Journal of Cardiothoracic Surgery

CASE REPORT

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Fulminant myocarditis caused by influenza B virus in a male child: a case report and literature review

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Abstract

Background Influenza B virus induced myocarditis is a rare complication with potentially wide variations in severity and clinical presentation, and the pathogenesis is unclear.

Case presentation We describe a rare case of a 7-year-old boy who developed fulminant myocarditis (FM) due to influenza B virus infection. Treatment measures included mechanical ventilation, vasoactive agents, Extracorporeal membrane oxygenation (ECMO), Continuous Renal Replacement Therapy (CRRT), anti-inflammatory, antiviral, anti-infection, and enteral nutrition support. After 10 days of treatment, the patient succumbed to multiorgan failure.

Conclusions After a systematic review of the literature, we found that this disease predominantly affects females, with pediatric cases exceedingly rare. Fulminant myocarditis (FM) progresses rapidly, poses significant treatment challenges sporadic, and carries a poor prognosis. Interestingly, literature reports suggest that anti-thymocyte globulin therapy may have a positive impact in treating FM, potentially offering new insights into its pathogenesis and clinical management.

Keywords Myocarditis, Influenza B virus, Viral myocarditis, Fulminant myocarditis

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Background

Myocarditis is an inflammatory disease of the myocardium caused by viral infection, allergic reaction, and autoimmune etiologies, which in its acute phase can range from mild transient clinical symptoms to fulminant symptoms of acute heart failure, cardiogenic shock, or severe arrhythmia [1]. Viral myocarditis (VM) is an infection of the myocardium with cardiotropic viruses resulting in limited or diffuse inflammatory myocardial lesions, the common pathogens including coxsackieviruses, influenza viruses and adenoviruses and so on, about 12.5% of patients with VM can develop into dilated cardiomyopathy (DCM) and then congestive heart failure [2]. Fulminant myocarditis (FM) is a clinical syndrome of suspected myocarditis with varying degrees of cardiogenic shock and arrhythmias. FM accounts for approximately 10% of diagnosed myocarditis and may have a completely different etiology and pathogenesis than myocarditis, requiring circulatory support and even extracorporeal membrane oxygenation (ECMO) or intra-aortic balloon pump (IABP) in many cases, with most patients can recover [3]. The incidence of myocarditis caused by influenza viruses has increased since influenza A (H1N1) outbroke in 2009 [4]. However, FM caused by influenza B virus is rare. We present a case of FM caused by Influenza B virus in a child and describes the management of the case and literature review.

Case presentation

The patient, a 7-year-old boy, admitted to the local hospital on 24 February 2022 due to intermittent fever for 3 days with the maximum body temperature of 38.3 °C, projectile vomiting twice, lethargy and was initially diagnosed with acute myocarditis. He presented a generalized convulsive crisis that night which was relieved by intramuscular injection of 5 mg diazepam. For further treatment, the child was transferred to our hospital and admitted to the pediatric intensive care unit (PICU) at 23:00 in the evening. On admission, the patient was sanity, axillary temperature was 36.4 °C, pulse rate was168 per minute, COVID-19 nucleic acid was negative, electrocardiogram revealed part of ST-segment elevation, N-terminal pro-brain natriuretic peptide (NT-pro BNP) was 5094Pg/ml, Nasopharyngeal swab tested positive for influenza B virus, cardiac troponin I (cTnI) was 169.6Pg/ml, IL-6 was 1610Pg/ml, PCT was 70.75ug/L, aspartate transaminase (ALT) was 84U/L, total protein was 54.29 g/L, albumin was 34.21 g/L, lactate dehydrogenase (LDH) was 522IU/L, arterial lactate was 5.7mmol/L, alpha hydroxybutyric acid dehydrogenase was 398IU/L, creatine phosphokinase was 240IU/L, creatine kinase isoenzyme was 233IU/L, creatinine was 34umol/L. Point-of-care color doppler echocardiography exhibited the diffuse reduction of motion amplitude in left ventricular wall and pericardial effusion existing. The next day at 3:40, the child experienced sudden respiratory and cardiac arrest, with the electrocardiogram monitor showing ventricular fibrillation. Immediate interventions included CPR, endotracheal intubation, mechanical ventilation, intravenous administration of adrenaline at 1.0 mg every 3 min, and two rounds of defibrillation (2 J/kg). The child successfully resuscitated after 25 min. Though the aggressive vasoactive agents therapy was conducted, hemodynamic impairment of the child was persistent. At 6:30 in the next day, the child received veno-arterial extracorporeal membrane oxygenation (VA-ECMO) support via the right femoral artery and vein. At 8:00 the child was transferred to the emergency ICU (EICU) for continued treatment, but subsequently developed multiple organ failure and severe lower limb ischemia. Treatment included continuous renal replacement therapy (CRRT), vasoactive agents, anti-inflammatory therapy with methylprednisolone (200mng twice daily, reduced after three days), anti-virus (oseltamivir, interferon), anti-infection (cefperazone-sulbactam) therapy, pericardiocentesis and drainage and enteral nutrition. The volume of pericardial puncture drainage on the third day following admission to our hospital was 350 ml.

