



Ischemic Priapism in a Patient Secondary to Crush Syndrome: A Case Report

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Abstract

Ischemic priapism is a rare condition characterized by a rigid and painful erection with little or no cavernosal blood flow. Stasis occurs within the penile tissue secondary to venous obstruction. There are various identifiable causes of priapism, however the majority of cases are idiopathic. Herein, we present a case of a 23-year-old male with priapism due to multiple trauma and crush syndrome.

Introduction

Crush syndrome, otherwise known as traumatic rhabdomyolysis, is a systemic disorder that is caused by sudden and rapidly developing muscle damage. Rhabdomyolysis in crush syndrome occurs as a result of exposure of muscle tissue to long-term and continuous pressure, and the most important cause is earthquakes. This syndrome includes medical and surgical complications such as Acute Tubular Necrosis (ATN), electrolyte disorders (especially hyperkalemia), hypovolemic shock, heart failure, respiratory failure, infections, compartment syndrome and bleeding [1].

Priapism is defined as a long-term erection lasting more than 4 h unrelated to sexual interest or stimulation. There are various identifiable causes of priapism, however the majority of cases are idiopathic. Large population-based studies report the incidence of priapism, which was lower than 1/100,000 in the '90s, as 1.5/100,000 at the beginning of the current century [2]. Priapism has been divided into three main groups as ischemic (veno-occlusive, low flow), non-ischemic (arterial, high-flow) and intermittent (stuttering) priapism. Ischemic priapism is the most commonly observed and critical type of priapism and accounts for 95% of all cases [3].

In this report, we present a 23-year-old man with ischemic priapism due to multiple trauma and crush syndrome.

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Case Presentation

A 23-year-old earthquake survivor was referred to our hospital from south-central Turkey with bad general condition, confusion, bilateral below-knee amputee in both lower extremities, pelvic trauma, necrotic wounds above the knee, cyanotic discoloration in the scrotum and penis. In the intensive care unit, his vital signs were a temperature of 38.1°C, heart rate of 162 beats/min, blood pressure of 105/45 mmHg, respiratory rate of 18 breaths/min, and an O₂ saturation of 81%. His laboratory results were notable for a white blood cell count of 27,000 cells/ μ L, hemoglobin of 8.2 g/dL, hematocrit of 40.3%, and platelets of 45,000/mm³. His sodium was 131 mmol/L, potassium was 5.5 mmol/L, chloride was 104 mmol/L, bicarbonate was 13.5 mmol/L, blood urea nitrogen was 112 mmol/L, creatinine was 4.6 μ mol/L, glucose was 129 mmol/L, Aspartate Aminotransferase (AST) was 2999 IU/L, Alanine Aminotransferase (ALT) was 661 IU/L, amylase was 818 IU/L and Lactate Dehydrogenase (LDH) was 11417 U/L. The patient's Creatinine Kinase (CK) was 332,000 IU/L. The patient had pure Crush syndrome clinic. He was taken emergently to the operating room for fasciotomies of his lower extremity and scrotal debridement. After the procedure, the patient remained intubated in intensive care unit. He continued to receive aggressive fluid hydration at 250 mL/h to 500 mL/h to maintain a urine output of at least 200 mL/h. Priapism was observed in the patient on the 1st postoperative day (Figure 1). We entered to patient's intracavernous tissue with 4 thick intracaths, aspirated cavernosal blood and saline irrigation followed by phenylephrine injection. In the follow-up period, partial detumescence was observed in the patient. The patient's erection ceased, but necrosis began to occur in the whole penis. Total penectomy, perineostomy, bilateral orchidectomy and Fournier debridement were applied to the patient due to increased necrosis in the penis and scrotum during the follow-up of the patient (Figure 2). However, the patient unfortunately died on the 10th day of the intensive care follow-up due to multiple organ



Figure 1: Priapism was observed in the patient on the 1st postoperative day.



Figure 2: Increased necrosis in the penis and scrotum.

failure and septicemia.

