabdomyolysis, AKI, Status epilepticus, APD

INTRODUCTION

Peritoneal dialysis (PD) is used less and less inAKI patients, and is being replaced by continuous venovenous therapies. However, it should not be discarded as a worthless therapeutic option for AKI patients. PD offers several advantages over hemodialysis (HD), including its technical simplicity, cardiovascular tolerance, absence of an extracorporeal circuit, lack of bleeding risks, and low risk of hydroelectrolyte imbalance. PD, however, has some limitations, as it needs an intact peritonealcavity, carries risks of peritoneal infections and protein losses, and has an overall lower effectiveness when compared with HD. Because daily solute clearance is lower with PD than with daily HD, there have been concerns that PD cannot control uremia in AKI patients. Controversies exist concerning its use in patients with severe hypercatabolic states; in such cases, daily hemodialysis or continuous venovenous therapies have been preferred. A little was reported in literature on PD in AKI patients, and what

exists does not address fundamental parameters such as adequate quantification of dialysis and patient catabolism. Here we report a30- year old male who presented with severe rhabdomyolysis and AKland was treated with APD with full recovery. To our knowledge there are few reports of such association in the literature.

CASE HISTORY

A 30 year old Saudi male known to have seizure disorder for the last seven years, maintained on carbamazepine and levetiracetam (Keppra), admitted due to one week history of generalized body aches and pains that was associated with nausea and dark colored urine without change in urine volume.

His symptoms were preceded with seizures that lasted for about 15 minutes. No history of fever, vomiting, trauma, alcohol intake or substance abuse. The patient works in private hospital as a clerk. He is non smoker. He denied symptoms of hypo or hyperthyroidism as well as connective tissue disorders. There was no history of renal disease in the family.

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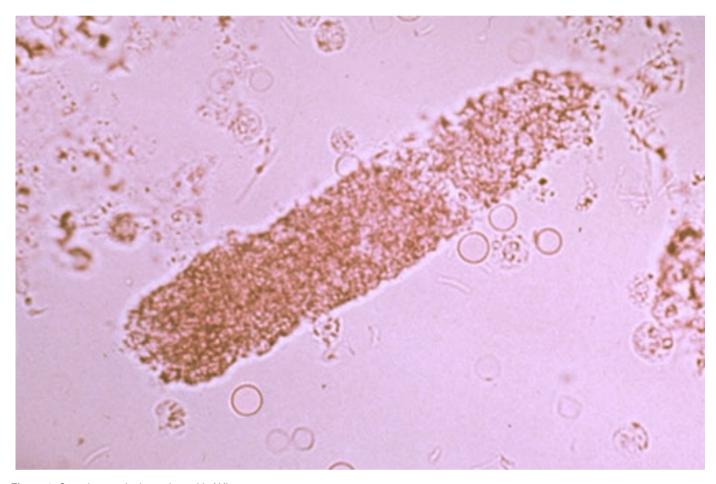


Figure 1. Granular cast in the patient with AKI

Physical Examination

The patient was Conscious, alert, oriented to time place and person not in respiratory distress.

BP: 130/72 mmHg supine and standing 125/70 mmHg, heart rate 120/minute and regular, respiratory rate 22/minute, temperature37.1C° andO2Sat was 98% on room air.

JVP was normal; trachea was central, no palpable lymph nodes and no thyroid enlargement.

Cardiovascular system: normal S1-S2 no addedsounds or murmurs.

Chest was clear for percussion and auscultation.

Abdomen: soft, lax, non-tender, nopalpable organs and no ascites.

Lower limbs: no edema, intactperipheral pulses.

Central nervous system examination was grossly intact.

Investigations

Urine analysis: large blood and urine microscopy showed multiple granular casts (Figure 1).

