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Acute respiratory failure in a healthy adult with human parvovirus B19 infection

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Abstract

Human parvovirus B19 infection is generally known as childhood erythema infectiosum. Most adults infected with parvovirus B19 develop mainly flu-like symptoms with arthritis. Few cases of life-threatening parvovirus infections in immunocompetent adults have been reported to date. We report a previously healthy, middle-aged male who developed severe acute respiratory failure due to parvovirus B19 infection. The patient recovered with supportive care, including non-invasive positive pressure ventilation. Parvovirus B19 may represent a severe cause of acute respiratory failure.

Keywords

human parvovirus B19; acute respiratory failure

Introduction

Human parvovirus B19 infection is common worldwide and generally known as childhood erythema infectiosum. Most individuals are infected by 15 years of age. Infection continues at a lower rate throughout adult life, and by the age of 50 years, 80% of the population is seropositive [1]. In adults, symptoms in the first phase are mainly flu-like, including fever, headache, or myalgia. Symptoms in the second phase are arthralgia, rash, and edema [2-4]. Generally, parvovirus B19 infection in immunocompetent individuals displays a self-limiting course. However, a few cases of severe respiratory failure in immunocompetent hosts have been reported in the literature. Herein, we present a case of an adult male patient with acute respiratory failure who was eventually diagnosed with parvovirus B19 infection.

Case Report

A previously healthy, 37-year-old Japanese man complaining of fever, rash, headache, and dyspnea had been admitted to a local community hospital during the winter season of Japan (day 1). He had smoked one pack of cigarettes a day for 17 years. His 3-year-old son who lived with him had also exhibited a rash on his face and was diagnosed with erythema infectiosum. His respiratory condition had worsened despite being treated with ciprofloxacin (600 mg/day), meropenem (1.5 g/day), and intravenous immunoglobulin(IVIg) (5 g/day). Blood cultures and sputum cultures were negative. He was

transferred to our hospital on day 3. His physical findings on day 3 were as follows: body temperature = 38.6°C, blood pressure = 147/86 mmHg, heart rate = 90 beats/min, respiratory rate = 30 /min, $SpO_2$94% (reservoir mask 12 L/min); and coarse crackles in all lung fields on both sides. Laboratory findings on day 3 are shown in Table 1. Laboratory findings showed normal creatinine kinase levels(151 U/L; normal: 62—287 U/L), but elevated brain natriuretic peptide (BNP) levels(197.4 pg/mL; normal: 0—18.4 pg/mL).

There were no electrocardiograph changes. A transthoracic echocardiogram showed good motion of cardiac walls, normal ejection fraction of 67%, and no right ventricular overload. Chest radiograph demonstrated bilateral pulmonary infiltration and pleural effusion (Fig. 1). The PaO_2/FiO_2 ratio was 110, indicating acute respiratory failure. He was placed on a non-invasive positive pressure ventilator and treated with methylprednisolone pulse therapy (1 g/day), sivelestat sodium (300 mg/day), and torasemide (8mg/day), additively.

History and physical findings strongly suggested parvovirus B19 infection. He was finally diagnosed with parvovirus B19 infection due to serum elevation of anti-parvovirus B19 immunoglobulin M (IgM) and a polymerase chain reaction (PCR)-positive parvovirus B19 DNA result on day 3 (Table 1). The patient's respiratory condition subsequently improved, and he was ultimately discharged with complete recovery on day14 (Fig. 2).

Discussion

This is a case of acute respiratory failure in a healthy adult with evidence of recent parvovirus infection. Diagnosis of parvovirus B19 was based on increased antibody titers. Elevation in the IgM titers are seen 7–10 days after the initial infection and can remain elevated up to 1 month [5]. Although the DNA PCR test is available and is more sensitive, parvovirus B19 DNA can be found months to years later depending on the source of the sample. Therefore, a positive test does not necessarily indicate an acute infection [6,7].

The first case of respiratory failure caused by parvovirus B19 infection in an immunocompetent adult was reported in 1998 [8], and there have since been a few reports suggesting that parvovirus B19 induces respiratory failure [9-11]. Although the treatment protocol for acute respiratory failure with parvovirus B19 infection has not yet been established, treatment of IVIg resulted in decreased viremia and improvement of symptoms and has a curative role in parvovirus B19-associated chronic fatigue syndrome and pure red cell aplasia [12-14]. We believe that IVIg was beneficial in this case.

In this case, although there was no direct evidence of heart failure on the echocardiogram, bilateral pulmonary infiltration and pleural effusion on the chest X-ray and elevated BNP levels indicated that there may have been an involvement of congestive heart failure as a cause of acute respiratory failure.

