

Case and Review

Peritoneal *Chlamydia trachomatis* Infection as a Cause of Ascites: A Diagnosis Not to Be Missed

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Keywords

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Abstract

Ascites is a common complication of several conditions, but it is rare in cases of *Chlamydia trachomatis* infection. We report a 36-year-old patient presenting with abdominal swelling for a week prior to hospitalization. An extensive workup excluded liver or heart disease and malignancy. A computed tomography scan demonstrated massive ascites and severe thickening of peritoneal reflections. Laboratory tests showed low serum-ascites albumin gradient, high total protein, and low adenosine. Diagnostic laparoscopy revealed inflammatory signs of both fallopian tubes. The histopathological results from peritoneal biopsy were consistent with lymphoid proliferation with reactive lymphoplasmacytic infiltrate. A gynecological investigation showed a positive DNA for *C. trachomatis* in the cervical swab. After treatment with doxycycline, there was a complete resolution of ascites.

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Introduction

Cirrhosis, heart failure, nephrotic syndrome, and peritoneal disease are the most common causes of ascites. The combination of serum-ascites albumin gradient (SAAG) and ascites total protein content has been used to help in the differential diagnosis of the etiology of ascites.

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SAAG >1.1 g/dL suggests liver or heart disease, whereas values <1.1 g/dL other causes of ascites. A high protein level of >2.5 mg/dL in ascitic fluid is usually found in heart failure or peritoneal disease [1].

Ascites associated with low SAAG values comprises a heterogeneous group of conditions that include pancreatic and renal disease, peritoneal malignancy, and infections such as tuberculosis or permeability changes (i.e., hypothyroidism and paraproteinemias) [2]. *Chlamydia* peritoneal infection is a rare cause of ascites with low SAAG in young people [3].

Chlamydia trachomatis is responsible for the most common sexually transmitted infectious disease in the world. There has been an increasing incidence and awareness over the last decades, likely due to the precise diagnosis with nucleic acid amplification testing. Although most of the patients remain asymptomatic, if left untreated, *Chlamydia* may cause chronic pelvic pain, infertility, and pelvic inflammatory disease (PID) [4]. The Centers for Disease Control and Prevention (CDC) current guidelines recommend routine screening in all sexually active women under 25 years to prevent complications [5].

However, due to the rarity of *Chlamydia*-induced ascites, there is no formal recommendation for performing routine testing in the workup diagnostic algorithms of ascites. Here, we report a case of a young woman presenting with ascites secondary to *Chlamydia* infection.

Case Report

A 36-year-old Caucasian female presented at the emergency department with a history of progressive abdominal distension and weight gain of 8 kg for the past 2 months, increasing from 60 to 68 kg. She also reported fatigue and reduced appetite for 1 week prior to hospitalization. A review of systems was negative for fever, night sweats, chills, jaundice, dark urine, vomiting, or diarrhea.

Her past medical history was remarkable for recurrent bacterial and yeast vulvovaginitis, but was asymptomatic over the last year. She denied known liver disease, alcohol abuse, or smoking. Her family history was uneventful.

Physical exam revealed normal vital signs with a blood pressure of 100/60 mm Hg and a heart rate of 90 beats per minute, and she was afebrile. She presented with a pale appearing, without jaundice. Moderate abdominal distension, with marked ascites, diffuse tenderness, and palpable liver edge 3 cm below the costal margin were the major findings. There were no peripheral stigmata of chronic liver disease or congestive heart failure.

Laboratory tests demonstrated iron deficiency anemia with a hemoglobin level of 9.5 mg/dL and a mean corpuscular volume of 75 fL. She had a normal white blood count of 6,050 mL/mm³ and a platelet count of 302 mL/mm³. C-reactive protein was 29 mg/dL and serum creatinine 0.74 mg/dL. She had normal liver enzymes: alanine transaminase 11 U/L, aspartate transaminase 22 U/L, alkaline phosphatase 51 U/L, and gamma-glutamyl transferase 9 U/L. Liver function tests showed a total bilirubin level of 0.2 mg/dL, serum albumin 3.6 g/dL, and an international normalized ratio 1.17. CA-125 was markedly elevated to 452 U/mL. An extensive workup diagnosis excluded hepatotropic virus infections, such as hepatitis A, B, and C, Epstein-Barr, and cytomegalovirus. Blood testing was seronegative for antinuclear, anti-smooth muscle, and antimitochondrial antibodies. Thyroid function was normal, and the B-hCG test was negative.

