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Address for Correspondence: Dr. Basri Amasyali,
Dumlupınar Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı,
43000, Kütahya-Türkiye
Phone: +90 274 265 20 31
Fax: +90 274 265 20 16
E-mail: dramasyali@yahoo.com



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A rare cause of circulatory shock

Cihan Altın, Arzu İzmir*, Sevdâ Osmanoğlu, Esin Gezmiş***, Afşin Sağduyu******

Departments of Cardiology, *Chest Diseases, **Internal Medicine, *Radiology and ****Psychiatry, Faculty of Medicine, Başkent University, İzmir-Turkey**

Introduction

Circulatory shock is a life-threatening clinical syndrome characterized by hypotension, tachycardia and symptoms of end-organ damage/failure. Hypovolemic, cardiogenic, distributive, obstructive and neurogenic shocks are the main types of circulatory shock. To find/diagnose the cause and to give the accurate treatment according to the underlying event are crucial in the management of circulatory shock. Herein we present an interesting shock case with unknown etiology which underlines the importance of detailed anamnesis and systemic examination (1).

Case Report

A 43-year-old male patient who works as a stockbroker, was admitted to our emergency unit with complaints of nausea, vomiting and dizziness preceding 4-6 hours. On physical examination; he was con-

scious and oriented, his skin was pale, cold and clammy. He had hypotension (70/40 mm Hg) and sinus tachycardia. Other physical and neurological examinations were normal. On his first anamnesis; there was no history of systemic disease or medication. Only he had had a viral upper respiratory infection two weeks ago. There was no suspected toxin exposure except eating cultivated mushroom 8 hours ago. Multi-systemic examination and multiple consultations were done in order to find out the predisposing factor of this circulatory shock. Which type of shock is this? What is responsible for this clinical syndrome?

His hemogram and biochemical parameters including troponine-I were unremarkable except elevated renal function tests (Creatinine: 2.11 mg/dL). Arterial blood gases revealed hypoxia and hypocapnia. Except sinus tachycardia his all electrocardiographic and echocardiographic findings were normal. Our patient did not have any infection symptoms and his all sepsis parameters were unremarkable. Therefore; hypovolemic, cardiogenic and septic shocks were ruled out. Computerize tomography (CT) was performed to rule out/find if the reason was pulmonary embolism. However pulmonary embolism was not detected and thoracoabdominal CT was also reported as normal. Mushroom intoxication was not considered because there was no elevation in patients liver function tests. Diagnosis could have been adrenal insufficiency induced by viral infection. Nevertheless patients' morning cortisol level was normal and there was no significant response on hemodynamic parameters after intravenous steroid therapy. We were not able to rule out adrenal insufficiency with these findings so that Synacthen test was planned.

Despite appropriate and sufficient hydrations (crystalloid and colloid), patient's hypotension, hypoxia and oliguria did not improve. Renal impairment progressed. Additionally the patient became complicated by pulmonary edema which might be the result of prolonged hypotension and fluid resuscitation (Fig. 1). To evaluate intravascular volume, central venous catheterization was planned. Before this invasive procedure the patient confessed that he got 20 pills of amlodine (100 mg) as a suicide attempt because he had lost a high amount of money in stock market. During all these non-invasive diagnostic tests he did not confess this suicide attempt. There was no doubt for suicide attempt because he had no depressive mood or behaviors during his hospitalization. Because we could not provide further treatments, the patient was referred to another clinic.

Discussion

Amlodipine is one of the longest half-life (30-50 hours) dihydropyridine calcium channel blocker (CCB) with a large volume of distribution (2). Toxicity may be seen in doses up to 5-10 times the therapeutic dose and occurs within 30-60 minutes following ingestion (3). In severe cases; it can result in prolonged hypotension (up to 10 days), dysrhythmias and cardiac arrest (2). Our patient developed prolonged hypotension and hypoxia without significant effect on systolic functions and cardiac pacemaker activity. He was complicated with acute renal failure and pulmonary edema. Non-cardiogenic pulmonary edema associated with CCB overdose is previously described in the literature (3-5). The pulmonary capillary transudations related to pre-capillary vasodilatation was reported as the possible mechanism of pulmonary edema (5). CCB overdose is frequently complicated by renal failure, related to the severe hypoperfusion and end-organ ischemia (2).