On the 4th day after admission, craniocerebral CT showed extensive cerebral oedema, and lung CT showed bilateral hydrothorax (see Fig. 1). So, the dehydration and decompression therapy were given.

On the 5th day, ECMO was evacuated from the patients, and the color doppler echocardiography showed a reverse flow signals in the bicuspid and tricuspid valves, pericardial effusion, slightly enlarged left ventricle, thickening of the left ventricular wall, and a left ventricular ejection fraction (LVEF) of 38.5%. Transcranial Doppler (TCD) ultrasound revealed abnormal spectrum of bilateral middle-cerebral arteries, with a peak systolic velocity (PSV) of 39.4 cm/s and an end diastolic velocity (EDV) of -21.1 cm/s (early diastolic flow reversal) on the right side, and PSV of 24.1 cm/s and EDV of -14.1 cm/s (early diastolic flow reversal) on the left side, with unclear flow in bilateral anterior and posterior cerebral arteries, and the immunological test for Aspergillus was positive (Aspergillus galactomannan: 1.36 ug/L); On day 9, the child suffered a cardiac arrest at 10:37, the chest compression and intravenous injection of 1 mg epinephrine twice were immediately conducted, and after 1 min of resuscitation, the patient restored the spontaneous circulation. But unfortunately, at 8:20 on the 10th day of admission, the child suffered cardiac arrest again and died. The results of the relevant laboratory tests of the patient during the treatment process are shown in Table 1.

