CASE REPORT

Acute gross painless transudative ascites in a patient with lupus

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SUMMARY

Gross ascites is a rare presentation of lupus. Ascites in lupus may be due to lupus peritonitis or secondary to one of the complications including nephrotic syndrome. The ascites due to lupus peritonitis has been described as exudative with a serum-ascites albumin gradient (SAAG) below 11 g/L, unless associated with nephrotic syndrome. We report an unusual case of lupus ascites in a 23-year-old woman who presented with acute painless gross ascites with no constitutional, skin or musculoskeletal symptoms of a lupus flare. The ascites was a transudate with SAAG above 11 g/L with no associated nephrotic syndrome. She was treated with corticosteroids, mycophenolate mofetil and diuretics with a good response and no recurrence of her ascites.

BACKGROUND

Gross ascites as a manifestation of lupus is rare.^{1–3} It may present acutely or chronically with or without pain.² Ascites in lupus may be due to lupus peritonitis or secondary to one of the complications including nephrotic syndrome, protein-losing enteropathy, constrictive pericarditis or Budd-Chiari syndrome.² Acute lupus ascites secondary to lupus peritonitis has been described as exudative with a serum-ascites albumin gradient (SAAG) below 11 g/L,^{4–8} unless associated with nephrotic syndrome.⁹ It is also usually associated with abdominal pain, and other symptoms of a lupus flare.⁴ We report an unusual case of gross ascites in a patient with lupus.

CASE PRESENTATION

A 23-year-old Caucasian woman was diagnosed with systemic lupus erythematosus when she initially presented with malar rash, arthralgia, alopecia and the right leg vasculitic ulcer. One year later she presented to our hospital with a 2-week history of abdominal distention and dyspnoea. She had no associated fever, skin rash or arthritis. There was no abdominal pain or diarrhoea. Her medications included hydroxychloroquine. Her history included one miscarriage. She was a non-smoker and non-alcohol drinker. On examination, she was morbidly obese with a body mass index of 45 and weight of 144 kg. Her vital signs showed blood pressure of 153/107, pulse rate of 100/min, respiratory rate of 18/min and oxygen saturation of 98% on room air and she was afebrile. There was no skin rash and there was no evidence of arthritis. Cardiovascular and respiratory examinations were unremarkable. On abdominal examination, there was gross abdominal distention with positive shifting dullness and no tenderness. There was mild pitting oedema

in the lower limbs with a small vasculitic ulcer on the right foot.

INVESTIGATIONS

Investigations included a full blood count, which showed hypochromic microcytic anaemia with haemoglobin of 103 g/L (115–165 g/L), mean corpuscular haemoglobin of 25 pg (27–34 pg) and mean corpuscular volume of 77 fL (79–99 fL). Iron deficiency was confirmed on iron studies. There was acute renal impairment with creatinine of 201 µmol/L (30–100 µmol/L), estimated glomerular filtration rate (eGFR) of 27 mL/min/1.73 m² and urea of 15.1 mmol/L (2.5–6.7 mmol/L). Her previous creatinine levels were between 85 and 95 µmol/L. Liver function tests showed albumin of 30 g/L (38–50 g/L), aspartate aminotransferase of 53 U/L (<30 U/L), alanine aminotransferase of 49 U/L (<30 U/L) and γ-glutamyl transferase of 49 U/L (<30 U/L) consistent with mild hypoalbuminaemia and mild liver dysfunction. Coagulation studies were normal. The blood glucose was 4.5 mmol/L (3.4–7.7 mmol/L), and her cholesterol was 4.8 mmol/L (3.5–5.4 mmol/L), which were within normal range. Erythrocyte sedimentation and C reactive protein (CRP) were elevated to 58 mm/h (<30 mm/h) and 17.9 mg/L (<6.0 mg/L), respectively.

Her 24 h urine showed proteinuria of 1.03 g/day (0.0–0.14 g/day) with an albumin/creatinine ratio of 18.4 mg/mol (<3.5 mg/mol). Urine analysis showed microscopic haematuria and proteinuria with hyaline casts. Abdominal CT scan showed gross ascites, mild right pleural effusion, normal renal size, no organomegaly or lymphadenopathy (figure 1). The patient underwent paracentesis with a total of 14 L drained. The ascitic fluid was clear with no leucocytes and fluid culture showed no growth. There were no malignant cells seen. The fluid biochemistry showed reduced albumin (16 g/L) and reduced protein (<20 g/L), glucose of 5.3 mmol/L, lactate dehydrogenase of 153 U/L and amylase of 20 U/L. The SAAG was 14 g/L (>11 g/L), consistent with transudate fluid. Hepatitis screening tests including hepatitis A, B, C, HIV, smooth-muscle antibody, anti-mitochondrial antibody were negative.

