

# Post-*Streptococcus mitis* infection polyserositis

Paula Grech,<sup>1</sup> Jessica Mangion,<sup>2</sup> Sandro Vella<sup>3</sup>

<sup>1</sup>General Medicine, Mater Dei Hospital, Msida, Malta

<sup>2</sup>Diabetes and Endocrinology, Mater Dei Hospital, Msida, Malta

<sup>3</sup>Medicine, Mater Dei Hospital, Msida, Malta

## Correspondence to

Dr Paula Grech;  
paulagrech93@gmail.com

Accepted 31 December 2020

## SUMMARY

We report a case of a 42-year-old man who presented with acute epigastric and retrosternal chest pain and exertional dyspnoea, and was subsequently diagnosed with polyserositis secondary to post-*Streptococcal mitis* infection. A CT scan showed a large pericardial effusion requiring pericardiocentesis, small bilateral pleural effusions and small amount of ascites. Several serological tests were done, which were all found to be normal. Pericardial and pleural fluid aspirates revealed an exudate. Culture of the pleural fluid yielded growth of *S. mitis* and this was deemed the cause of the polyserositis, which is rare. The patient made a spontaneous recovery. He was started on colchicine by the cardiologists to help prevent pericardial fluid recurrence and this was continued for 3 months. A dental review confirmed the presence of dental caries, the possible source of infection. On follow-up, the patient remained well with no further relapses.

## BACKGROUND

Polyserositis is defined as a general inflammation of the serous membranes causing serous effusions in different body cavities. There are many causes of polyserositis, including connective tissue diseases, malignancy, iatrogenic, metabolic and infective. In this case report, all serological tests were normal and malignancy was excluded. Culture of the pleural fluid revealed growth of *Streptococcus mitis* (*S. mitis*). To the authors' knowledge, polyserositis secondary to *S. mitis* has never been documented yet.

## CASE PRESENTATION

A 42-year-old healthy male Maltese with a history of depression presented to the emergency department with a 4-hour history of epigastric and retrosternal chest pain which radiated to the right iliac fossa. The pain was more marked after food intake and woke him up at night. He also complained of recent exertional shortness of breath, limiting his ability to walk on flat ground but he denied any orthopnoea or paroxysmal nocturnal dyspnoea. At times, he felt palpitations but these were self-limiting. He denied any other systemic symptoms. He was not taking any regular medications. There was no relevant family history of connective tissue disease or malignancy. He was an ex-smoker with 20 pack-year history, and worked as a police officer with regular contact with irregular immigrants. There was no recent travel history and had birds as pets.

He was afebrile and his vital signs were normal. Cardiorespiratory examination revealed muffled heart sounds and reduced air entry at both lung bases. The patient's abdomen was soft, although

tender in the epigastrium and right iliac fossa. An erythematous macular rash was noted on the trunk. An ECG showed normal sinus rhythm at 77 beats/min with small QRS complexes.

## INVESTIGATIONS

His initial laboratory values showed a C reactive protein (CRP) of 73 mg/L in the setting of a normal white cell count and a normal erythrocyte sedimentation rate (ESR). His blood film showed mature leucocytes, poikilocytosis and platelet anisocytosis but no atypia. His renal function and thyroid function tests were normal but he had mildly deranged liver function tests with an alanine transaminase of 61 U/L and a gamma-glutamyl transferase of 76 U/L. Amylase was normal. Two troponins sampled 3 hours apart were low, excluding a myocardial infarction. NT-proB-type natriuretic peptide was also normal, ruling out heart failure. The albumin level was >40 g/L; therefore, polyserositis was not due to hypoalbuminaemia.