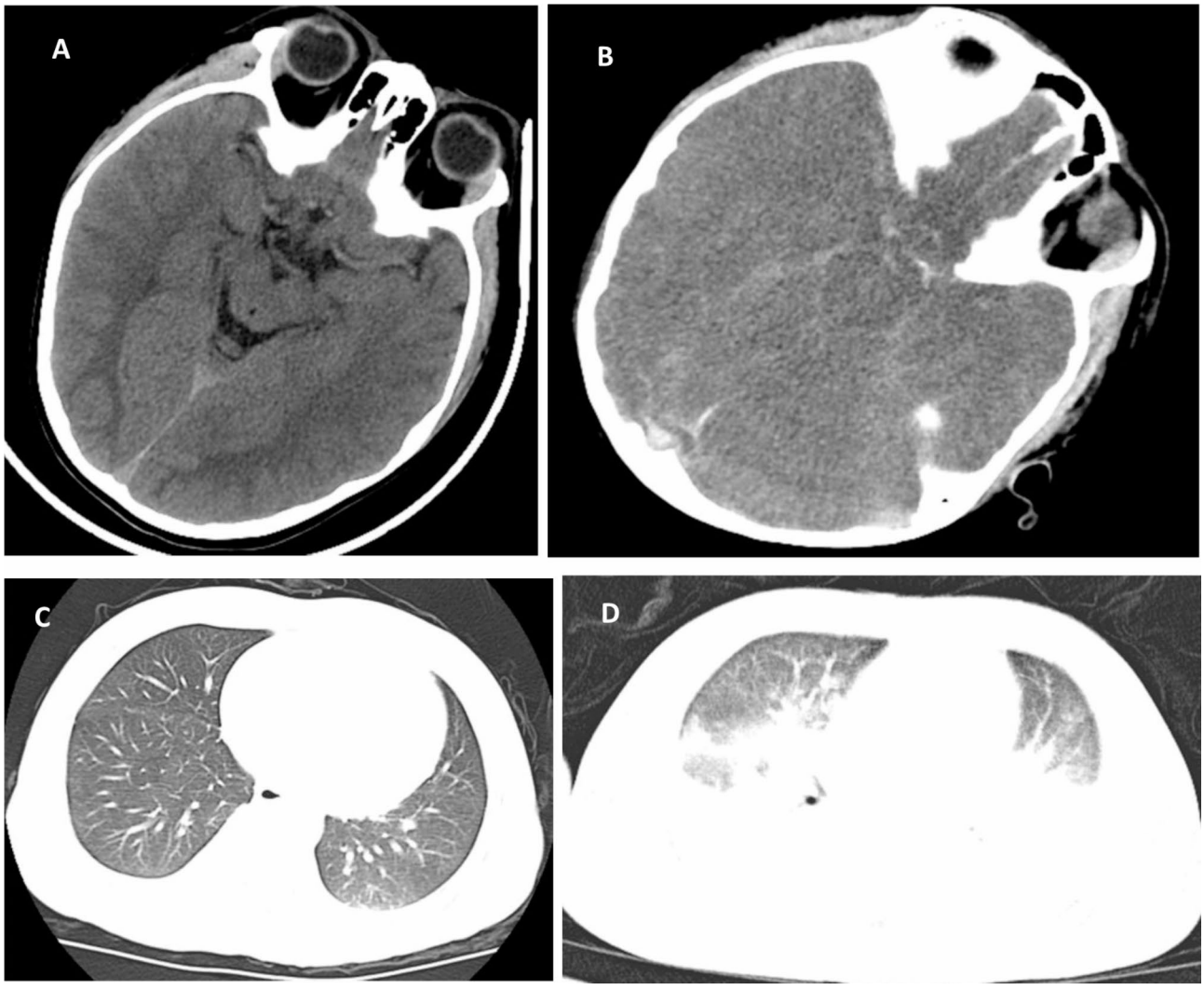

Fig. 1 Head and chest imaging findings during the patient's treatment. On the day of admission, the child's CT scan of the brain and lungs showed no abnormalities (**Image A, C**); On the fourth day of admission, the child's CT scan of the brain revealed encephalocoele shrinkage, increased density, extensive brain edema, diffuse brain ischemic swelling with subarachnoid hemorrhage (**Image B**), and the lung CT scan showed pleural effusion and incomplete expansion of the lower lungs (**Image D**)

Table 1 Results of laboratory tests during the patient's treatment

Laboratory test	Reference range	Time							
		Day 1	Day 2	Day 3	Day 3	Day 4	Day 5	Day 6	Day 7
WBC (10^9/L)	3.5 ~ 9.9	17.37	6.58	11.23	12.59	14.60	15.12	14.14	19.67
Neutrophil count (10^9/L)	1.8 ~ 6.3	12.29	5.87	10.39	11.65	12.92	13.55	12.43	16.57
Neutrophil percentage (%)	40.0 ~ 75.0	70.8	89.2	92.5	92.5	88.4	89.6	87.9	84.1
CRP (mg/L)	0 ~ 10	1.83	8.51	22.70	1.87	/	7.80	5.36	/
CK (IU/L)	50 ~ 310	8650	66,170	70,270	8956	27,457	19,252	10,462	4662
CKMB (IU/L)	0 ~ 25	540	714	1055	563	604	611	588	480
LDH (IU/L)	120 ~ 250	923	2918	4825	929	5141	6410	4762	4586

