Rocky Mountain Spotted Fever Presenting with Chest Pain and Electrocardiogram Ischemic Changes

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R ocky Mountain spotted fever classically presents with the triad of fever, rash, and history of tick exposure. Although it is a life-threatening disease, symptoms and signs may vary from very mild to severe. These signs and symptoms may include headache, nausea, vomiting, abdominal pain, conjunctivitis, splenomegaly, hepatomegaly, pneumonitis, coma, seizures, or shock.¹ When signs and symptoms other than fever, headache, and rash are the presenting complaint, the diagnosis may be difficult to make, thus delaying appropriate treatment. The following case illustrates an unusual presentation for Rocky Mountain spotted fever with the acute onset of crushing substernal chest pain, electrocardiogram (ECG) changes, and subsequent left upper quadrant abdominal pain with splenomegaly occurring before the onset of rash and fever.

CASE REPORT

A 55-year-old woman came to her family physician complaining of mild substernal chest discomfort of one week's duration that progressed to severe, crushing, substernal chest pressure several hours prior to presentation. It was described as a band-like sensation of "someone sitting on my chest" and was worse with exercise. The severe chest pressure was accompanied by diaphoresis and headache. Administration of nitroglycerin had failed to relieve the pressure, but an ECG revealed a sinus rhythm with the new onset of ST segment depression. The patient's cardiac history was negative except for an episode of chest pain 3 years previously, which was followed by nondiagnostic findings on thallium stress testing. There was no history of recent exposure to infectious disease in general. She denied nausea, vomiting, diarrhea, fever, or pain anywhere else.

On initial physical examination, the patient was diaphoretic and afebrile, with no rash or tick noted. Her heart rate was normal with only a mild sinus arrhythmia. The lungs were clear to auscultation, and examination of the abdomen was normal. There was no detectable splenomegaly.

The patient was admitted to the hospital with chest pain of unknown cause that was suggestive of possible myocardial infarction. Pertinent laboratory findings throughout the hospitalization are listed in Table 1. The initial ECGs revealed a sinus rhythm with anterolateral ST segment depression and T wave inversion, which were not present on an ECG done 1 year ago. Lactate dehydrogenase, creatine phosphokinase, and SGOT levels rose steadily until initiation of effective antibiotic treatment, but tests were negative for cardiac muscle isoenzymes.

The severe chest pressure improved during the 1st day of hospitalization. Two days after admission, however, the patient became febrile, developed left upper quadrant abdominal pain, and had worsening of the headache. On the 5th day after admission, a maculopapular, fine, erythematous rash was observed on the proximal extremities, sparing the palms and soles. Over the next 2 days the rash extended onto the trunk and distal extremities and became more pronounced. Although the fever persisted, there were no signs of cough, shortness of breath, or pleuritic pain.

Cardiac catheterization performed on the 3rd day after admission showed normal coronary arteries and normal left ventricular function. After the cardiac catheterization, further studies were done in an attempt to diagnose the cause of the chest pressure and the developing abdominal pain. These studies included an echocardiogram and a ventilation perfusion scan, the findings on both of which were normal. A search for causes of the abdominal pain included liver and spleen scan followed by a computerized tomography scan and an intravenous pyelogram. The liver and spleen scan and computerized tomography scan revealed splenomegaly. The intravenous pyelogram was continued on page 585

