Acute Q fever: A 2- year experience at a Tertiary-Care Center

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INTRODUCTION

- The clinical diagnosis of acute Q fever can be challenging.
- This is due in part to the non-specific clinical presentation, negative Q fever serologies when obtained early in the illness course, in addition to the low index of suspicion
- Identifying and treating patients with acute Q fever who are at increased risk for progression to persistent focalized infection is crucial.

OBJECTIVES

Our goal is to describe the demographics, clinical presentation, laboratory and radiographic findings in addition to treatment of patients diagnosed with acute Q fever at our institution over the past 2 years

METHODS

We performed a retrospective chart review and identified all patients diagnosed with acute fever between December 2019 and June 2022 at our institution.

Patients who were 18 years or older who had a Q fever anti-phase II IgM ≥ 1:50 and anti-phase II IgG ≥ 200 were included. Patient who had an initial anti-phase II IgG < 200 were included if they had a documented fourfold rise in anti-phase II IgG levels at follow-up within 3-6 weeks [1]

RESULTS

Table 1.Clinical characteristics of patients diagnosed with acute Q fever

Patient demographics	**Number (n = 15)**
Gender (male)	12 (80%)
Age in years, median (range)	51 (32-68)
Documented exposure history	13 (87%)
Risk factors for future complications	
Elevated antiphospholipid (APL) IgG Ab titers *	6 / 8 (75%)
Baseline TTE at time of diagnosis ^	11 (73%)
Valvulopathy / cardiac aneurysms	5 (33%)
Endovascular graft	2 (13%)
Prosthetic joints	0
Immunosuppression	2 (13%)
Patients with risks for future complications	10 (67%)
Clinical findings	
Non-specific febrile illness	13 (87%)
Shortness of breath	3 (20%)
Abdominal pain	4 (27%)
Headaches	3 (20%)
Rash	1 (7%)
Duration of symptoms prior to diagnosis in days, median (Q1,Q3)	20 (13,90)
Radiographic findings	
Multifocal pulmonary infiltrates	2 / 14 (14%)
Nonspecific pulmonary nodules	2 / 14 (14%)
Inflammatory changes surrounding an endovascular graft	1 / 2 (50%)
Splenomegaly	3 / 14 (21%)
Splenic infarcts	2 / 14 (14%)
Acute pancreatitis	1 / 14 (11%)
Lymphadenopathy	2 / 14 (14%)
Antibiotic therapy	
Doxycycline monotherapy	7
Duration in days, median (range)	15 (11-30)
Doxycycline + hydroxychloroquine	8
Duration in months, median (range)	15 (12-28)

* Defined as > 15 IgG Phospholipid units (GPL)
^ TTE: Transthoracic echocardiogram

RESULTS

Laboratory findings at the time of diagnosis

Elevated C-reactive protein #	11 (73%)
Elevated alanine and/or aspartate transaminases ^	9 (60%)
Elevated alkaline phosphatase ¶	4 (26%)
Elevated bilirubin levels Δ	2 (13%)
Thrombocytopenia α	2 (13%)
Leukopenia β	2 (13%)
Positive *Coxiella burnetii* PCR in blood	0 / 9 (0%)
Phase II IgG on presentation, median (range)	1:2024 (1:16 – 1:8192)
Phase II IgM on presentation, median (range)	1:1024 (1:16 – 1:2048)
Phase I IgG on presentation, median (range)	1:16 (1:16 – 1:1024)

Defined as > 3.0 mg/dL
^ Defined as alanine aminotransferase > 55U/L and aspartate aminotransferase > 48 U/L
¶ Defined as alkaline phosphatase > 129 U/L
Δ Defined as total bilirubin > 1.2 mg/dL
α Defined a platelet count < 135 x 10(9)/L
β Defined as total white blood cell count < 3.4-9.6 x 10^9/L

RESULTS

- Three patients had detectable Coxiella DNA in the blood during the acute phase using cell free DNA next generation sequencing for pathogen detection (Karius)
- Ten patients had underlying risk factors that would place them at risk for future complications
- Eleven patients had follow-up appointments ranging between 2 weeks to 2 years
  - One patient had lymphadenitis with a protracted disease course and another one developed infective endocarditis

CONCLUSIONS

We noted an increased number of cases of acute Q fever diagnosed during this study period compared to a prior study[2]. This could be related to a combination of increased awareness and increasing incidence of the infection

Acute Q fever was mainly diagnosed in hospitalized patients in this series. Milder cases are potentially missed and likely to be diagnosed later if they develop focal persistent disease

More patients in our cohort had baseline TTE (73%) and APL IgG levels (53%) compared to a prior study[2]. This reflects a shift in our approach to these cases, which is geared towards risk stratification and early preventive strategies

Patients with risk factors for progression into persistent disease were often placed on a longer course of a combination therapy in attempt to prevent chronic focal infections

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