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Case Report Open Access

Multiple Organ Failure as a Result of Extensive Physical Exertion – Case Report

Zielińska-Borkowska U, Radzikowski K, Horosz B and Malec-Milewska M*

Department of Anesthesiology and Intensive Care, Medical Center for Postgraduate Education, Warsaw, Poland

***Corresponding author:** Malec-Milewska M, Ul. Czerniakowska 231, 01-416 Warszawa, Poland, Tel: 0048 22 5841220; Fax: 0048 22 5841342; E-mail: lmilewski@post.pl

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Abstract

This report presents a case of 25 years old male, who had unexpectedly developed multiple organ failure after major physical strain – running the marathon. Appropriate medical conduct (quick diagnosis and prompt therapy of rhabdomyolysis) resulted in full recovery with no neurological sequelae.

Keywords: Physical exertion; Multiple organ failure; Rhabdomyolysis

Introduction

Physical exertion is described as systemic and organ changes linked to skeletal muscle function. According to this definition the long distance running is described as dynamic, generalized (more than 30% of muscle mass is engaged) and long-lasting exertion (more than 30 min). Its intensity is described by oxygen consumption [1]. Muscular work contributes most to physical activity, as muscles form approx. 42% of the body weight. Our energy requirements are much greater during physical exercise than in rest. Oxygen consumption is also more and is directly proportional to work intensity, which human body adapts to by inreasing respiratory rate, heart rate and stroke volume (hence increased cardiac output). Greater physical exertion results in higher oxygen consumption (VO_2). Maximal oxygen consumption is termed maximal aerobic capacity and reflects the physical fitness of an individual. Regular exercise acts towards increasing maximal aerobic capacity, thus the fit person would be able to withstand significantly more demanding exercise with much smaller physiological cost. After maximal aerobic capacity is reached the anaerobic processes begin to prevail, where adenosine triphosphate (ATP) resynthesis occurs and lactate is bound to increase rapidly, resulting in metabolic acidosis that if not compensated leads to multiple organ failure (MOF)[2].

The aim of this report is to present the case of multiple organ failure in a young male, which occurred during the long-distance run. Patient has consented to his case to be described and published.

Case report

25 years old male, one of the runners in the city marathon had lost consciousness after running 9 km. At the time of Emergency Department admission he was unconscious (Glasgow Coma Score 7), pale and sweating. His temperature was 40°C. Physical examination revealed that he was tachycardiac (160/min) and hypotensive 90/50 mmHg. Due to the fact that his borderline respiratory failure presented as shortness of breath and increased respiratory rate (40/min) he was given oxygen 6 l/min via face mask, which did not prevent his oxygen saturation (O_2Sat) from further deterioration. Neurologic assessment revealed that there was no response to pain, his pupils were dilated with poor but symmetrical reaction to light and no deep tendon reflexes present. Computed tomography (CT) of the head was normal. Initial blood results were normal apart from serum creatinine (2.4 mg/dl) and glucose (214 mg/dl). Electrocardiogram (ECG) has shown a non-specific ST-T changes suggestive of ischemia. Patient was admitted to intensive care unit (ICU). No abnormalities were found in physical examination of the chest and abdomen. Repeated ECG revealed supraventricular tachycardia 150/min, with no signs suggestive of acute myocardial infarct. An episode of tonic seizures was observed with O_2Sat drop to 80%. As his respiratory function was deteriorating he was intubated and mechanically ventilated with fraction of inspired oxygen (FiO_2) 0.3, O_2Sat returned to normal at 97%. Neurological review had suggested meningococcal meningitis. Routine swabs, as well as pharyngeal swab and blood were sent for culture and sensitivity. Ceftriaxone i.v. was started and i.v. fluids increased which brought the heart rate down to 120/min. During the course of differential diagnosis a number of investigations was done: eye exam (fundus examination), toxicology tests to exclude intake of psychoactive substances, abdominal ultrasound and echocardiogram.

All of them were normal. Increase in indices of renal failure (serum creatinine 2.46–2.75mg/dl, GFR 45.3 ml), fresh and dysmorphic erythrocytes and protein (1.8 g/l) found in urinanalysis suggested rhabdomyolysis. It was later confirmed by subsequent increase in serum creatinine kinase (1378 U/l – 14460 U/l) and lactate (79 mg/dl) levels. Intravenous fluid load was increased and 20% mannitol infusion started at 10 ml/h. At the same time major clotting disturbances and significant reduction in platelets count (243–34–31–15 k/ml) were noted within first 24 hrs of admission, which suggested either sepsis of unknown origin or thrombocytopenic purpura. I.V. steroids were started. Gradual increase in troponin levels was observed (3.63-8.68-13.9 ng/ml), which was consistent with cardiac injury. Invasively monitored cardiac index (CI) was 2.8–3.1, but cardiac fraction of creatine kinase was no more than 10% of its total concentration (227 and 14460, respectively) and repetitive ECGs have shown no ST-T abnormalities. Although serum troponin changes were similar to those usually observed in myocardial infarct, the dynamics of cardiac isoform of creatine kinase levels were markedly different (Figure 1), which was valuable in excluding acute coronary ischemia. Increased liver function tests and clotting abnormalities (INR of more than 2) suggested liver failure. In a search for its etiology hepatitis B

