Research Paper Medical Science

Management of Rhabdomyolysis Complicating Traditional Bone Setters Treatment of Fracture

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ABSTRACT

AIM: To highlight the complications of rhabdomyolysis caused by the use of tight splint for the treatment of humeral and femoral fractures by traditional bone setter in Delta State, Nigeria.

PATIENTS AND METHODS: A retrospective study of patients treated for rhabdomyolysis complicating traditional bone setter treatment of humeral and femoral fractures with tight splint in DELSUTH from August 2012 to December2014. Inclusion criterion was those with rhabdomyolysis caused by TBS treatment of humeral and femoral fracture with tight splint. Exclusion criteria were patients with ischemic gangrene, Volkmann ischemic contracture (VIC), sickle cell and diabetic mellitus patients. Investigations done were limb x-ray, electrocardiography, chest x-ray, serum electrolyte, urea and creatinine, urinalysis with dip-stick test, creatine kinase assay and serum calcium. Treatment protocol used was fluid resuscitation with normal saline, frusemide + mannitol-alkaline diuresis.

RESULTS: A total of 6 patients were seen in the study. 4 males and 2 female with M:F ratio of 2:1. The age range of patients seen was 36-77 years (mean=54.3years). Five patients used the treatment protocol and survived while one did not use the protocol and eventually died because the diagnosis of rhabdomyolysis was not made on time.

CONCLUSION: Rhabdomyolysis is a fatal complication of reperfusion injury of muscles following release of the very tight splint used by TBS for the treatment of limb fractures. A good knowledge of the mode of presentation of the patients and necessary investigations plus the immediate commencement of treatment or amputation of the affected limb will avert the associated mortality from cardiac arrest and acute renal failure.

KEYWORDS rhabdomyolysis, myoglobinuria, renal failure, reperfusion injury, muscle injury, tourniquet

INTRODUCTION

Rhabdomyolysis occurs when muscles are compressed or crushed causing myocyte damage[1,2]. Following myocyte damage, there is release of intracellular contents into systemic circulation that are toxic to the kidney and heart[2]. Muscle injury can also occur when pressure in a prolonged ischemic muscle is suddenly released. This is called reperfusion injury[3]. It results when free oxygen radicals present in the fresh oxygenated blood are released into the ischemic muscles. Other causes rhabdomyolysis include alcohol abuse, muscular compression from coma and seizure, exertion, hyperthermia, drugs, infections and genetic disorders of muscle cell metabolism[4,5,6]. The injured muscle cells release myoglobulin, potassium, phosphate and uric acid salts. Myoglobulin and uric salts are filtered into the kidney but precipitate in the renal tubules in the presence of metabolic acidosis to cause renal failure. Hyperkalemia on the other hand is cardiotoxic. Damaged muscle cells absorb calcium and water resulting in hypocalcaemia and hypovolaemic shock as up to 10litres of water can accumulate in damaged muscles[1-3].

Historically, rhabdomyolysis was traced to Bible history when the Israelites were poisoned by ingestion of hemlock-poisoned quail birds[6]. Hemlock is now known to cause muscle damage. Seigo Minami[7], a Japanese physician 1st discovered the 'Crush syndrome' during World War 1 in 1923. He studied the pathology of 3 dead soldiers. They had renal insufficiency from renal changes due to methaemoglobin infarction. Bywaters and Beall[8] in 1941 related acute renal failure to crush injuries in victims of London bombing in Second World War. Much of the experiences in the management of rhabdomyolysis have come from treatment of victims of mass casualties of collapsed building, wreckages and disaster in the developed world[9]. Good outcome is achieved because adequate preparations for the treatment have been made ready before the patients are rescued from the wreckage. Treatment is instituted immediately to prevent the complications of reperfusion injury from setting in. These patients are managed right from the disaster site (field management) and treatment is continued in centres designated for such cases that have intensive care unit and renal dialysis (in-hospital management)[10].

In Nigeria, rhabdomyolysis has been observed in some group of patients treated by traditional bone setters (TBS) for fracture of humerus and femurs. Very tight wooden splints are applied at the middle of the arm and thigh to splint the fractures[11,12]. This causes very tight constriction in this region leading to gross swelling and ischemia of the forearm and leg distal to the splint. If the tourniquet-like splint is released early for those that present to orthopaedic surgeons, reperfusion injury may set. If released late, the limb may suffer ischemic gangrene or Volkmann ischemic contracture (VIC)[11,12].