CBC: wbc-9.1, Hb- 13 gm/dl, platelets- 172,000/mm³,

Neutrophils-85% and lymphocytes- 11%

BUN-106 mg/dl, Creatinine- 14.8mg/dl, serum sodium-138 mEq/l, potassium- 5.8 mEq/l,CO2 - 10 mEq/l, albumin 3.7 g/dl, anion gap- 26mEq/l, LDH 471, CPK - 32,490 IU/l, myoglobin- 2867IU/l, serum calcium 8.2 mg/dl, phosphorus 10 mg/dl, uric acid 11/mg/dl .

Abdominal ultrasound: both kidneyare normal in size, shape with increased echogenicity, no stones, space occupying lesions or hydronephrosis.

PT and PTT: normal values. Serum and urine sample for toxicology were negative. ANA, C3,C4, T3,T4 and TSH were all normal.

The patient was admitted to the general ward under impression of AKI secondary to rhabdomyolysis. Peritoneal dialysis double-cuff Tenckhoff catheter was inserted in the procedure room under local anesthesia using Al-Hwiesh technique (Al-Hwiesh, et al., 2012) (Figure 2) without complication and peritoneal dialysis was started immediately with 15 liters Physioneal 1.36% over 24 hour, each fill 1.5 letter tidal70% with IV fluid o.9 normal saline at rate 120 cc per hour. Table 1 shows the patient's laboratory results over five days.



Figure 2. Double-cuff peritoneal dialysis catheter in pelvic position

Table 1. Patient's chemical profile over 5 days

	Day 1	Day2	Day3	Day4	Day5
BUN	106	92	63	51	40
Cr	14.8	13.1	11	9.6	4.5
K	5.8	5	4.5	3.8	3.2
Po4	10	8	5.8	4.9	4
Co2	10	14	19	24	26
An Gap	26	20	17	13	8
CPK	32490	9810	5920	1701	800

BUN: blood urea nitrogen, Cr: serum creatinine, K: serum potassium, PO4: serum phosphorus, CO2: serum bicarbonate, An Gap: anion gap, CPK: serum creatine phospho-kinase.

DISCUSSION

Rhabdomyolysis has been described for millennia. In the Bible a condition with characteristics similar to rhabdomyolysis is described when the Jews suffered a 'plague' during their exodus from Egypt, after abundant consumption of quail (Book of Numbers). This biblical catastrophe is assumed to have been caused by intoxication with hemlock herbs that quails consume during spring migration (Rizzi et al., 1991).

Musculoskeletal trauma, in particular crush syndrome, accounts for a large proportion of the cases of rhabdomyolysis. The first cases of crush syndrome were reported in 1908 in the German military literature (Bywaters and Beall, 1941). Crush victims who developed AKI were reported during the bombing of London during the Second World War. Pigmented casts were found in the renal tubules at autopsy. Rhabdomyolysis means destruction or disintegration of striated muscle (Farmer, 1997). This syndrome is characterized by muscle breakdown and necrosis resulting in the leakage of the intracellular muscle constituents into the circulation and extracellular fluids (Warren et al., 2002). Rhabdomyolysis ranges from an asymptomatic illness with elevation in the creatine phospho-kinase (CPK) levels, to a life threatening condition associated with extreme elevations in CPK, imbalances, together with electrolyte AKI disseminated intravascular coagulation. The cause of rhabdomyolysis is usually easily identified; however, in some instances the etiology is elusive. Muscular trauma is the most common cause of rhabdomyolysis. Less common causes include muscle enzyme deficiencies, electrolyte abnormalities, infectious causes, drugs, toxins and endocrinopathies. Rhabdomyolysis is commonly associated with myoglobinuria, and if this is sufficiently severe, it may cause AKI. Weakness, myalgias and teacolored urine are the main clinical manifestations. The most sensitive laboratory finding of muscle injury is an elevated CPK levels. In the absence of myocardial or brain infarction, CPK more than 5000 U/I indicates serious muscle injury. In our patient CPK level was 32,590u/l. Management of patients with rhabdomyolysis includes advanced life support (airway, breathing and circulation) measures, followed by attempts at preserving renal functions — the latter includes vigorous hydration. The use of alkalinizing agents and osmotic diuretics, while commonly used, remains of unproven value. In our case AKI was managed with tidal PD as described before.No IV alkalizing agents were used.Metabolic acidosis and hyperkalemia dramatically improved. The first human PD was performed by Ganter in 1923 (Teschner and Heidland, 2004) on a patient with AKIsecondary to bilateral ureteral obstruction by uterine carcinoma. Ganter used a rigid PD catheter and saline infusate. From then until the end of the 1950s, mostattempts at clinical PD were unsuccessful (Wear et al., 1938; Muehrcke, 1969; Maxwell et al., 1959). Permanent access to the peritoneal cavity was a limitingfactor for chronic dialysis use until that time. In 1968, Tenckhoff's flexible catheter (Tenckhoff and Schechter, 1968) was first used in 1975. In the 1980s, automated peritoneal dialysis (APD) by cycler was consolidated (Swartz, 1984; Diaz-Buxo, 1999) In the 1970s, PD had been widely used in AKIpatients by means of a rigid catheter in a manual and intermittent

