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Case Report

Fatal Progress of Viral Infection during Influenza Epidemic - Acute Myocarditis with Peracute Course

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Abstract

We report here a case of acute cardiac failure due to myocarditis during culmination of epidemic influenza during March 2012 in the Czech Republic. A 17 years old, previously relatively healthy boy was rushed to the Pediatric Emergency Department, reporting 3 days fever, fatigue, headache, dry cough, with progression to syncope that morning. Because of hypotension and general appearance, the patient stayed for short-stay observation, infusion therapy and additional examination. Laboratory tests revealed elevated levels of CK-MB and troponin I; echocardiogram confirmed suspicion of acute myocarditis. After hypotension progressed, the patient was transferred to Pediatric Intense Care Unit for complete resuscitation care, where echo was repeated. The dysfunction of left cardiac ventricle was detected and extracorporeal membrane oxygenation (ECMO) was applied. Despite 11 hours of ECMO, situation led to akinesis of both ventricles with infaust prognosis. This case report demonstrates the key need for wide differential diagnosis of syncope and influenza-like symptoms.

Keywords: *Acute Myocarditis; Syncope; Influenza-Like Symptoms; ECMO*

Abbreviations

BP: Blood Pressure; HR: Heart Rate; RR: Respiratory Rate; CPR: Cardiopulmonary Resuscitation; ECMO: Extracorporeal Membrane Oxygenation; CAD: Coronary Arterial Disease; CK: Creatine Kinase; (P)ED: (Pediatric) Emergency Department; LV: Left Ventricle; CBC: Complete Blood Count; MOF: Multiorgan Failure.

Introduction

Syncope is a self-limited loss of consciousness with inability to maintain postural tone that followed by spontaneous recovery. Syncope is not very common in pediatric population, all the more so it warrant careful evaluation. We discuss the patient with acute myocarditis presented by syncope after a 3 days history of influenza-like symptoms presented on ED during culmination of influenza epidemic.

Case Report

HPI

17 years old previously relatively healthy boy presented to PED after a syncopal event that morning. He was found by parents of bathroom's floor around 5 AM. He was reported 3 days of fever (38 - 39°C), fatigue, headache and dry couch, no complaining of chest pain, shortness of breath or palpitation.

Physical examination revealed a pale and fatigue adolescent male, vital signs were as follows: afebrile, BP 87/ 64, HR 91/min, RR 18/ min, puls ox 100%, with good peripheral perfusion, eupnoic, no rashes, with no neurologic deficit.

Because of hypotension and general appearance, the patient was admitted for short-stay observation, infusion therapy and additional evaluation.

History: Past medical, family, social

On his past medical history, he was hospitalized for adenectomy at 4 years.

He was evaluated on immunology for higher sickness rate during infancy (with good effect of immunomodulating therapy, with improvement during childhood) and for fatigue syndrome at the age of 13 (susp. after EBV infection), no immunodeficiency was detected. He was evaluated for varicocele and for chronic venous insufficiency, the surgery was planned. Mild hypercholesterolemia. Otherwise healthy boy. No chronic medication.

On his family history, his father has goiter, hypercholesterolemia, his mother is healthy, his younger brother has higher sickness rate, the older one has AV block, grandparents have DM and CAD.

Social history: Student, football player.

Hospital course

During short-stay observation on PED laboratory tests were obtained, there was no elevation of inflammatory markers, normal blood count, but slightly elevated levels of CK-MB (3,45 μkat/l) and troponin I (0,628 ng/l) was revealed.

Evaluation by cardiologist showed decreased contractility and edema of left ventricle, with small amount of pericardial effusion on ECHO, on EKG nonspecific changes of ST line were detected. The diagnosis of acute myocarditis was established.

At the time of neurologic evaluation the patient's condition changed, there were suffusion-like exanthema on his legs and positive meningeal signs, so neurologist conclusion was suspicion of neuroinfection.

Patient status deteriorated, with progression of hypotension (BP 72/59) and centralized perfusion, with inadequate peripheral oxygenation (puls ox 88%), there were suffusion-like exanthema on his legs an positive meningeal signs. Patient was transferred to Resuscitation unit for complete resuscitation care and further evaluation.