Virus (HBV), Hepatitis C Virus (HCV), Herpes viruses, Ebstein-Barr Virus (EBV) and influenza virus infection should have been considered. Due to sustained kidney failure autoantibody screen was requested, which was negative (lupus, antinuclear, anti-mitochondrial and anti-glomerular basement membrane antibody, rheumatoid factor, Waaler-rose test). Antistreptolysin titre (ASO) was less than 200 U/ml. Apart from occasional nose bleeds and non-specific papular rash his medical history was not significant. Family history revealed no blood or muscular disorders. He was an occasional runner. After 24 hours his condition had begun to improve, his consciousness became less disturbed, breathing improved and he was extubated. Reported no pain. On the third day of ICU stay his condition was much better, gradual increase in platelets levels and positive trend in other blood tests was noted. Microbiology was negative. He was transferred to medical ward for further rehabilitation in a stable condition, fully recovered in terms of respiratory and cardiac function. He was discharged fit and well after 14 days of hospital stay.

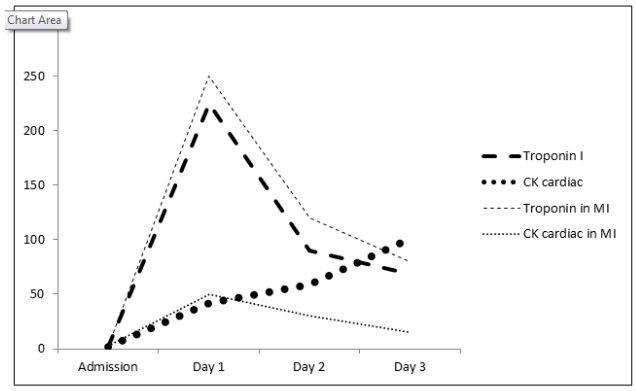

Figure 1: Troponin I and cardiac isoform of creatine kinase levels in case described (in bold) as compared to their dynamics in acute MI. Levels are given as multiples of the upper limit of normal.

Discussion

Diagnosis of multiple organ failure is established when two or more organs or systems of a human body fail to function adequately. Our patient presented with respiratory, kidney, liver and circulatory failure. It was accompanied by altered level of consciousness (7 points in GCS), which later improved (12-15 points). One of the most commonly described disturbances in the course of MOF is kidney failure, which may be caused by rhabdomyolysis. Rhabdomyolysis may be caused by mechanical, thermal and ischemic factors (large area of hypoperfusion or immobility), drugs or alcohol toxicity, food, infection, sepsis, cerebro-vascular accidents, hipo- and hyperthermia. It have been linked to some of the very common drugs (statins). May be also evoked by electrolyte disturbances and acidosis, as a complication of status epilepticus, neuroleptic malignant syndrome, malignant hyperthermia and some genetic enzyme deficiency, like muscle glycogen phosphorylase deficiency [3]. Rhabdomyolysis may also be caused by excessive physical exertion. There are reports describing rhabdomyolysis as a result of intensive physical exercise during physical education classes and fitness session in healthy, young individuals [4,5]. This condition is characterized by muscle cells lysis, content of which is released into extracellular space. Abundance of released lactate and organic acids results in metabolic acidosis, which superimposed on increased potassium levels of the same origin may facilitate cardiac arrhythmias. Myoglobin is the single most important factor contributing to renal insufficiency, as it is easily excreted into primary urine. Along with uric acid it may cause obturation of renal tubuli, which directly leads to renal failure. Heme that is released in a process of myoglobin destruction is capable of incurring direct damage to renal tubuli. Formation of free radicals is catalyzed by ferric ions released from heme in acidic conditions. Together with hypovolemia and shock the above processes lead to acute tubular necrosis and-effectively–renal failure [2].