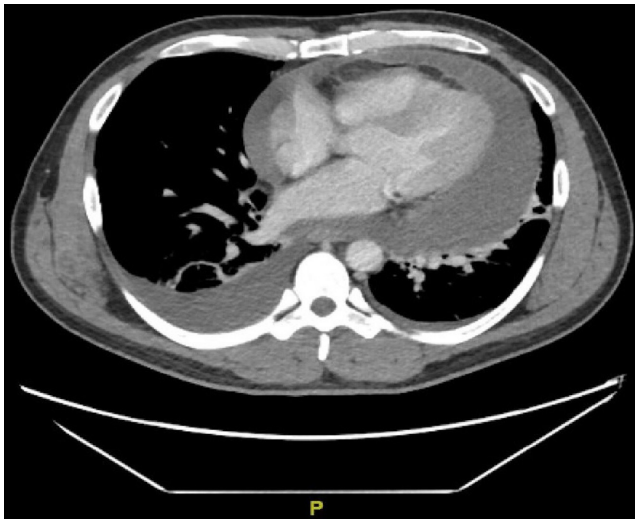
A chest X-ray showed bilateral pleural effusions, larger on the right, with an increased cardiothoracic ratio. In view of the patients' symptoms, signs and the chest X-ray findings, a CT scan of the thorax, abdomen and pelvis was requested, revealing bilateral pleural effusions, more prominent on the right, and a large pericardial effusion measuring up to 50 mm, as shown in figure 1. Additionally, there were radiological signs of tricuspid valve insufficiency and liver congestion. The liver was mildly enlarged but had normal texture and no focal lesions. There were few mildly enlarged retroperitoneal lymph nodes along the aorta and a small amount of ascites in both flanks of the abdomen and in the pelvis. The bowel, gall bladder, pancreas, spleen and kidneys were normal. There were no signs of underlying malignancy.

Echocardiogram confirmed a large pericardial effusion, with preservation of ejection fraction. The right atrium was collapsed but the right ventricular function was preserved. The inferior vena cava (IVC) was dilated at 2 cm with decreased collapse and mild tricuspid regurgitation. Pericardiocentesis was performed and 1150 mL of serosanguinous fluid was drained. The fluid was sent for cytology and there were no malignant cells present, microscopy revealed numerous polymorphs and red blood cells, while culture had no bacterial growth. Biochemistry showed a transudative effusion according to Light's criteria. Colchicine was initiated at 0.5 mg two times per day as an anti-inflammatory to help reduce fluid accumulation. Pericardial fluid analysis showed exudative characteristics, with neutrophil and lymphocyte infiltration, negative culture and normal cytology. Repeat echocardiography post



© BMJ Publishing Group Limited 2021. No commercial re-use. See rights and permissions. Published by BMJ.

**To cite:** Grech P, Mangion J, Vella S. *BMJ Case Rep* 2021;**14**:e236704. doi:10.1136/bcr-2020-236704



**Figure 1** CT scan of the thorax showing a large pericardial effusion and bilateral pleural effusions.

pericardiocentesis confirmed no residual right atrial collapse and decreased IVC dilation.

Viral screen including serology for Epstein-Barr virus, cytomegalovirus, toxoplasma, parvovirus, enterovirus and HIV were all negative. Hepatitis screen was also normal. In view of the patient's contact with irregular immigrants, a Quantiferon test was also performed which was negative for tuberculosis. Respiratory screen, blood cultures and urine cultures showed no significant growth. Anti-streptolysin-O-titre (ASOT) was significantly elevated at 422 U/mL (0–200 U/mL).

Rheumatology screen revealed normal rheumatoid factor, normal immunoglobulins, normal complement levels (C3 and C4), positive antinuclear antibodies (1/1000) with normal anti-double-stranded DNA, normal extractable nuclear antigen antibodies and normal anti-neutrophil cytoplasmic antibody. In the absence of connective disease symptoms and signs, an autoimmune aetiology for the polyserositis was deemed unlikely by the consulting rheumatologist.

Familial Mediterranean fever screen and ACE level for sarcoidosis also yielded a negative result. Lactate dehydrogenase (LDH) was also found to be negative.

Given paucity of diagnostic clues, diagnostic pleural tapping was carried out under ultrasound guidance. This confirmed an exudative pleural effusion, with no evidence of empyema (pH 7.72). The fluid tested negative for acid-fast bacilli and the culture yielded growth of *S. mitis*. Acting on advice by infectious diseases specialists, this patient was not treated with antibiotics at this stage given the spontaneous resolution of his symptoms, signs and laboratory parameters, including CRP. A dental review confirmed the presence of dental caries, necessitating extraction of a molar tooth.

#### OUTCOME AND FOLLOW-UP

The patient remained well and continued colchicine for 3 months. Repeat echocardiograms 12 days after colchicine was started and again 3 months afterwards which showed no fluid reaccumulation. A repeat chest X-ray was done 3 weeks after colchicine initiation, and it showed complete resolution of bilateral effusions. Repeat abdominal ultrasound carried out 1 month after initiation of therapy confirmed resolution of ascites. Regular dental reviews ensured maintenance of a healthy buccal cavity. The patient was followed up after cessation of treatment

for two consecutive years and he remained well with no signs of recurrence.