manner (Posen and Luiscello, 1980; Steiner, 1989). In the 1980s, a flexible catheter and automated PD were introduced, and studies have reported its superiority over intermittent hemodialysis in metabolic and electrolytic control (Ash and Bever, 1995; Sipkins and Kjellstrand, 1981). In the 1990s, technical advances in hemodialysis with proportional machines, controlled ultrafiltration, and safer anticoagulation have led to reduction in PD use for AKI patients that accounted for 5 to 8% of all hospitalized patients and up to 30% of those in intensive care units (ICU) (Nash et al., 2002; Burdmann, 1997; Balbi et al., 2003; Zats, 2000). The high mortality rates (50%-80%) in critically ill patients with AKI persisted, despite advances renal replacement therapy (National Foundation, 1997; Schiff and Lang, 2002). The definition of adequate dialysis in AKI is complexand involves time of referral to dialysis, dose, and dialytic methods. There is no consensus in literature on the best method or ideal dialysis dose in AKI, although recent studies have reported that continuous methods providing the highest possible dialysis dose are beneficial in hypercatabolic patients and those with cardiovascular instability (National Kidney Foundation, 1997; Ronco et al., 2002). The nephrologist's experience with the procedure and the availability of different dialysis modalities play an important role in this regard. In a recent study involving 345 nephrology centersdistributed over five continents, Ronco et al (Ronco et al., 2002; Ronco et al., 2001) reported that continuous venovenous dialysis techniques were the major methods used in patients with AKI (45.6% of services), while peritoneal dialysis was used in 23.9% of those centers and intermittent hemodialysis in 30.5%. These two modalities presented advantages over intermittent hemodialysis, as they offer better volume control and nitrogen solute clearance. Another positive aspect is they cause lesshemodynamic instability in ICU patients (Ronco et al., 2002) However, there are no studies reporting the survival rate differences for patients submitted to these methods (Zats, 2000). Peritoneal dialysis (PD) for AKI patients still constitutes the mainstay of therapy in many of the developing countries due to its availability and ease of administration. Although its safety is being confirmed, PD is used less and less in patientswith AKI, this might be because of lack of experience, lack of facilities or both. In our PDcenter at King Fahd Hospital of the University we have developed a new technique in PD catheter insertion that made the procedure of even much simpler, safer and more efficient than before (Al-Hwiesh, et al., 2012). By using our technique we believe that PD should not be discarded as worthily therapeutic option for patient with AKI due to its technical simplicity. excellent cardiovascular tolerance, absence of an extracorporeal circuit, lack of bleeding risk and low risk of hydro-electrolyte imbalances.