The abdominal computed tomography was remarkable for massive ascites, severe thickening, and enhancement of peritoneal reflections, stranding, and thickening of the omentum (Fig. 1). The liver parenchymal attenuation was preserved, while the main portal and hepatic veins were patent. An upper endoscopy ruled out esophageal varices.



Fig. 1. Coronal contrast-enhanced CT scan of the abdomen showed severe ascites, peritoneal thickening, and enhancement of peritoneal reflections and the omentum (yellow arrow).

A diagnostic paracentesis was performed, which showed a citrine-yellow fluid with an SAAG of 0.8 and a total protein of 2.8 g/dL, suggesting peritoneal disease. The total cytological count was 6,200 leukocytes/mL, 43% of neutrophils, 28% of lymphocytes, and 23% of monocytes. Adenosine deaminase of the fluid, polymerase chain reaction for tuberculosis, and oncotic cytology were all negative.

The patient was treated with intravenous antibiotics for spontaneous bacterial peritonitis without clinical improvement. She subsequently underwent a diagnostic laparoscopy that revealed macroscopically clear fluid and inflammatory signs of the tubes, suggesting PID. There were no neoplastic features, and the etiology of the ascites remained undetermined (Fig. 2).

Histopathological results demonstrated fibrous tissue on multiple peritoneal and pelvic nodules, and the omental biopsy showed lymphoid proliferation (Fig. 3). The immunohistochemical staining demonstrated a reactive lymphoplasmacytic infiltrate.

A gynecological investigation with a cervical swab showed a positive DNA for *C. trachomatis*. The combination of the findings in diagnostic laparoscopy, peritoneal biopsy, and ascitic fluid analysis led to the diagnosis of PID. The patient underwent treatment with oral doxycycline for 2 weeks, with complete resolution of all clinical manifestations. No recurrence of ascites was observed in the 3-month follow-up.

Discussion

We report the case of a young woman with recent-onset ascites without clear etiology. The ascitic fluid's workup showed a low SAAG and a high total protein, suggesting peritoneal disease. After an extensive investigation and exclusion of common causes of ascites, the detection of *C. trachomatis* DNA in the cervical swab combined with laparoscopy findings confirmed the diagnosis of PID. Treatment with doxycycline resulted in complete resolution of ascites.

PID is an inflammatory disorder of the upper genital tract in women due to infection. Many organisms might cause this condition, but *Neisseria gonorrhoeae* and *C. trachomatis* are