In this study, our preliminary experience in the management of rhabdomyolysis complicating humeral and femoral fracture by TBS in Delta State University Teaching Hospital (DELSUTH), Oghara is presented.

PATIENTS AND METHOD

This is a retrospective study of patients treated for rhabdomyolysis between August 2012 and December 2014 in DELSUTH. Inclusion criterion is patient managed for humeral and femoral fractures by traditional bone setters using very tight splint on the arm or thigh respectively. Exclusion criteria are patients with ischemic gangrene and VIC from TBS treatment of humeral and femoral fractures, sickle cell and diabetic mellitus patients. Information was obtained from case notes. The biodata include sex, age, and occupation. Others are fracture suffered, duration of treatment by TBS before presentation, symptoms and signs, clinical photograph, x-ray of the limb, outcome of treatment of rhabdomyolysis and fracture.

METHOD

Patient was admitted, clinical photograph of the patient was taken. A wide bore cannula was inserted into the vein and normal saline infusion put up. Patient was catheterized. The tourniquet-like splint was removed. Blood and urine samples of the patient was taken for test before starting infusion and subsequently everyday. The tests were dip stick test (urinalysis) for myoglobulin, electrolyte, urea and creatinine, serum creatine kinase enzyme test and serum Ca^{2+} for calcium. The patient's vital signs were monitored for evidence of hypovolemic shock. ECG was done to monitor cardiac arrhythmi-

as from the effect of hyperkalemia and hypocalcemia. Chest x–ray was done for patient above 40 years to rule out cardiomegaly.

The treatment given was; intravenous normal saline of 6-12L/day. Hyperkalemia was managed using soluble insulin 8-10i.u and bolus of 50% glucose 1-2 ampoules intravenous bolus. 20% mannitol and frusemide were used to mobilize fluid out of injured muscle and to effect dieresis daily. A urine output of 200ml/hr was ensured except in the 77 year old man that was a cardiac patient, here 150ml/hr was aimed at . Chest was auscultated to rule out crepitation from fluid overload. Acidosis was treated with sodium bicarbonate at 1meq/kg intravenous injection to keep the urine pH at ≥6.5 and prevent myoglobulin and uric acid deposition in the renal tubules.

RESULTS

A total of 6 patients were seen with rhabdomyolysis complicating their fracture injury treatment by TBS. 4 males and 2 females with a M:F of 2:1. The age range was 36-77 years (mean=54.3 years). Investigation results showed hyperkalemia, hypocalcaemia and metabolic acidosis before starting treatment protocol. Five of them received the treatment protocol while one did not because the possibility of the complication was not considered. All the 5 patients that received the treatment protocol did not suffer any complication associated with failure of treating rhabdomyolysis and were later treated for their fracture injury successfully. Their investigation results became normal after the treatment. The patient that did not receive treatment protocol was successfully treated for her fracture but later developed hypovolemic shock by 5th day post-operation and acute renal failure on the 7th day post-operation. She was initially delirious and later became unconscious. Her urine was scanty and tea-colored. She had hyperkalemia, acidosis and hypocalcaemia. She died during resuscitation before renal dialysis could be done.

TABLE 1: CLINICAL INFORMATION OF PATIENTS

AGE	SEX	OCCUPATION	DURATION OF TBS RX	FRACTURE INJURY	TREATMENT PROTOCOL	OUTCOME
36YRS	M	POLICEMAN	3months	Humerus	Used	Good
52YRS	F	TRADER	10 weeks	Femur	Used	Good
55YRS	M	LECTURER	8weeks	Humerus	Used	Good
77YRS	M	RETIRED TEACHER	12weeks	Femur	Used	Good
58YRS	F	HOUSE WIFE	8weeks	Femur	Not used	Poor (Died)
48YRS	M	Trader	10weeks	Femur	Used	Good

TABLE 2: SYMPTOMS AND SIGNS SEEN IN PATIENTS WITH RHABDOMYOLYSIS TREATED BY TBS FOR FEMORAL AND HUMERAL FRACTURES

Severe pains in limb part distal to tourniquet-like splint.