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Laboratory and Clinical Findings	Day																
	11	12	13	14	15	16	17	August 18	19	20	21	22	23	24	25	Septer 1	nbei 2
Leucocyte count 10º/L (10 mm ³)		4.9 (4.9)		5.1 (5.1)	4.5 (4.5)	4.8 (4.8)	5.1 (5.1)	4.9 (4.9)	5.7 (5.7)	5.8 (5.8)	5.7 (5.7)	6.0 (6.0)	5.3 (5.3)	5.6 (5.6)		(3.2)	3.2
Bands 1(%)		.13 (13)		.22 (22)	.31 (31)	.24 (24)	.24 (24)	.12 (12)	.17 (17)	.22 (22)	.10 (10)	.12 (12)	.15 (15)	.06 (06)			0 (0)
Hemoglobin g/L (g/dL)		144 (14.4)		132 (13.2)	122 (12.2)	112 (11.2)	108 (10.8)	108 (10.8)	108 (10.8)	108 (10.8)	97 (9.7)	100 (10.0)	93 (9.3)	103 (10.3)		(111
Reticulocyte count 10 ⁻³ (%)		26 (2.6)		38 (3.8)	43 (4.3)	45 (4.5)	47 (4.7)	60 (6.0)	63 (6.3)	37 (3.7)							
Erythrocyte sedimentation rate mm/h (mm/h)		14 (14)			33 (33)			49 (49)									40 (40)
Bilirubin (total) µmol/L (mg/dL)		23.94 (1.4)			29.07 (1.7)		23.94 (1.4)	27.36 (1.6)						22.23 (1.3)			
Bilirubin direct μmol/L (mg/dL)		3.42 (.2)			3.42 (.2)		5.13 (.3)	6.84 (.4)						3.42 (.2)			
Alkaline phosphatase µkat/L (IU/mL)		1.0 (59)			1.0 (63)			1.6 (98)						2.3 (135)			
Lactate dehydrogenase µkat/L (IU/mL)	4.63 (276)	4.95 (297)		8.54 (512)	9.32 (559)		10.19 (611)	11.07 (664)	10.94 (656)	10.62 (637)	9.40 (564)	8.80 (528)		7.98 (479)			
SGOT µkat/L (IU/mL)		.88 (53)			1.22 (73)			2.08 (125)						1.35 (81)			.67 (40)
SGPT µkat/L (IU/mL)		.58 (35)			.78 (47)			1.63 (98)						1.68 (101)			.73 (44)
Creatine kinase µkat/L (IU/mL)	2.53 (152)	1.95 (117)			1.00 (60)			.52 (31)									.67 (40)
Gamma glutamyl transferase µkat/L (IU/mL)		.55 (33)			.65 (39)			1.47 (88)						2.25 (135)			.90 (54)
Sodium mmol/L (mEq/L)	134 (134)	131 (131)		129 (129)	130 (130)	132 (132)	129 (129)	130 (130)	133 (133)	131 (131)	133 (133)	135 (135)					138 (138
Chloride mmol/L (mEq/L)	91 (91)	93 (93)		90 (90)	92 (92)	91 (91)	90 (90)	95 (95)	95 (95)	95 (95)	95 (95)	98 (98)					100 (100
Left upper quadrant pain				+	+	+	+	+	+	+							
Headache		+	+	+	+	+	+	+	+	+							
Rash	1				+	+	+	+	+	+	+	+	+	+	+	+	+
Tetracycline administration									+	+	+	+	+	+	+	+	+
Maximum daily temperature, °C	36.8	38	38.8	37.2	39	38.8	38.2	38	38.6	38.8	39	38.4	37.2	37	37	37	37

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nondiagnostic. Complete blood counts showed a moderate left shift with a normal white cell count and red cell changes consistent with hemolytic anemia. Platelet counts remained normal as did the coagulation studies, prothrombin time, and partial thromboplastin time. Sigmoidoscopy was done during the search for the cause of the abdominal pain, fever, and anemia; however, findings were normal except for two polyps later removed during colonoscopy.

Because of her persistent fever, the left shift on blood counts, and worsening symptoms, the patient was started on antibiotic therapy with ceftriaxone 5 days following admission. Ceftriaxone was used in an attempt to offer broad-spectrum coverage for suspected bacterial infection. The antibiotic was changed 3 days later to tetracycline, as the possibility of rickettsial disease increased. Two days after the patient started tetracycline, her headache and left upper quadrant pain disappeared. By the 3rd day of tetracycline, the patient was afebrile. Skin biopsies of the rash were reported as "mild chronic nonspecific dermatitis."