Discussion

The southeastern region of Turkey suffered from earthquakes with 7.6 and 7.8 magnificence On February 6th 2023 Government data reported at least 44,324 deaths and nearly 200,000 injured survivors. Evidences of mortality reasons have not been clearly known. But we explicitly know from other earthquakes that many survivors died due to crush syndrome and associated metabolic effects [4].

Crush syndrome, one of the common complications after the earthquake, was first reported in 1909 after the Messina earthquake and World War II [5]. Crush syndrome occurs due to prolonged and continuous exposure of muscle tissue to pressure. In addition to the direct trauma of the compression, the tissue is deprived of blood flow and becomes ischemic, with both mechanisms causing lysis of muscle cells, leading to critical metabolic imbalance and organ failure [6]. Patients may experience pain, increased pressure, paresthesia, pulselessness, paresis, and pallor in the trauma area. While pain is an early sign of irreversible ischemia, pulselessness can be seen in late phase. The simplest and most effective treatment for compartment syndrome are the fasciotomy [4]. In summary, Crush syndrome is the second most common cause of death in disasters after deaths due to direct trauma. Crush syndrome is a syndrome that can be seen in major earthquakes, disasters or after heavy exercise due to muscle injury and may include complications such as acute renal failure,

electrolyte disturbances (especially hyperkalemia), hypovolemic shock, heart failure, respiratory failure, infections, compartment syndrome and bleeding.

Ischemic priapism represents compartment syndrome in the corpus cavernosa. Clinically, it is characterized by a rigid and painful erection. Stasis occurs within the penile tissue secondary to venous obstruction. A situation dominated by hypoxia is present. For this reason, necrosis begins to occur in the penile tissue in the following 4 h to 6 h [3]. Although the etiology of ischemic priapism is mostly unknown, many factors such as drug abuse, hematological abnormalities, metabolic disorders and perineal traumas are blamed [7]. Priapism of traumatic etiology has a good prognosis and is explained by the high incidence of patients aged 20 to 30 years [8]. The balance between nitric oxide/cyclic guanosine monophosphate activation, phosphodiesterase-5 mediated inactivation, and adrenergic/RhoA vasoconstriction is critical to normal erectile function. Imbalances in these pathways may cause vasodilation and vasoconstriction abnormalities of penile tissues, resulting in prolonged erection and priapism [9]. In summary, priapism can occur with or without sexual stimuli. Priapism is defined as an erection lasting more than 4 h without orgasm, non-ischemic and stuttering (recurrent or intermittent). Ischemic priapism is a clinical picture requiring urgent intervention and the failure to achieve rapid detumescence can lead to may result in irreversible corporal tissue necrosis. Necrotic damage to erectile tissues resulting in fibrosis and loss of erectile function. In this case, we see an ischemic priapism caused by trauma.

Critical metabolic changes occur in multiple trauma and crush syndrome. On the other hand, sympathetic and parasympathetic nerve pathways are also adversely affected. Secondary to these changes in metabolism, the dynamics of penile tissues are affected at different levels. In this case report, the priapism clinic and the associated devastating effects in the patient followed in the intensive care unit with crush syndrome are documented.

In this case, ischemic priapism developed in addition to the crush syndrome that developed after trauma and progressed to necrotizing fasciitis. Necrotizing fasciitis is characterized by rapidly spreading necrosis of soft tissue and fascia. It is rare but can be fatal if not managed appropriately. Despite debridement 3 times and multiple antibiotic treatment, necrotizing fasciitis progressed rapidly. The patient did not respond to treatment and had to undergo total penectomy, perineostomy and bilateral orchiectomy. In these patients, decisions should be taken very quickly and treatments should be applied before necrotizing fasciitis develops.

Conclusion

Crush syndrome may develop in patients as a result of trauma after natural disasters. At the same time, ischemic priapism may also develop in these patients. Although treatment of ischemic priapism is attempted, crush syndrome may aggravate the clinic and may progress to necrotizing fasciitis and gangrene in patients. Clinicians should always consider that priapism that may develop after trauma may progress to penile gangrene and necrotizing fasciitis and should intervene early.

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