Gross swelling distal to tourniquet-like splint

Differential coldness in the affected limb

Peripheral pulses not easy to palpate because of marked swelling

Passing of concentrated or tea-colored urine before and after release of splint

Immediate relieve of pain following release of splint.

Fig 1: Picture of right arm splinted for humeral fracture

DISCUSSION

When victims of disasters trapped in wreckages are rescued, they experience relieve from their agony only to die some days after as a result of reperfusion injury suffered by the muscles of the trapped limb. This condition is nick-named ''smiling death"[13]. Patients with humeral and femoral fractures treated by TBS and subsequently seen by an unsuspecting orthopaedic surgeon are exposed to similar scenario if efforts to treat rhabdomyolysis are not immediately commenced. This was the situation of the patient that did not receive the treatment protocol for rhabdomyolysis in our study. By the time it was realized, it was already too late as the various complications had set in viz a viz; hypovolemic shock, and cardiac arrest and acute renal failure. The key to stemming these complications is to have a high index of suspicion when these patients present to the surgeon. The tea- colored urine plus a positive simple dip-stick[14] test for myoglobulin in urine and elevated serum creatine kinase enzyme[17] will confirm ongoing rhabdomyolysis in the affected limb. All that is necessary to manage this type of patient must be put be in place including a renal dialysis unit and if possible an intensive care unit. This is the kind of arrangement, centres that have good outcomes in treatment of disaster victims have put in place to succeed. These centres are usually prepared and ready to care for disaster victims that are brought to them[18]. In 1988, Spitak earthquake in Armenia prompted the establishment of Renal Disaster Relief Task Force, a working group of the international society of Nephrologists (a worldwide body of kidney expert)[19]. The use of renal dialysis in the treatment of rhabdomyolysis was 1st introduced in 1950-1953 during the Korean War [20]. The lessons learnt from the mortality case in our study together with the experience gotten from the management of victims of disaster led to the good outcomes we had in our study.

Binnaz et al[21] reiterated the possibility of missing the diagnosis of rhabdomyolysis by medical personnel that are not used to seeing the presentation of rhabdomyolysis. The patient in their

case study died due to delay in diagnosis from complication of ARF just like the patient that died in our study. He pointed out that an increase in potassium level together with metabolic acidosis and a pre-operative increase in lactic acid dehydrogenase confirms diagnosis of rhabdomyolysis.

Winburn et al[22] advised that in the event of a deteriorating physiologic state of a patient with diagnosed rhabdomyolysis, amputation of the affected limb should be done to prevent further release of the toxin into the circulation to save the life of the patient. This is similar to what is done in disaster site where the trapped limb of the victim is amputated before extricating him from the site. That way the patient is completely isolated from the toxins in the injured muscle and saved from the associated complications.

The age range of the patients seen in the study belonged to the working class except the 77 year old man. Considering the poor prognosis associated with failure of instituting the treatment protocol on time, this population subset will be depleted and reduce the work force of a nation. The nation's economy will be adversely affected. For those that escape reperfusion injury, they may succumb to ischemic gangrene of the limb or VIC later.

It is interesting to note that the category of patients seen in this study included a lecturer, a retired teacher and a police-officer. This shows that even educated people still patronize TBS and become victims of their poor treatment of fractures. Thanni[23] revealed that patients patronize TBS irrespective of their educational background because of the strong belief they have in them. In addition TBS services are cheap and easy to access.

To the best of our knowledge, there is no study on the management of rhabdomyolysis complicating fracture limb treatment by TBS in our literature search for us to compare our study with. This is a preliminary study that has limitation in the number of patients managed. It is, however, strengthened by its similarity with the victims of disaster management. It is hoped that the study will draw attention of the orthopaedic community to the problem of rhabdomyolysis created by TBS in Nigeria and generate more research in it.