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REFERENCES

- Al-Hwiesh, et al (2012). Peritoneal Dialysis International. In Press.doi: 10.3747/pdi.2012.00160
- Ash SR, Bever LS (1995). Peritoneal dialysis for acute renal failure: the safe, effective and low cust modality. Adv. Ren. Replace. Ther. 2:160–163.
- Balbi AL, Ponce D, Silva VS, Silva RPS, Torres AS, Barsante RC (2003). Insuficiência renal aguda: análise de 500 pacientesatendidosem hospital universitário. J. Bras. Nefrol. m. 25(Suppl.):50.
- Book of Numbers: The Bible.The New English Bible, 1970 Joint Comitee on the New Translation of the Bible. New York: Cambridge University Press; 11:31-35.
- Burdmann EA (1997). Epidemiologia. In Schor N, Santos O, Boim M, eds. Insuficiência Renal Aguda: Fisiopatologia, Clínica e Tratamento, 2nd ed. São Paulo, Brazil: Atheneu. 1–7.
- Bywaters E, Beall D (1941). Crush injuries with impairment of renal function. Br. Med. J. 1:427-432
- Diaz-Buxo JA (1999). Automated peritoneal dialysis: a therapy in evolution. Contrib. Nephrol. 129:1–14.
- Farmer J (1997). Rhabdomyolysis. In Critical Care.2nd edition.Edited by Civetta J, Taylor R, Kirby R. Philadephia. PA: Lippincott. pp. 1785-1791.
- Maxwell MH, Rockney RE, Kleeman CR, Twiss MR (1959). Peritoneal dialysis. JAMA. 170(8):917–924.
- Muehrcke RC (1969). Acute Renal Failure. Philadelphia: W.B. Saunders. p. 274.

- Nash K, Hafeez A, Hou S (2002). Hospital acquired renal insufficiency. Am. J. Kidney Dis. 39:930–936.
- National Kidney Foundation (1997). NFK- DOQI Clinical Practice Guideline for Hemodyalisis Adequacy. Am. J. Kidney Dis. 30(Suppl. 2):15–66.
- Posen GA, Luiscello J (1980). Continuous equilibration peritoneal dialysis in the treatment of acute renal failure. Peri. Dial. Bull. 1:6–8.
- Rizzi D, Basile C, Di Maggio A (1991). Clinical spectrum of accidental hemlock poisoning: neurologic manifestations, rhabdomyolysis and acute tubular necrosis. Nephrol. Dial. Transplant. 6:939-943.
- Ronco C, Bellomo R, Honel P (2002). Effects of different doses in continuous veno-venous haemofiltration on outcomes of acute renal failure: a prospective randomized trial. Lancet. 356:26–30.
- Ronco C, Zanella M, Brendolan A, Milan M (2001). Management of severe acute renal failure in critically ill patients: an international survey in 345 centers. Nephrol. Dial. Transplant. 16:230–237.
- Schiff HMD, Lang MS (2002). Daily hemodialysis and the outcome of acute renal failure. N. Engl. J. Med. 348:305–310
- Sipkins JH, Kjellstrand CM (1981). Severe head trauma and acute renal failure. Nephron. 28:36.
- Steiner RW (1989). Continuous equilibration peritoneal dialysis in acute renal failure. Peri. Dial. Int. 9:5–7.
- Swartz RD (1984). The Tenckhoff catheter for peritoneal dialysis. Nephron. 36(Suppl. 2): 144.
- Tenckhoff H, Schechter H (1968). A bacteriology safe peritoneal access device. Trans. Am. Soc. Artif. Intern. Organs. 14:181–187
- Teschner M, Heidland A (2004). Georg Ganter—a pioneer of peritoneal dialysis and his tragic academic demise at the hand of the Nazi regime. J. Nephrol. 17(Suppl. 3): 457–460.
- Warren J, Blumberg P, Thompson P (2002). Rhabdomyolysis: a review. Muscle Nerve. 25:332-347
- Wear JB, Sisck JR, Trinkle AJ (1938). Peritoneal lavage in the treatment of uremia. J. Urol. pp. 39–53.
- Zats R (2000). Insuficiência renal aguda. In Rocha E, Silva M, eds. Fisiopatologia Renal, 2nd ed. São Paulo, Brazil: Atheneu. pp. 261–278.