WBC: white blood cell. CRP: C-reactive protein. CK: creatine kinase. CKMB: creatine kinase isoenzymes. LDH: lactate dehydrogenase

Literature review

We performed a literature review of all fulminant myocarditis (FM) caused by influenza B virus infection cases within the time frame from January 1, 2000 to July 1, 2023. A total of 52 articles were retrieved from PubMed, Web of Science, CNKI and Wanfang databases identified that mention the following search keywords (combination of MeSH and non-MeSH terms): ("Influenza B virus"

OR "Influenza B viruses" OR "Orthomyxoviruses Type B" OR "Influenza Viruses Type B") AND ("Myocarditis" OR "Myocarditides" OR "Carditis" OR "Fulminant myocarditis". Out of the retrieved literature, 14 case reports were identified after excluding reviews, conference papers, and incomplete data [4–17] (see Table 2). Combining the information from these case reports with 14 other relevant articles, we found that FM induced by influenza B virus infection mostly occurred in adult females, with pediatric cases being rare in recent years. FM associated with influenza B virus infection was found to have a relatively severe prognosis, often leading to acute heart failure, cardiogenic shock, and other severe complications in a short period. The disease progresses rapidly, difficulty in therapy, which may involve interventions such as intra-aortic balloon pump (IABP), ventricular assist devices (VAD), ECMO, anti-inflammatory and anti-infective therapies, hemodialysis, and symptomatic supportive treatment. The prognosis of some patients was reported to be poor.

Discussion

Influenza virus is one of the main causes of acute respiratory infections in adults, affecting 5–15% of the global population yearly [18]. Though myocarditis is a rare complication of influenza, its incidence has increased in recent years due to widespread influenza virus outbreaks, and the true rate of occurrence may be underestimated [19]. While researches have indicated a significant association between the influenza, acute respiratory infections and acute myocardial infarction, but the clinical data on the risk factors and complications of influenza B are still limited [20]. Current studies reported that most influenza myocarditis cases are associated with influenza A virus [21] with only 8% of cases resulting from influenza B virus infection [4]. The clinical manifestations of influenza B infection are always mild and has a good prognosis, with the fatal complications being rare [22]. In this case, though we not confirm the presence of influenza B virus in myocardial tissue, the detection of influenza B virus in throat swabs was positive, as well as the existing of specific IgA and IgG antibodies in the serum, and the absence of other causes of myocardial damage. All those evidences support a direct association between influenza B virus infection and acute myocarditis.

This study retrospectively reviewed 15 cases of influenza B virus myocarditis of which 11 cases were female (73.3%) and all were diagnosed with fulminant myocarditis (FM), which indicating a gender difference in the occurrence and progression of this disease. Studies have reported that the H1N1 influenza virus has tropism for striated muscle and is prone to causing rhabdomyolysis, which can lead to acute renal failure [23]. Although the pathophysiology mechanisms of this process are still debated, some scholars have proposed the viral toxin circulation hypothesis, which indicated that the virus can penetrate various muscle tissues including skeletal muscle and myocardium, leading to muscle damage [24]. Till now, the pathophysiological mechanisms of FM are not fully understood, but some theories have been proposed to explain myocardial injury. For example, some researcher indicated that viral proteases can mediate the direct myocardial cell damage and induce widespread and early cell lysis, lead to the development of heart failure as a result [25, 26]. This process also activates $CD4^+$ T lymphocytes and drives their differentiation to the Th1 and Th17 cells, which may also promote inflammation by activating macrophages, neutrophils, $CD8^+$ T lymphocytes and B lymphocytes, releasing autoantibodies against cardiac cells, and sustaining the myocardial damage [27]. In addition, the cytokine storm hypothesis can better explain the acute phase of myocardial damage, as it indicated that the release of inflammatory cytokines such as IL-1,IL-6 and tumor necrosis factor-α (TNF-α) were account for the fulminant property of the disease [25].