The backbone of diagnosis is serum creatine kinase (CK) level. Five-fold increase or readings above 1000 U are consistent with diagnosis of rhabdomyolysis. Muscle damage indices are found in both physiological and abnormal conditions. The most important is CK. Its serum levels are dependent on extent and type of physical exercise. An increase in CK levels is found between 2nd and 7th day following the excessive exercise. Its specificity is very high (100%), with slightly worse sensitivity. Increase in CK is also found in the course of myocardial infarct (MI) and stroke, but its raise is always less than in cases with rhabdomyolysis present [2,6]. In MI the rate of cardiac isoform is more than 10% of the total CK. Additionally in MI troponin is increased. In case described the high levels of CK, dynamics of its change and less than 10% of its cardiac isoform allowed to exclude MI diagnosis. Test results are given in Table 1. The most sensitive marker is obviously the myoglobin. Myoglobin in urine is indicated by its dark or reddish-brown colour. In this case myoglobinuria was present at the time of ICU admission, which is easily detected when serum myoglobin level is more than 100 mg% [3]. CK measurements confirmed the diagnosis. There are multiple signs of rhabdomyolysis, from very discreet to spectacular ones. Conscious patients may report pain, muscle weakness or rigidity. Multitude of unspecific symptoms may accompany rhabdomyolysis, like increased temperature, nausea, vomiting and confusion. Blood results are the most important in establishing diagnosis, which should effect in immediate treatment. The goal of fluid therapy is to achieve hemodynamic stability and avoid acute renal failure [7]. Sodium bicarbonate given i.v. effects in increase of urine pH, which prevents myoglobin and uric acid from precipitating in renal tubuli. The backbone of the management is forced diuresis with the use of furosemide and 20% mannitol. Mannitol diminishes muscle cells oedema and is known to be a potent free radicals scavenger. Contraindications to its use are hypervolemia and hyperosmolarity. Furosemide is used at the dose of 40–200 mg/24hrs. When used in hypovolemia it may cause damage in already injured kidneys and facilitate myoglobin precipitation. When pharmacologic management fails and urine output is not satisfactory – dialysis should be considered. In nearly 40% of cases renal failure occurs, of which 40% will require renal replacement therapy (RRT). The most efficacious in this case is hemofiltration, as myoglobin is not effectively eliminated with both hemodialysis and ultrafiltration. Continuous techniques of RRT are recommended, as hemodynamic stability is not affected with their use [8,9]. An important part of management is to maintain the right concentration of uric acid. Allopurinol should be considered if readings above 20 mg/dl are noted, in order to decrease the risk of uric acid precipitation in renal tubuli. Supplementary management, like appropriate sedation (critical in mechanically ventilated patients) and pain management is equally important. Case presented underlines the complex character of MOF. There are reports describing rhabdomyolysis in healthy, young subjects after intensive exercise and football game. It is severe physical

exertion that triggers rhabdomyolysis during long-lasting physical strain, like marathon, biathlon or triathlon [10]. It is important to introduce the preventive measures, especially in patients with the history of this life-threatening condition. Appropriate volemia before and during physical exercise should help to avoid rhabdomyolysis [11,12]. Maintaining fluid homeostasis facilitates an adequate electrolyte turnover, thus preventing the occurrence of acidosis and other disturbances that may progress to multiple organ failure.

	Day 1	Day 2	Day 3	Day 4	Day 7	Day 10	Day 14	Discharge
CK (U/l)	1378	99831	177706	216425	117600	4251	691	172
CK cardiac (U/l)	13	1054	1480	2521	-	-	23	18
Troponin (ng/ml)	0.09	12.64	5.04	3.89	-	-	0.02	0.02
LDH (U/l)	346	-	370	-	-	-	-	-
ALAT (U/l)	50	186	1500	1664	3619	1312	770	426
ASPAT (U/l)	271	1094	3120	2547	1592	546	164	126
Creatinine (mg/dl)	2.41	2.51	2.54	2.27	1.18	0.91	0.89	0.89
K (mmol/l)	4.0	3.5	4.0	4.0	3.7	3.2	4.0	4.6
Ca (mg/dl)	-	-	8.24	8.2	8.75	-	-	-
Diuresis (ml/24hrs)	3400	4000	5000	5850	6200	4000	-	-
Platelets (k/ml)	256	15	51	-	-	390	-	-
Lactate (mg/dl)	79	18	15.3	-	14	-	-	-
Uric acid (mg/dl)	22	11	10	-	-	10	-	-

Table 1: Blood results. CK: Creatinine Kinase; CK cardiac: Cardiac Isoform of Creatinine Kinase; LDH: Lactate Dehydrogenase; ASPAT: Aspartate Transaminase; ALAT: Alanine Transaminase; K: Potassium; Ca: Calcium

Conclusion

In order not to increase the risk of serious, potentially life-threatening conditions caused by extensive physical exertion it is essential to adopt and prepare. Proper hydration and electrolyte supplementation is vital. Sports are good for you, but only when combined with a common sense.

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