Abdominal ultrasound showed patent hepatic veins with no evidence of portal hypertension or cirrhosis. No ovarian pathology was seen in pelvic ultrasound. Faecal α1 antitrypsin was checked for protein-losing enteropathy, which was negative. The echocardiogram was normal. Further investigation showed antinuclear antibody 1:640 with homogeneous pattern, anti-ds-DNA >80 IU/mL (<7.5 IU/mL), low complements with C3 of

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To cite: Erfani T, Manolios N, Howe G, *et al.* *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-201503

Figure 1 Abdominal CT scan showing gross ascites.

0.44 g/L (0.78–1.82 g/L) and C4 of 0.01 g/L (0.13–0.52 g/L). The extractable nuclear antigens was positive for anti-Sm and antihistones. Lupus anticoagulant was not detected and β 2 glycoprotein IgG was normal. Cardiolipin IgG and IgM were mildly elevated, 23 Gpl (<5 Gpl) and 15 Mpl (<5 Mpl), respectively. Renal biopsy was unsuccessful due to body habitus.

TREATMENT

The patient was diagnosed with active lupus and lupus nephritis with nephritic range proteinuria. She was treated by pulse intravenous methylprednisolone 1 g daily for 3 days and then oral prednisone 60 mg daily, mycophenolate 1 g twice a day, and furosemide 80 mg daily.

OUTCOME AND FOLLOW-UP

One month later her weight was decreased by 15 kgs and her symptoms were improved. The renal function, liver function and CRP remained normal and the serum complements and anti-DNA were returned to normal. Six months later successful renal biopsy was performed while she was on the treatment, which showed diffuse proliferative lupus nephritis. On one-year follow-up visit, she was well with no reoccurrence of ascites.

DISCUSSION

To the best of our knowledge this is the first reported case of gross ascites, which was a transudate, secondary to systemic lupus erythematosus without associated nephrotic syndrome. All the possible causes of ascites, including cardiac failure, constrictive pericarditis, Budd-Chiari syndrome, liver cirrhosis, were

excluded in our patient. This case was unique as the massive ascites was transudate by being associated with nephritic but not nephrotic syndrome. It was unlikely that the degree of proteinuria (nephritic range) and mild hypoalbuminaemia alone would have explained the massive ascites. It was more likely that the ascites was due to active systemic lupus erythematosus and responded to the treatment of it with no recurrence.

The mechanism of peritonitis in lupus is not well understood. The histopathology results from biopsy or autopsy reveal a multifactorial process including inflammatory mechanism with infiltration of lymphocytes and macrophages, deposition of immune complexes in peritoneum and vasculitis of peritoneal small vessels.^{2 5}

Learning points

- ▶ Ascites can be a manifestation of systemic lupus erythematosus.
- ▶ Gross ascites in lupus is rare.
- ▶ When detected, peritonitis and/or perforation should be excluded by paracentesis.
- ▶ If a transudate, a trial of glucocorticoids $\pm$ immunosuppressive, diuretics should be considered.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Man BL, Mok CC. Serositis related to systemic lupus erythematosus prevalence and outcome. *Lupus* 2005;14:822–6.
- 2 Ebert EC, Hagspiel KD. Gastrointestinal and hepatic manifestations of systemic lupus erythematosus. *J Clin Gastroenterol* 2011;45:436–41.
- 3 Weinstein PJ, Noyer CM. Rapid onset of massive ascites as the initial presentation of systemic lupus erythematosus. *Am J Gastroenterol* 2000;95:302–3.
- 4 Forouhar-Graff H, Dennis-Yawingu KA, Parke AL. Insidious onset of massive painless ascites as initial manifestation of systemic lupus erythematosus. *Lupus* 2011;20:754–7.
- 5 Junior HP, Neto AA, Teixeira MAB, *et al*. Ascites due to lupus peritonitis: a rare form of onset of systemic lupus erythematosus. *Rev Bras Reumatol* 2012;52:113–19.
- 6 Prasad S, Abujam B, Lawrence A, *et al*. Massive Ascites as a presenting feature of lupus. *Int J Rheu Dis* 2012;15:15–16.
- 7 Schousboe JT, Koch AE, Chang RW. Chronic lupus peritonitis with ascites: review of the literature with a case report. *Semin Arthritis Rheum* 1988;18:121–6.
- 8 Mier A, Weir W. Ascites in systemic lupus erythematosus. *Ann Rheum Dis* 1985;44:778–9.
- 9 Fuke Y, Ikeda K, Shimizu C, *et al*. Longstanding intractable massive ascites caused by chronic lupus peritonitis with nephrotic syndrome. *Japan J Nephrol* 2009;51:1067–74.

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