On Department of Anesthesiology and Resuscitation, complete laboratory tests with lumbar puncture was performed, aggressive antibiotic and antivirotic therapy started, with intensive volumotherapy and ionotropic therapy. Despite this situation reached asystolia, with no measured BP, complete CPR was performed. There were akinetic left ventricle on ECHO, the patient was indicated for ECMO. After 3 hours of CPR ECMO was applied. During therapy, ECHO was repeated, which detected worsening dysfunction of left ventricle, with hypertrophy, edema and effusion, on laboratory, extremely high levels of cardioenzymes were detected.

Despite 11 hours of ECMO, situation led to akinesis of both ventricles with infaust prognosis.

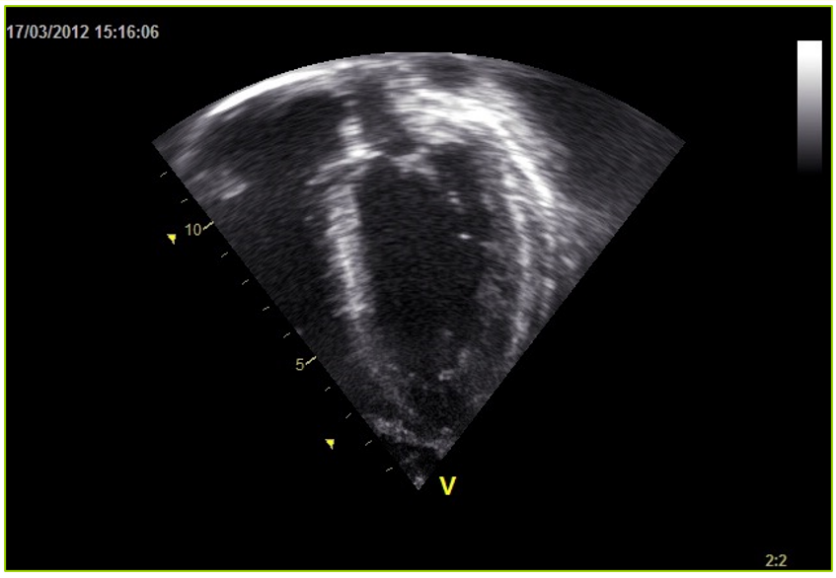


Figure 1: *Pericardial effusion.*

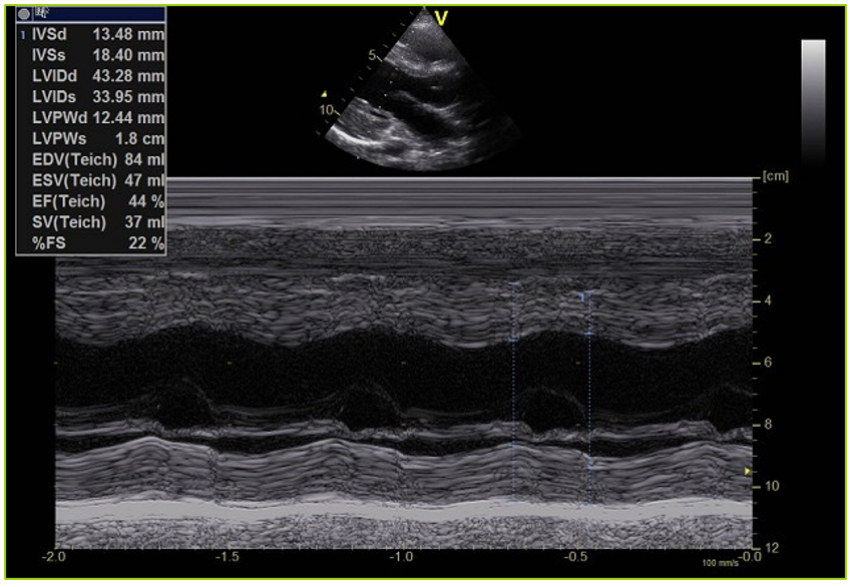


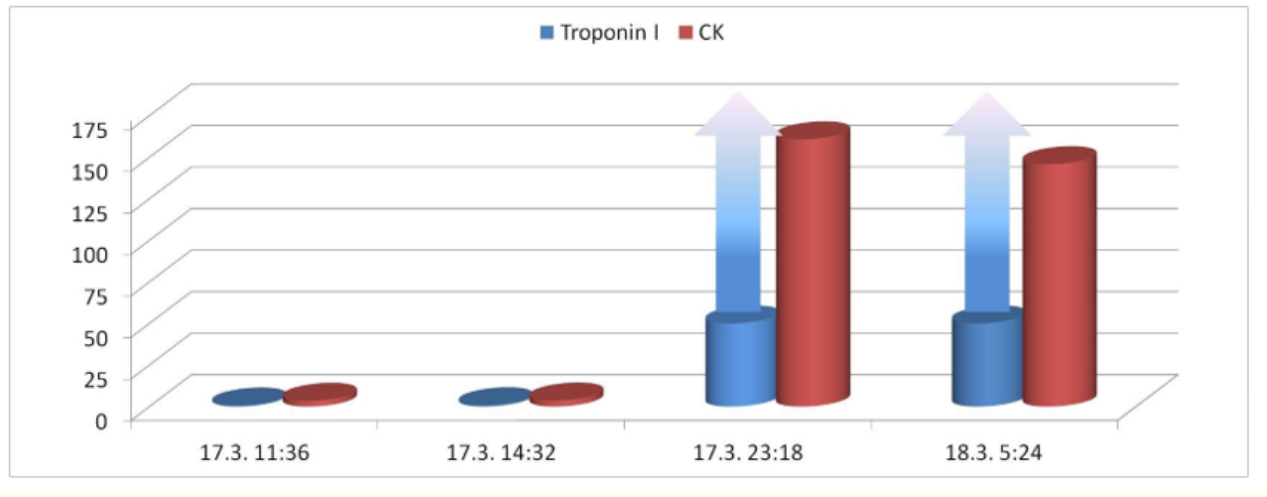
Figure 2: *Decreased contractility and edema of left ventricle.*

Time	Troponin I	CK	CK-MB	CK-MB mass	Myoglobin
17.3. 11:36	0,628	3,45	0,7		52
17.3. 14:32	781	3,61			
17.3. 23:18	50	160, 74		300	438
18.3. 5:24		145,8		300	

Table 1: *Markers of myocardial damage - laboratory values in time.*
Normal values: Troponin I 0.000 - 0.028 ug/l, CK 0.41 - 3.24 ukat/l, CK-MB 0.00 - 0.42 ukat/l CK-MB mass 0.00 - 7.20 ug/l, myoglobin 23.0 - 72.0 ug/l.

Other results:

- Serology: Negative for CMV, HSV, VZV, HHV 6, HHV 7, respiratory viruses, positive EBV anamnestic antibodies.
- Myocardial biopsy: Trace amount of EBV, HHV 7, adenovirus and parvovirus B17.
- Myocarial necropsy: Parvovirus B 17.
- Autopsy report
- Clinical diagnosis: Heart arrest, MOF, septic shock, cardiac failure, myocarditis
- Anatomic diagnosis: Tracheobronchitis haemorrhagica acute, bronchiolitis acute, lymphocytosis oedema pulmonum, pneumorrhagiae disperse, myocarditis acute lymphocytic, myomalacia larvae disperse, dilatation myogenes cordis