#### DISCUSSION

The aetiology of polyserositis is complex. There is a wide range of causes which can be classified into infective, malignant, iatrogenic, metabolic and autoimmune. In this case report, we highlight the diagnostic workup of polyserositis to identify the underlying aetiology.

*S. mitis* is an alpha-haemolytic streptococcus part of the *Streptococcus viridans* family. It is mostly found as a commensal in the buccal cavity. The *S. mitis* group of organisms contains several species and is biochemically very inert, which can make species level identification very challenging. It is closely related to *S. pneumoniae* and other *S. mitis* group organisms and therefore accurate identification can be difficult. In our case, *S. mitis* was detected on pleural effusion culture, although not on culture of pericardial fluid. ASOT was also positive, supporting the diagnosis.<sup>1</sup>

*S. mitis* can be commonly associated with infections in the immunocompromised. It can be responsible for pneumonia, septicaemia, infectious endocarditis, pericarditis, ophthalmitis and meningitis.<sup>2–5</sup> There is also documentation of urinary tract *S. mitis* infection.<sup>6,7</sup> *S. mitis* has been linked to hepatitis and spontaneous bacterial peritonitis in cirrhotic patients.<sup>8</sup> In our case, *S. mitis* complicating dental caries predominantly affected this otherwise healthy patient's serous membranes, leading to polyserositis manifesting with epigastric pain and exertional dyspnoea. There are various post-streptococcal infection autoimmune disorders such as rheumatic fever, post-streptococcal glomerulonephritis, post-streptococcal reactive arthritis, post-streptococcal uveitis and paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections.<sup>8</sup> To our knowledge, this is the first reported case of *S. mitis* post-streptococcal polyserositis.

Given the young age of the patient and the absence of renal and thyroid abnormalities, connective tissue disease, an underlying haematological malignancy such as acute myelogenous leukaemia and chronic myeloid leukaemia, pheochromocytoma and sarcoidosis had to be excluded.<sup>9–12</sup> The low ESR and the normal autoimmune screen made connective tissue diseases unlikely. The probability of an underlying lymphoproliferative disorder was also low with a normal blood film and LDH and no significant lymphadenopathy on CT scan. In view of the patient's ethnicity and presentation, familial Mediterranean fever also needed to be excluded. The albumin level was normal therefore polyserositis could not be secondary to hypoalbuminaemia.

Our patient warranted an immediate pericardiocentesis given that the European Society of Cardiology (ESC) scoring for cardiac tamponade was 10 (if >6 needs urgent drainage).<sup>13</sup> The removal of the large pericardial effusion alleviated the pressure on the right side of the heart as evidenced on the post-pericardiocentesis echocardiogram.

Colchicine played an important anti-inflammatory role in mitigating serositis and hence further accumulation of fluid in the serous cavities. Evidence suggests that if aspirin and non-steroidal are not enough to treat polyserositis or prevent recurrence, long-term colchicine is the second-line agent. If still resistant to colchicine, steroids are the third-line agents of choice. In very persistent pericarditis, immunomodulators such as azathioprine, intravenous immunoglobulins and anakinra can be used.<sup>14</sup> Given the low inflammatory markers, and the patient was afebrile throughout the admission, an ongoing

infective process was unlikely and therefore antibiotics were not administered. After stopping a 3-month course of colchicine, the patient remained well and had no recurrence. According to the ESC guidelines for treatment of pericarditis, colchicine prevents chemotaxis, degranulation and phagocytosis thereby reducing serous membrane inflammation in the long term.<sup>13</sup>

Dental hygiene is vital to prevent *S. mitis* bacteraemia. Our patient was additionally encouraged to get the pneumococcal vaccine two times per day to reduce the risk of recurrence of infection. Evidence shows that the pneumococcal vaccine can help prevent *S. mitis* infections since *S. mitis* has a similar structure to *S. pneumoniae* and belongs to the same streptococcal group.<sup>15</sup>

### Patient's perspective

I was very anxious at the start of the illness as I had no idea what could have caused the polyserositis. I had an immediate symptomatic relief with the therapeutic pericardiocentesis. Once *S. mitis* was cultured and the dental review confirmed the source of infection I was more reassured. I am glad that so far I did not get a recurrence.