A limitation of this study is that it is unclear whether lung injury resulted from direct infection or immune response of the host because lung biopsy or bronchoalveolar lavage could not be performed due to poor respiratory conditions. It has been reported that the cellular receptor for parvovirus B19, erythrocyte P antigen, is expressed not only in erythroid cells but also in other tissues, including the lung [15].

Figures

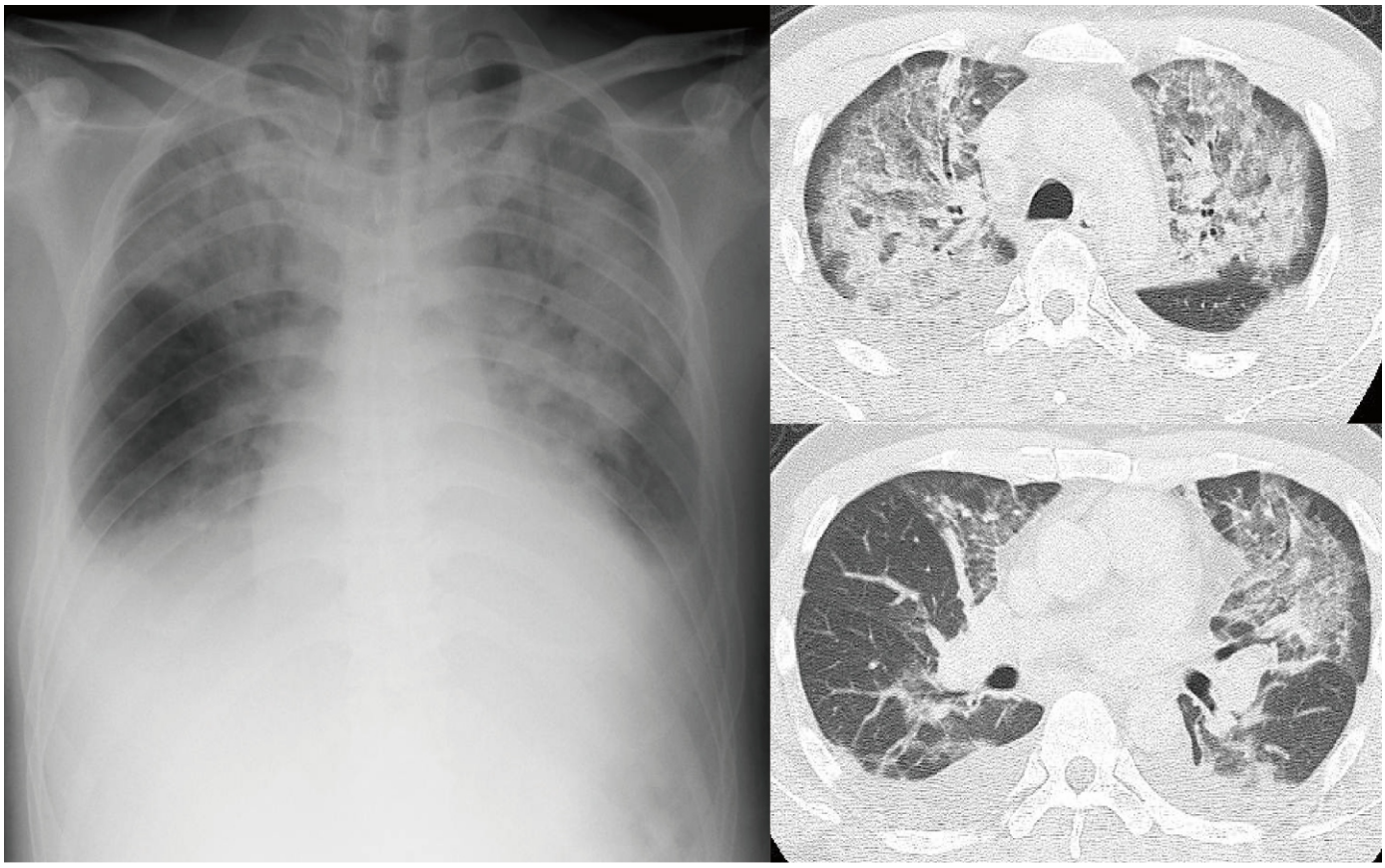

Figure 1: Chest imaging findings on admission of a 37-year-old male patient with acute respiratory failure due to parvovirus B19 infection.

A chest X-ray showed diffuse pulmonary infiltration. A CT scan showed bilateral consolidation with an air bronchogram, ground glass opacity, and pleural effusion.