Figure

Discussion

Syncope in pediatric population is most often benign, but it could preceed life-threatening condition, especially syncopes of cardiac etiology. The evaluation on ED should be target to identify this life-threatening conditions.

Differential diagnosis of syncope:

- Life-threatening: Cardiac (electrical disturbances, structural heart diseases).
- Benign: Vasovagal, breath holding spells, orthostatic, drug effects, anemia, hypoglycemia, migraine.
- Conditions that mimic syncope (seizure, hysteria…).

The evaluation on PED should be target to identify this life-threatening conditions:

- Careful history: Be aware of this historical features: No prodromes, syncope during exertion, palpitation or chest pain, family history of congenital heart disease.
- Physical examination (vital signs, cardiac murmurs), neurologic and cardiologic examination.
- EKG, ECHO.
- Laboratory test: Glycemia, hematocrit, HCG test, urine toxicology.
- (Other laboratory tests: CBC, electrolytes, cardiac enzymes).
- (Chest X ray).

In our case the etiology of syncope was viral myocarditis. Detailed etiology remains unknown, influenza because of epidemiology situation or parvovirus B 19 because of necropsy result should be considered. What remains as a question is why was the course in this case fulminant. Where there any predisposed factors? [1-6].

Conclusion

Syncope in the pediatric population should be always completely evaluated.

Despite the fact that the majority of syncope in children is benign, some etiology should have fatal progression. Especially episodes accompany infection should warrant our alertness.

Acknowledgement

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