### Learning points

- ▶ The aetiology of polyserositis is complex. There is a wide range of causes which can be classified into infective, malignant, iatrogenic, metabolic and autoimmune.
- ▶ *Streptococcus mitis* is an alpha-haemolytic streptococcus which is mostly found as commensal in the buccal cavity.
- ▶ Serious *S. mitis* infections usually occur in immunocompromised or those with underlying conditions.
- ▶ Infections in otherwise healthy individuals are rare but may occur.
- ▶ Dental caries can be a source of *S. mitis* polyserositis.
- ▶ Urgent drainage of pericardial effusion is needed when ESC score is >6 for cardiac tamponade.

**Contributors** PG: corresponding author. JM: coauthor. SV: supervising author.

**Funding** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

**Competing interests** None declared.

**Patient consent for publication** Obtained.

**Provenance and peer review** Not commissioned; externally peer reviewed.

### REFERENCES

- 1 Werno AM, Christner M, Anderson TP, *et al.* Differentiation of *Streptococcus pneumoniae* from nonpneumococcal streptococci of the *Streptococcus mitis* group by matrix-assisted laser desorption ionization-time of flight mass spectrometry. *J Clin Microbiol* 2012;50:2863–7.
- 2 Shelburne SA, Sahasrabhojane P, Saldana M, *et al.* *Streptococcus mitis* strains causing severe clinical disease in cancer patients. *Emerg Infect Dis* 2014;20:762–71.
- 3 Zbinden A, Mueller NJ, Tarr PE, *et al.* *Streptococcus tigurinus*, a novel member of the *Streptococcus mitis* group, causes invasive infections. *J Clin Microbiol* 2012;50:2969–73.
- 4 Douglas CW, Heath J, Hampton KK, *et al.* Identity of viridans streptococci isolated from cases of infective endocarditis. *J Med Microbiol* 1993;39:179–82.
- 5 Kutlu SS, Sacar S, Cevahir N, *et al.* Community-acquired *Streptococcus mitis* meningitis: a case report. *Int J Infect Dis* 2008;12:e107–9.
- 6 Swain B, Otta S. *Streptococcus mitis*: An Unusual Causative Agent for Urinary Tract Infection. *J Lab Physicians* 2013;5:144–5.
- 7 Chou Y-S, Horng C-T, Huang H-S, *et al.* Reactive arthritis following *Streptococcus viridans* urinary tract infection. *Ocul Immunol Inflamm* 2010;18:52–3.
- 8 Galanakis E, Kritikou-Pliota E, Pappa C, *et al.* *Streptococcus mitis* septicaemia and hepatitis. *Scand J Infect Dis* 2000;32:214–5.
- 9 Heidarpour M, Haghighatpanah MA, Rezvanian H, *et al.* Polyserositis: an extremely rare life-threatening manifestation of pheochromocytoma. *Case Rep Endocrinol* 2020;2020:1–3.
- 10 Kunnumpurath A, Desikan SP, McClain C, *et al.* Chronic myelomonocytic leukemia presenting with polyserositis and seropositivity for rheumatoid arthritis. *J Investig Med High Impact Case Rep* 2020;8:232470962096686.
- 11 Pi Y, Wang B, Wang L, *et al.* Polyserositis as a primary clinical manifestation of CD7+ acute myelogenous leukemia with myeloid sarcoma: a case report. *Medicine* 2020;99:e23615.
- 12 Qiu JK, Dwivedi A, Alter E, *et al.* A rare case of Sarcoidosis-Induced polyserositis and steroid-induced mediastinal lipomatosis masquerading as an epicardial tumor. *CASE* 2020;4:166–9.
- 13 Adler Y, Charron P, Imazio M, *et al.* 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J* 2015;36:2921–64.
- 14 Imazio M, Lazaros G, Brucato A, *et al.* Recurrent pericarditis: new and emerging therapeutic options. *Nat Rev Cardiol* 2016;13:99–105.
- 15 Reglinski M, Ercoli G, Plumtre C, *et al.* A recombinant conjugated pneumococcal vaccine that protects against murine infections with a similar efficacy to Prevnar-13. *NPJ Vaccines* 2018;3:53.

Copyright 2021 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <https://www.bmj.com/company/products-services/rights-and-licensing/permissions/>  
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

#### Customer Service

If you have any further queries about your subscription, please contact our customer services team on +44 (0) 207111 1105 or via email at [support@bmj.com](mailto:support@bmj.com).

Visit [casereports.bmj.com](http://casereports.bmj.com) for more articles like this and to become a Fellow