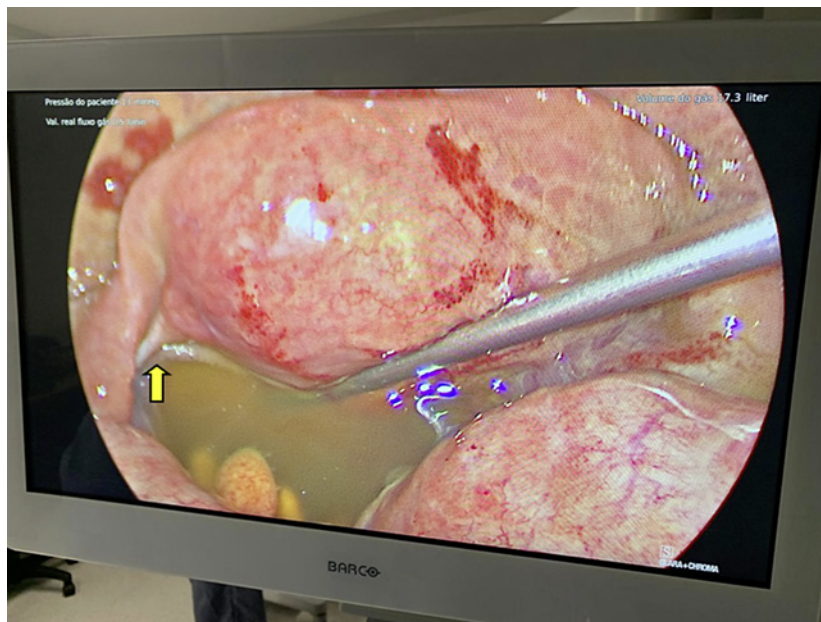


Fig. 2. Inflammatory signs of the fallopian tubes and ascites in laparoscopy (yellow arrow).

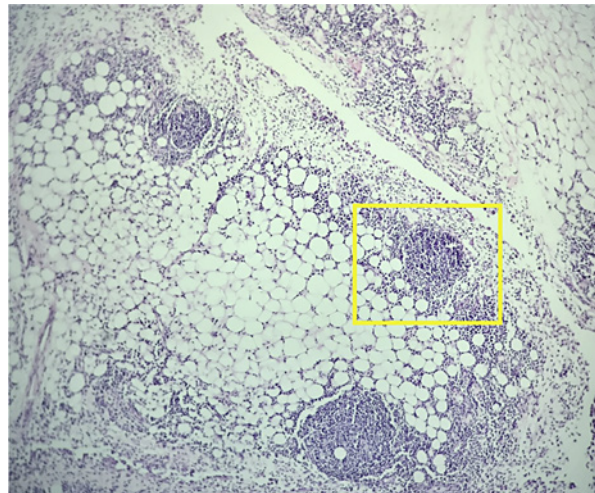


Fig. 3. Reactive lymphocytic aggregates and lymphoid follicles in the subperitoneal fat and connective tissue specimen (yellow box). Hematoxylin and eosin staining. $\times 4$.

the main pathogens. PID commonly presents as endometritis, salpingitis, tubo-ovarian abscess, and pelvic peritonitis. Ascites is considered a rare manifestation of the disease. The diagnosis can be challenging in clinical practice given the wide range of symptoms [6].

Since the first description of ascites caused by *C. trachomatis* in 1978, few cases were reported, but all occurred in young sexually active women, otherwise healthy. The largest series, by Muller-Schoop et al. [7], described 11 patients with abdominal pain and signs of peritonitis. Nine had strong serological evidence of acute infection by *C. trachomatis*, and 6 of them had ascites with negative results of ascitic fluid cultures [7].

In *Chlamydia* peritoneal infection, the ascitic fluid analysis shows a pattern of low SAAG, high protein levels, and lymphocytic predominance, mimicking other more common peritoneal diseases [8, 9]. Although high adenosine deaminase levels are often found in peritoneal tuberculosis, increased values have also been reported in *Chlamydia* infection [10].

Chlamydia ascites is a diagnosis not to be missed because of the long-term sequelae and disability associated with the infection. Although antibody titers may be measured in the ascitic fluid, the diagnosis more commonly relies on nucleic acid amplification testing on cervical mucus, urine, or serum, yielding higher accuracy rates for the former [6].

This report has described a rare manifestation of infection by *C. trachomatis* in a sexually active young woman. Clinicians should be aware of this condition when no clear cause for ascites is identified in the initial workup, particularly if the fluid analysis exhibits low SAAG and lymphocytic predominance.

Statement of Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. Ethical approval was not required for this study in accordance with local and national guidelines.

Conflict of Interest Statement

The authors of this manuscript do not have any conflicts of interest to declare.

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Author Contributions

All authors equally contributed to this study's conception, study and design, literature review and analysis, drafting, critical revision and editing, and final approval of the final version.

Data Availability Statement

The data that support the findings of this study cannot be shared due to the General Data Protection Regulation published by Brazilian laws and authorities. Queries regarding the data in this article should be addressed to B.L.L. (luisa.barros@hc.fm.usp.br).

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