In the literature review and the case we reported, patients always got exacerbation in a short time, with dying less than 3 days after admission, which indicated that influenza B virus myocarditis often progressed rapidly and could develop into fulminant myocarditis easily. Besides, all the patients in the literature we retrieved had hemodynamic deterioration with a poor response to the vasoactive agents, and 8 of them received extracorporeal membrane oxygenation (ECMO) for circulation support. The Circulation support, especially in the early stages of the disease, may play a key role in preventing the fatal outcome.

Early diagnosis of FM is always hard to achieved, for the cardiogenic shock often occurring before viral test results are available. Laboratory test typically shows the elevated cardiac damage biomarkers and observable changes in electrocardiograms [28]. Echocardiograms and cardiac magnetic resonance imaging (MRI) can assess the impaired cardiac function and revealed the areas of inflammation, necrosis and fibrosis [29, 30]. While the histopathological examination, is the gold standard for diagnosing FM, but its invasiveness limits its availability [31]. Therefore, FM is always an etiological diagnosis in the clinical situation. The treatment of FM at present often focus on the early-stage antiviral therapy and timely circulation support. Some cases also have shown the potential safety and effectiveness of anti-thymocyte globulin (ATG) in treating severe acute viral myocarditis in children., which can play a protective by reducing the antibodies against the viral and cardiac antigens and cytokines produced by T lymphocytes [15]. This suggests a new approach and direction for clinical treatment, but

Table 2 Case report information of 15 cases of fulminant myocarditis caused by influenza B virus

Author	Country	Gender/Age(years)	Diagnosis	Medical history	Treatment	Prognosis	Time(day)
Frank H 2010 [5]	Germany	female/5	Fulminant myocarditis	upper respiratory tract infection, cough, fever (39 °C), cardiogenic shock	dopamine, dobutamine, adrenalin, mechanical ventilation	Death. Postmortem autopsy shows the entire heart was highly dilated.	1
Tabbutt S 2004 [6]	USA	female/4.5	Fulminant myocarditis	cardiogenic shock, serum lactate of 6.4 mmol/L, White blood cell count of 13.6×10^9/L, CK 203,412 U/L, pancreatitis	milrinone, dopamine, dobutamine, mechanical ventilation, ECMO, plasma exchange	Survival, A small atrial septal defect after 2 years, other developed normal	31
Muneuchi J 2009 [7]	Japan	male/15	Viral myocarditis	acute heart failure、cardiogenic shock、fever, CRP 41 mg/L, CK 624 IU/L、LDH236 IU/L	zanamivir, isosorbide dinitrate, nifedipine	Survival	7
Marchetti L 2019 [8]	Italy	male/44	Viral myocarditis	heart failure, cardiogenic shock、fever, LVEF 15%, pericardial effusion	dobutamine, norepinephrine, mechanical ventilation, ECMO, IABP, antibiotic	Survival At the 90-day follow-up visit, the patient was alive without significant cardiologic abnormalities.	17
Roto D 2018 [9]	USA	female/57	Fulminant myocarditis	fever (39.4℃), Septic shock, a large pericardial effusion with tamponade physiology, asystolic cardiac arrest, serum lactate of 16.2 mmol/L	norepinephrine, antibiotic, mechanical ventilation、pericardiocentesis	Death The endomyocardial biopsy shows multifocal cardiac cell necrosis and subendocardial septal hemorrhage、CD3(+)、strongly positive viral PCR	1
Taremi M 2013 [10]	USA	female/52	Fulminant myocarditis	fever, septic shock、LVEF10%、pseudoaneurysm of the left common femoral artery secondary	norepinephrine, dobutamine, vasopressin, antibiotic, mechanical ventilation, ECMO	Survival Transthoracic echocardiography one month after discharge demonstrated mitral regurgitation.	7
Siskin M 2017 [11]	USA	female/22	Fulminant myocarditis	cardiogenic shock, pericardial effusion	dobutamine、steroids 、oseltamivir	Survival The endomyocardial biopsy showed focal myocyte necrosis with lymphocytes infiltration	8
Hékimian G 2018 [12]	France	female/28	Fulminant myocarditis	LVEF 30%, pericardial effusion, serum lactate 9.8 mmol/L	oseltamivir, dobutamine, ECMO	Survival	6
Elikowski W 2018 [13]	Poland	female/89	Viral myocarditis	fever(39℃), heart failure, LVEF24%	oseltamivir	Survival	10
Dickey T 2018 [14]	USA	female/34	fulminant myocarditis	bilateral pleural effusions, septic shock, pulmonary edema	ceftriaxone, vancomycin, and doxycycline	Death	1
Silva E 2019 [15]	Columbia	female/21	Fulminant myocarditis	Heart failure, cardiogenic shock, pericardial effusion, asystolic cardiac arrest, cTnI 248 ng/L, multiple organ dysfunction	mechanical ventilation, ECMO, oseltamivir、CRRT	Death	3
		female/62	Fulminant myocarditis	heart failure, pericardial effusion, LVEF 15%, cTnI 13,373 ng/L	oseltamivir、IABP	Survival	8
Jenna A 2018 [16]	USA	Female/13	Fulminant myocarditis	heart failure, upper respiratory tract infection, chest pain, pericardial effusion	ECMO, oseltamivir, glucocorticoids, anti-thymocyte globulin, milrinone, epinephrine	Survival	32