Febrile agglutinin tests obtained on the day of beginning tetracycline therapy showed a positive OX19 titer of \geq 1:320. This titer is higher than that of <1:20 for parainfluenza A and B, brucellosis, typhus H, and salmonella A; <1:40 for parainfluenza C, OX2, and OXK; <1:80 for salmonella B, C, and E; and <1:160 for salmonella D. Antibody titers to *Rickettsia rickettsii* were elevated at 1:32 (normal = <1:16).

As a result of the excellent clinical response to tetracycline (resolution of left upper quadrant pain and headache) and the high OX19 titer, it was felt that this woman had Rocky Mountain spotted fever with a very unusual presentation of crushing substernal chest pain and ECG changes suggestive of ischemia. One week after treatment the patient remained free of chest pain, and an ECG showed resolution of the ST segment changes. There was never any tick found on her body, nor could she recall any history of a tick bite. Fourteen days prior to admission, however, she had been in a wooded area that was endemic for Rocky Mountain spotted fever.

DISCUSSION

From 1980 to 1984 Rocky Mountain spotted fever caused about 40 deaths annually in the United States.² The overall case mortality in 1986 was 3% with a provisional total of 755 cases.³

The case presented can be classified by Centers for Disease Control criteria as probable Rocky Mountain spotted fever because of the single $1:\geq 320$ OX19 titer in a clinically compatible case.³

Typically, patients with Rocky Mountain spotted fever feel better in 36 to 48 hours after treatment with tetracycline or chloramphenicol; headache and other symptoms abate. By the 3rd day of treatment, the temperature usually returns to normal, as it did in this case.²

It is interesting to note that the unusual presentation of

crushing substernal chest pain with ECG changes subsequently resolved following treatment with tetracycline. Ischemic injury has been described previously in multiple organ systems in relationship to the widespread vasculitis produced by R rickettsii. The pathologic concomitants of Rocky Mountain spotted fever involve a generalized vasculitis resulting from invasion of rickettsiae into endothelial and smooth muscle cells. This vasculitis is shown to be responsible for the cardiopulmonary and skin findings.⁴ Cardiac manifestations that have been reported previously in the literature include myocarditis, myocardial necrosis, abnormal left ventricular function, and ECG abnormalities including sinus and nodal tachycardias, first-degree block, and ST-T wave changes.5-7 Ischemic injury has also been shown to cause ischemic necrosis of the appendix as well as gangrene of the gallbladder.8

In 1986, 62% of the cases of Rocky Mountain spotted fever were associated with tick bites; 94% had fever, 89% had headache, 87% had myalgia, and 87% had rash. When the rash was present, it was located on the palms or soles 53% of the time.³ The authors are unaware of any cases presenting with chest pain.

Studies of Rocky Mountain spotted fever by Helmick et al¹ showed abdominal pain as the initial presentation (first 3 days) 30% of the time, with only 16% of patients experiencing splenomegaly at some time during the course of their illness. Only 1% experienced arrhythmias during the first 3 days.

When Rocky Mountain spotted fever presents in an atypical fashion, delay in diagnosis can mean delay in appropriate treatment. Awareness of the clinical spectrum of Rocky Mountain spotted fever and early treatment with tetracycline or chloramphenicol before the diagnostic criteria can be met may significantly reduce mortality from this disease. This disease should be seriously considered when someone who resides in or has visited an area known to be endemic for Rocky Mountain spotted fever suffers from headache, fever, prostration, or myalgia during the spring or summer months. The rash usually occurs 3 to 4 days after a tick bite, but may be delayed as long as 2 weeks. Of interest in this case report is the possibility that chest pain with ST segment and T wave inversion ischemic changes were the first sign of vasculitis caused by the rickettsiae. It would appear that in this case Rocky Mountain spotted fever, the great mimicker, presented as ischemic heart disease.

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