CONCLUSION

Rhabdomyolysis resulting from reperfusion injury is a dangerous complication sometimes associated with TBS treatment of fractures of humerus and femur. A good knowledge of its mode of presentation and treatment by the orthopaedic surgeon cannot be over emphasized to contain the mortality associated with it. Government as a matter of urgency, need to seriously take measures that will stop the menace posed by TBS activities in Nigeria. The populace needs to be enlightened about the risk associated with TBS treatment of fracture and know the right to seek treatment.

REFERENCES

1. Zager RA. Rhabdomyolysis and myohemoglobinuric acute renal failure. Kidney Int1996; 49: 314–326 | 2. Ron D, Taitelman U, Michaelson M, Bar- Joseph G, Bursztein S, Better OS. Prevention of acute renal failure in traumatic rhabdomyolysis. Arch Intern Med1984; 144: 277–280 | 3. Lameire N, Matthys E, Vanholder R et al. Causes and prognosis of acute renal failure in elderly patients. Nephrol Dial Transplant1987; 2: 316–322 | 4. Gabow PA, Kaehny WD, Kelleher SP. The spectrum of rhabdomyolysis. Medicine1982; 61: 141–15 | | 5. Poels PJE, Gabreëls FJM. Rhabdomyolysis: a review of the literature. Clin Neurol Neurosurg1993; 95: 175–192 | | 6. Rizzi D, Basile C, Di Maggio A et al. Clinical spectrum of accidental hemlock poisoning. Neurotoxic manifestations, rhabdomyolysis and acute tubular necrosis. Nephrol Dial Transplant 1991; 6: 939–943 | | 7. Brumback RA, Feeback DL, Leech RW. Rhabdomyolysis in childhood: a primer on normal muscle function and selected metabolic myopathies characterized by disordered energy production. Pediatr Clin N Am1992; 39: 821–858 | | 8. Bywaters EGL, Beall D. Crush injuries with impairment of renal function. Br Med J1941; 1: 427–432 | | 9. Odeh M. The role of reperfusion- induced injury in the pathogenesis of the crush syndrome. N Engl J Med1991; 324: 1417–1422 | | 10. Better OS, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. N Engl J Med1990; 322: 825–829 | | 11. Oginni L. The use of Traditional Fracture Splint for Bone Setting. Ni Med. Pract. 1992; Vol24 No 3: 49-51 | | | 12. Dabkana TM, Naku F, Olabode J. Volkmann Ischemic Contracture Treatment: Our Experience. 2012; 11(1):12-15. | | 13. Crush Syndrome. Wikipedia. The free encyclopedia | | 14. Sauret JM; Marinides G; Wang GK. "Rhabdomyolysis". American Family Physician 2002; 65 (5): 907–12. | | 15. Warren JD; Blumbergs PC; Thompson PD. "Rhabdomyolysis: a review". Muscle & Nerve March 2002; 25 (3): 332–47. | | 16. "Rhabdomyolysis: a review, with emphasis on the pediatric population". Pediatric Nephrology 25 (1): 7–18. | | 17. Acute Renal Failure related to Crush Syndrome: Towards an era of Seismo-Nephrology? Nephrology Dialy. Transplant 2000; 15(10):1517-21 | | 18. Rhabdomyolysis. Wikipedia, the free encyclopedia | | 19. Better OS. History of the crush syndrome. From the earthquakes of Messina, Sicily 1909 to Spitak, Armenia 1988. Am J Nephrol1997; 17: 392–3 | | 20. Sever MS; Vanholder R; Lameire M (2006). "Management of crush-related injuries after disasters". New England Journal of Medicine 2006; 354 (10): 1052–63. | 20. Schrier RW; Wang W; Poole B; Mitra A. "Acute renal failure: definitions, diagnosis, pathogenesis, and therapy". Journal of Clinical Investigation July 2004;114 (1): 5–14. | 21. Binnaz A, Vartik DI,Tumay UF,Yitmaz G. Peroperative diagnosis of Rhabdomyolysis. Marmara Medical Journal 2005;Cult 18,Sayi1,Sayfa(lar)035-038 | | 22. Winburn GB, Hawkins ML, Wood MC. Physiologic amputation prevents myoglobinuria from lower extremity myonecrosis. South Med J 1993;86: 1101-1105. | 23. Thanni LO. Factors influencing patronage of traditional bone setters. West Afr J Med. g2000; 19(3):220-4 |