Table 2 (continued)

Author	Country	Gender/Age(years)	Diagnosis	Medical history	Treatment	Prognosis	Time(day)
Noreen M 2023 [17]	USA	male/45	Fulminant myocarditis	exertional dyspnea, lower extremity edema, sinus rhythm with T wave inversion, LVEF decreased by 30–35%, right coronary artery stenosis, cTnI 1876 ng / L	antibiotics, oseltamivir, remdesivir, heparin, aspirin, atorvastatin, vasopressors, IABP	Survival	Not mentioned
Kyle Murnaghan 2022 [18]	Canada	male/53	Myocarditis	pneumonia, dyspnea, cTnI 3871 ng / L, blood lactic acid 9.7 mmol / L, heart failure, LVEF 15–20%, pericardial effusion, influenza B, intraosseous pressure 60 mmHg, sacral pitting edema	furosemide, vancomycin, piperacillin, oseltamivir, noradrenaline	Survival	12

IABP: Intra-aortic balloon pump. LVEF: Left ventricular ejection fractions. ECMO: Extracorporeal membrane oxygenation. CTnI: cardiac troponin I (reference range: 0.00-15.6 ng/L). CRRT: Continuous renal replacement therapy

further clinical studies are needed to elucidate the benefits of ATG for FM patients.

Conclusion

Influenza B fulminant myocarditis is rare clinical situation, especially in children, but with a slightly higher incidence in adult females. The progression of influenza B fulminant myocarditis is rapid, with a high risk of poor prognosis, requiring early initiation of antiviral drugs and circulation support such as ECMO and AIBP in treatment. Currently, the pathogenesis of FM remains unclear both domestically and internationally. Research on early diagnosis and treatment of FM plays a crucial role in improving survival rates and patient outcomes.

Author contributions

Investigation: Fei Tian, Zhekang Peng, Fu Ni, Yuqing Fan, Zhaohui Zhang. Methodology: Fei Tian, Yi Xiao, Zhekang Peng. Project administration: Fei Tian, Zuyang Xi, Zhaohui Zhang. Writing-original draft: Fei Tian. Writing-review & editing: Yi Xiao, Zhekang Peng.

Funding

No funding is available.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Helsinki Declaration. All methods were performed in compliance with relevant guidelines and regulations. The study was approved by the Ethics Committee of Zhongxin People's Hospital of Yichang City (Approval No. 2022-080-01). All participants in the study provided informed consent and had the right to withdraw from the study at any time for any reason. Furthermore, they were assured that the survey would be used only for research purposes.

Competing interests

The authors declare no competing interests.

Received: 6 March 2024 / Accepted: 13 August 2024
Published online: 24 August 2024

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