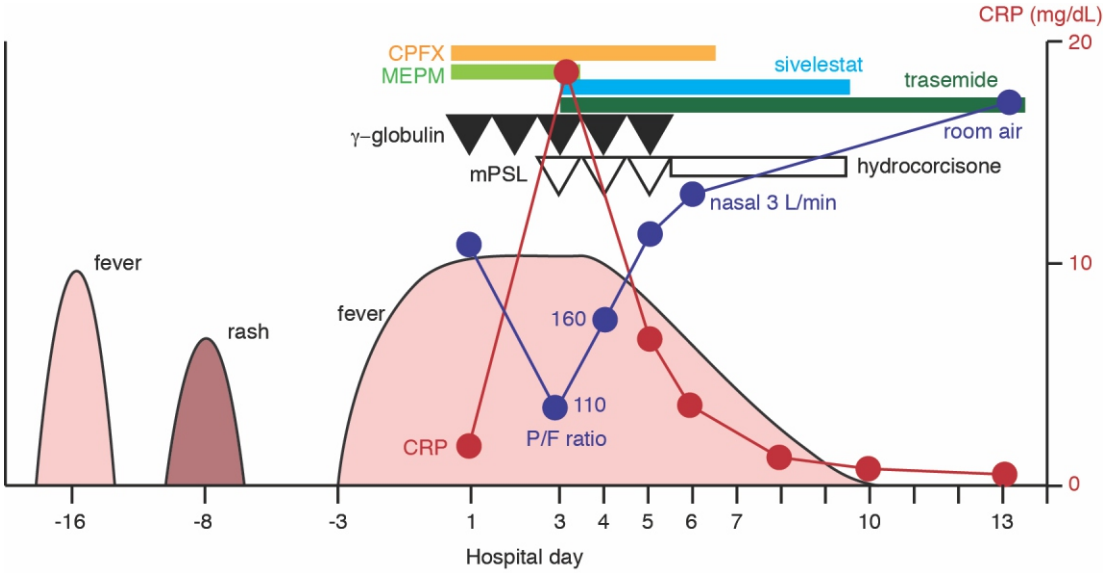

Figure 2: Clinical course of a 37-year-old male patientwith acute respiratory failure due to parvovirus B19 infection.

The patient's clinical course showed improvements in the inflammatory response and oxygenation following the administration of a gamma globulin and corticosteroids. The patient was discharged on hospital day 16. Abbreviations: CPFX; ciprofloxacin, MEPM; meropenem, P/F ratio; PaO_2/FiO_2 ratio, mPSL; methylprednisolone, CRP; C-reactive protein.

Table

Table 1: Laboratory findings for a 37-year-old male patient with acute respiratory failure due to parvovirus B19 infection.

Hematology			Arterial blood gas (CPAP, FiO_2 0.6)		
WBC	17210	/μL	pH	7.4	
Neut	88.5	%	PCO_2	40.7	Torr
Lymph	7.1	%	Po_2	66.6	Torr
Mono	4.0	%	HCO_3-	24.4	mmol/L
Eos	0.2	%	$AaDO_2$	588.4	Torr
Baso	0.2	%	BE	-0.4	mmol/L
RBC	3.65×10^6	/μL			
Hb	11.0	g/dL			
Ht	33.2	%			
Plt	41×10^4	/μL			
			Serology		
Biochemistry			CRP	17.7	mg/dL
TP	6.2	g/dL	KL-6	144	U/mL
Alb	3.1	g/dL	Sp-D	44.8	ng/mL
T.bil	0.6	mg/dL	Sp-A	76.6	ng/mL
AST	21	IU/L	BNP	197.4	pg/mL
ALT	20	IU/L	HIV-Ab	(−)	
LDH	379	IU/L	β-D-glucan	<4.101	pg/mL
BUN	19	mg/dL	Parvovirus B19 IgM	8.33 (+)	(< 0.80)
CRE	1.0	mg/dL			
Na	139	mol/L	Microbial examination		
K	4.5	mol/L	Urinary Ag for *Streptococcus pneumoniae*(−)		
Cl	104	mol/L	Urinary Ag for *Legionella pneumophila*(−)		
Glu	105	mg/dL	Influenza rapid diagnostic test A (−), B (−)		
CK	151	U/L	Parvovirus B19 DNA PCR (+)		

Conclusion

In conclusion, we herein reported a case of acute respiratory failure caused by parvovirus B19 infection. Although the prevalence of acute respiratory failure due to parvovirus B19 is very rare, physicians must consider this setting in the differential diagnosis of acute respiratory failure.

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