There is no specific efficacious antidote for CCB intoxication. Hyperinsulinemic euglycemia using dextrose and insulin infusion, cal-

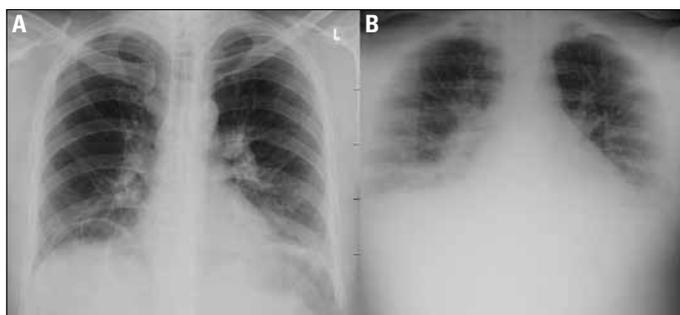


Figure 1. The patient's initial chest X-ray was normal (A). But on the chest X-ray taken next day; there was perihilar consolidations, increased width of vascular pedicle and peribronchial cuffing due to the newly occurring pulmonary edema (B).

cium gluconate and glucagon are considered as possible adjuvant therapy for CCB toxicity (3, 6, 7). According to currently available data these therapies should be considered for the refractory cases (7). Owing to high protein binding and extensive tissue distribution of CCBs hemofiltration or dialysis are not useful in overdose cases (4).

Physicians should be aware that patients may not be telling the truth every time. Especially in some psychiatric disorders such as factitious disorder (Münchausen syndrome); patient may act as if he/she has an illness by deliberately producing or exaggerating symptoms by taking drugs with overdoses (8). Factitious disorders are not rare psychiatric disorders and many clinicians may encounter them during their career. Our patient can be considered as malingering, because he had false physical symptoms intentionally produced and motivation of the behavior involved external incentives. Whereas factitious disorder has no other incentive than to be a patient and experience the sick role (8, 9).

Conclusion

This report underlines that the reliability of anamnesis should be questioned in complicated, suspicious shock cases with unknown origin. Drug overdoses and psychiatric disorders should be kept in mind in such suspicious patients.

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Address for Correspondence: Dr. Cihan Altın,
6471/5 Sokak, No:7, Yalı Mahallesi, Bostanlı, Karşıyaka, İzmir-Türkiye
Phone: +90 232 241 10 00
E-mail: drcihanaltin@hotmail.com

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Successful elimination of a Mahaim pathway using an 8 mm tip cryoablation catheter in a child

Neslihan Kıplapınar, Celal Akdeniz, Yakup Ergül, Volkan Tuzcu*
Departments of Pediatric Cardiology/Electrophysiology and *Pediatric Arrhythmia Center, Mehmet Akif Ersoy Cardiovascular Research and Training Hospital; İstanbul-Turkey

Introduction

Mahaim fibers typically demonstrate decremental conduction properties and constitute approximately 3% of preexcitation syndromes (1). Mahaim pathways are usually right-sided, however several left-sided cases have been reported (2). Conventionally Mahaim pathway ablation is performed with radiofrequency ablation (RFA). We report a patient who presented with a wide QRS tachycardia with left bundle branch (LBBB) pattern. The electrophysiology study demonstrated Mahaim tachycardia and the patient was successfully treated with cryoablation following a failed attempt with RFA.

Case Report

An 8-year-old girl was referred to our clinic with the preliminary diagnosis of ventricular tachycardia. Her palpitations started at the age of 2. Metoprolol was started 2 months prior to the presentation, however tachycardia episodes continued. A resting 12-lead electrocardiogram showed no abnormalities. A 24-hour Holter monitoring demonstrated wide QRS tachycardia with leftward superior axis deviation and LBBB morphology. During the exercise test tachycardia was induced and the test had to be terminated due to sustained wide QRS tachycardia (Fig. 1).

An electrophysiology study was carried out under general anesthesia, following the parents' consent for the procedure. Two quadripolar catheters and a decapolar catheter (St. Jude Medical Inc., St. Paul, MN) were positioned at the high right atrium, coronary sinus and right ventricle. The Ensite Velocity system (St. Jude Medical Inc., St. Paul, MN) was used for mapping and navigation of the catheters. A wide QRS tachycardia with LBBB and superior axis started spontaneously. Intravenous adenosine was administered as 200 mcg/kg, resulting in sudden termination of the tachycardia. However, following a few ventricular escape complexes, sinus rhythm returned with pre-excitation with the same QRS morphology similar to that of during the tachycardia. With programmed ventricular stimulation, earliest ventriculoatrial conduction was observed in the His region with decremental properties. When the diagnostic catheter was at the His position, there was no His signal preceding the ventricular signal during the wide QRS tachycardia. All of these electrophysiological features were strongly suggestive