CLINICAL RESEARCH

Diffuse peritonitis and chronic ascites due to infection with Chlamydia trachomatis in patients without liver disease: new presentation of the Fitz-Hugh–Curtis syndrome

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Abstract

Two women were admitted for increasing abdominal pain, vaginal discharge, and severe or moderate chronic ascites. Diffuse peritonitis without evidence of liver disease was found in both cases, and in one the ascites and vaginal discharge contained Chlamydia trachomatis. Both patients responded to doxycycline, and this and the laboratory findings pointed strongly to C trachomatis as the aetiological agent. C trachomatis may cause severe peritoneal infections with chronic ascites formation in the absence of liver disease in women with the Fitz-Hugh–Curtis syndrome. Prompt diagnosis and antibiotics lead to rapid cure.

Introduction

Chronic ascites in patients without concomitant liver disease has apparently not been reported in the Fitz-Hugh–Curtis syndrome.^{1,2} We therefore describe two patients with the syndrome who presented with diffuse peritonitis and ascites due to infection with Chlamydia trachomatis.

Case 1

A 28 year old woman was admitted with a history of increasing abdominal pain and ascites for five months after noticing a whitish vaginal discharge associated with using an intrauterine device.

Examination showed the patient to be in poor general condition with severe ascites. There were no gynaecological abnormalities and liver enzyme activities and prothrombin time were normal. The table gives the laboratory findings. Tuberculosis was excluded. Ascitic fluid contained 76 g protein per litre, few leucocytes, and no bacteria on culture. Laparoscopy showed multiple fibrous adhesions of the whole peritoneal cavity including the liver surface with fibrous plaques on both adnexa and large ascites. Biopsy of the peritoneum yielded fibrous tissue without inflammation. Biopsy of the liver showed non-specific reactive hepatitis. The skin was anergic to purified protein derivative of tuberculin and to seven common intracutaneous antigens (Multitest, Mérieux). Nothing abnormal was found on radiography and biopsy of the small intestine, computed tomography of the abdomen, smear and biopsy of the bone marrow, or bacterial and parasitological studies of the stool. Cultures of cervical and ascitic fluid grew no pathogens. Cultures for C trachomatis were taken only after starting treatment and also gave negative results.

Case 1. Chronological sequence of antibody titres against trachomatis and pathological laboratory data

	1984		1985	
	16 March	15 June	1 April	20 November
Complement fixation test	1 240	1 120	1 120	1 60
Immunofluorescent IgG	1 2560	1 2560	1 2560	1 2560
Immunofluorescent IgM	1 320	1 80	1 80	1 80
Immunofluorescent IgA	1 1280	1 1280	1 640	1 640
Erythrocyte sedimentation rate (mm in first hour)	108	15	13	4
Platelet count ($\times 10^9/l$)	927	478	377	417
Serum albumin (g/l)	26	42	44	43

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The intrauterine device was removed and the patient treated with doxycycline 200 mg daily for six weeks. She recovered rapidly, the pain and ascites disappearing completely. Four months later, two weeks after insertion of a new intrauterine device, she had a recurrence of abdominal pain with formation of ascites. Results of bacterial investigations were again negative. She responded well to a second course of doxycycline 200 mg daily for four weeks, followed by 100 mg daily for five months, and more than one year later remained symptom free. Helper/suppressor T cell ratio and lymphocyte responses to different plant mitogens were normal. No antibodies to human T cell lymphotropic virus type III were detected at follow up but her skin reactivity remained anergic.

Case 2

A 26 year old waitress with chronic alcohol abuse was admitted because of increasing, predominantly left sided abdominal pain for three weeks, formation of ascites, and yellow vaginal discharge. Laboratory findings were normal except for a sedimentation rate of 44 mm in the first hour and platelet count of $511 \times 10^9/l$. Laparoscopy showed severe diffuse fibrinous peritonitis, most pronounced in the right upper quadrant, and a moderate amount of ascites. *C trachomatis* was cultured from the ascites and vaginal discharge. The patient was treated successfully with doxycycline 200 mg daily for 10 days.

Comment

Shabot and colleagues found *C trachomatis* in ascitic fluid of patients with chronic liver disease but were unable to say whether the organism was pathogenic in those cases.³ Diffuse peritonitis and ascites formation in our patients without chronic liver disease were definitely due to an infectious agent, as shown by the rapid response to antibiotic treatment. Preceding genital infection and manipulation of the uterus are classic features of the Fitz-Hugh-Curtis syndrome.⁴ *C trachomatis* was the most likely aetiological agent in both patients.

In case 2 the organism was cultured from the cervical secretion and ascitic fluid and in case 1 the patient showed a fourfold decline of immunofluorescent IgM antibodies after treatment.⁵

These two patients show that *C trachomatis* may cause severe chronic peritoneal infections with chronic ascites formation. Timely diagnosis and correct treatment lead to rapid cure.

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Cigarette smoking and risk of premature stroke in men and women

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Abstract

A case-control study was carried out of the relation between cigarette smoking and hypertension and stroke. A total of 132 cases of stroke (79 in men, 53 in women) identified as a part of a population based register were compared with 1586 controls (1017 men, 569 women) from a survey of cardiovascular risk factors conducted in the same population. Cigarette smokers had a threefold increase in the risk of stroke compared with current non-smokers. This association remained significant after adjusting for hypertension. Those who both smoked and had hypertension had an increased risk of stroke of almost 20-fold compared with those who neither smoked nor had hypertension.

Overall, in this population roughly 37% of stroke events may be attributed to cigarette smoking and 36% to hypertension.

Introduction

High blood pressure is consistently reported as the most important known risk factor for stroke.¹⁻⁴ Other risk factors such as cigarette smoking or serum lipid, blood glucose, and plasma fibrinogen concentrations have shown varying associations.^{3,4} In particular,

many studies have failed to identify cigarette smoking as a significant risk factor for stroke. When a positive association has been found this has usually been confined to young men.^{5,6}

We present a population based case-control analysis of the association of cigarette smoking and hypertension with stroke in both men and women. The proportions of stroke events that could be attributed to smoking and hypertension are also estimated as this does not appear to have been done before. In addition, the study investigates the possible synergism between smoking and hypertension.

Subjects and methods

Cases of stroke were identified from a population based cardiovascular disease register, in which a 50% random sample of all new episodes of stroke in Auckland was studied in the year ending 1 March 1982. The study area (population 829 545) encompassed one quarter of the total population of New Zealand. Full details of the case finding methods have been published.⁷ The diagnostic criteria and methods were based on the World Health Organisation guidelines.⁸ Stroke was defined as the rapid onset of focal neurological deficit, presumably of vascular origin, lasting 24 hours or longer or leading to death. Patients with subarachnoid haemorrhage were excluded.

Information was obtained from patients using a standard questionnaire as soon after the event as possible. If the patient had died the same questionnaire was administered to a close relative after six weeks. Details about treatment for high blood pressure and cigarette smoking were collected. Current pharmacological treatment for high blood pressure was used as a surrogate measure of hypertension. A current smoker was defined as a person smoking at least one cigarette a day.

Controls for the analysis came from a population based survey in Auckland, the sampling methods and results of which have been reported.⁷ Briefly, 1017 men and 569 women aged 35-64 were interviewed between January and July 1982 after being randomly selected from the 1981 Auckland general electoral rolls (response rate 82%). The questionnaire included the same questions about blood pressure history and cigarette smoking as were asked of the patients with stroke.

Analyses were restricted to events that occurred in men and women aged

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