**CASE REPORT**

# Exercise-induced rhabdomyolysis from stationary biking: a case report

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**Abstract**

There are several reports concerning exercise and rabdomyolysis. There has been no report in the English literature of exercise induced rabdomyolisis from a stationary bike.

A 63-year-old female recreational athlete presented to our hospital seeking treatment for lower back, leg pain and stiffness after exercising on a stationary bicycle one day prior. Blood work showed a raised CK of 38,120 U/L, a myoglobin of 5330 and an AST 495 U/L with normal urea and electrolytes. Urinalysis remained negative. She was admitted for oral and intravenous hydration and fluid balance monitoring

This is a very rare case of rhabdomyolysis due to exercise. This study highlights the difficulties faced by accident and emergency teams in distinguishing delayed onset muscle soreness (DOMS) from exercise-induced rhabdomyolysis, and reinforces the concept that rhabdomyolysis can occur at any level of exercise intensity. Hippokratia 2010; 14 (4): 279-280

**Key words:** rhabdomyolysis, exertional rhabdomyolysis, delayed onset muscle soreness, exercise, biking

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Reported cases of exercise induced rhabdomyolysis from stationary bicycling appear to be extremely rare. The only similar case was reported in 1984 following a supervised spinning class[1]. However, recent deaths associated with rhabdomyolysis have been described in American college wrestlers attempting dehydration weight loss by high intensity long duration exercise biking in temperature extremes while wearing rubber suits. Fortunately, the NCAA was quick to discourage this dangerous practice by banning sauna exercise and rubber suits, adding weight allowances, and enforcing more eminent pre-match weigh in times[2].

Though exercise induced rhabdomyolysis usually occurs in extreme activity and follows a benign course, potentially life-threatening consequences can occur.

## Case report

A 63-year-old female recreational athlete presented to the Homerton University Hospital A&E seeking treatment for lower back, leg pain and stiffness after exercising on a stationary bicycle one day prior. Significant history included long-standing and re-occurring lower back pain and sciatica with prior year-long disability. Left buttock pain and peri-anal paresthesia lead to her having an intial lumbo-pelvic MRI in 2003, which revealed a moderate sized disc prolapsed at the L4-5 level with possible left nerve root impingement. Due to a strong family history of hypertension and non-insulin diabetes, she had proactively sought lifestyle changes and had been referred for exercise prescription. She reported taking Cocodymol for chronic pain and Citalopram for anxiety and depression. No drug allergies were reported. On presentation, she complained of lower back pain with bilateral sciatica, but she also described an immediate onset of pain and weakness in both of her thighs immediately post exercise, which felt different than her usual baseline pain. Vital signs and urine dipstick were unremarkable. She was discharged but returned the next day due to worsening leg muscle soreness and dark urine. Urinalysis remained negative. Blood work showed a raised CK of 38,120 a myoglobin of 5330 and an AST 495 with normal urea and electrolytes. She was admitted for oral and intravenous hydration and fluid balance monitoring. Her blood tests rapidly normalized and she was discharged home two days later.

## Discussion

Though historically identified in military and paramilitary personnel with little incidence data recognized in civilian populations[3], it seems reasonable to conclude that exertional rhabdomyolysis may be common yet underreported in recreational settings. Those engaged in sport, (particularly competition athletes), may have experienced exercise induced rhabdomyolysis without seeking medical care; attributing temporary weakness and pain to overtraining.

Some studies categorize exertional rhabdomyolysis and delayed onset muscle soreness (DOMS) in continuum, with the former representing the more extreme end of the spectrum. Both may manifest with tired, sore, painful muscles, 1-5 days after unaccustomed exercise. Lab correlates may include elevated plasma CK, myoglobinemia

and changes in muscle histology and ultrastucture[4]. Physical activity can frequently produce large increases in circulating CK activity without consequence and most cases of exertional rhabdomyolysis can and probably do, resolve on their own. Senert et al demonstrated that exertional rhabdomyolysis may have a much lower incidence of renal failure than other forms, even with haematuria and high creatine kinase levels. He showed that exercise induced rhabdomyolysis typically follows a benign course as the neprhrotoxic metabolite hematin is not produced as it is in other forms of rhabdomyolysis[5].

Exertional rhabdomyolysis especially in laboratory situations has demonstrated CK levels up to 100,000 U/L without the presence of nephrotoxic factors and subsequent inconsequential resolution without treatment. However, because few data confirm this in a clinical situation in which there may be comorbidities, it remains conservative and prudent in the A&E to hydrate intravenously and monitor closely to help avoid hyperkalemia and/or acute renal failure in the face of fulminant exertional rhabdomyolysis. Some evidence suggests that lactated Ringer's solution may be more effective than normal 0.9% saline for this purpose[6].

Muscular trauma is the most common cause of rhabdomyolysis. Risk factors for exertional rhabdomyolysis include exercising in extremes of heat, humidity, under hypoxic conditions (high altitude mountaineering), or with viral or bacterial illness. Drugs (aspirin, alcohol, statins, ergogenic aids, diuretics, toxins, endocrinopathies (diabetes, hypothyroidism), and inherited conditions such as (sickle cell trait, McArdle's disease sometimes with permanent muscle weakness), some foods and even poorly planned vegeteranian diet, may also predispose[7-11].

Drug history warrants fastidious review as aging athletes are also more likely to be on predisposing medications, such as statins, diuretics, and analgesics. A positive urine dipstick occurs in only 50% of patients with rhabdomyolysis, so a test cannot exclude the diagnosis[12].

Weakness, myalgia and tea-colored urine are the main clinical symptoms. If unrecognized and not promptly treated, rhabdomyolysis can have severe consequences including, renal failure, disseminated intravascular coagulopathy and fatal cardiac arrhythmias secondary to hyperkalemia.

At the end we must have in mind according to the literature that rhabdomyolysis should not be investigated only as a clinical syndrome; cellular metabolism genetic variation and intracellular signalling are involved resulting into the destruction of the skeletal muscle. During exercise the rapid consumption of ATP levels leads to a great intracellular accumulation of Ca++ through Na-K ATPase activity. The following step in this specific molecular pathway is the activation of proteases, phospholipases A2 and nucleases which stimulates the production of oxygen-derived free radicals and the hyperoxidosis of the lipids. Consequently there is degeneration of the external membrane and therefore cytolysis[13-15].

**What this study adds**

This study documents what may be the first case of self-induced exertional rhabdomyolysis following relatively mild unsupervised stationary bicycling.

It highlights the difficulties faced by accident and emergency teams in distinguishing DOMS from exercise-induced rhabdomyolysis, and reinforces the concept that rhabdomyolysis can occur at any level of exercise intensity.

Lack of awareness of this phenomenon may lead to untimely and inappropriate management with potentially life-threatening consequences.

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