

Case Report - Group A Streptococcus Primary Peritonitis a Rare Mimic of Pelvic Inflammatory Disease



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Investigations

Hb: 101g/L (115-165), WCC 16.6 (4-11), neutrophils 15.8 (2-8)
UEC - Creatinine: 274 micromol/L (40-90), eGFR 17ml/min/1.73m2 (>60) CRP 422 (<10) | B-HCG: negative | Lactate 3.8 (<2) LFTs - Bilirubin: 38 micromol/L (<20), AST 36 units/L (<30)
Cultures (collected after commencement of antibiotics) - High vaginal swab MCS: no growth | Urine MCS: no growth | Blood cultures: no growth Chlamydia/gonorrhoea high vaginal swab NAAT: negative
CT abdomen-pelvis: 32x44mm fluid collection in the mid-right pelvis with adjacent thickening and mucosal enhancement of the rectum. Multiple small pockets of free fluid within the adnexae and paracolic gutters. Appendix not visualised. Upper abdominal viscera normal. | TTE: NAD Peritoneal fluid MCS: moderate numbers of polymorphs. Growth of Group A streptococcus (pan sensitive), see figure 2.

Management

She was admitted under gynaecology and given a dose of IV ceftriaxone which was escalated to IV azithromycin, IV metronidazole and IV piperacillin-tazobactam for suspected PID. Despite IV antibiotics she deteriorated over the next eight hours and an emergency diagnostic laparoscopy was performed which showed widespread purulent fluid in the pelvis with some reactive inflammation but no obvious source (no tuboovarian abscess, appendix and bowels appeared normal), see figure 1. A 3L pelvic lavage was performed.





Figure 2: Gram stain microscopy of GAS²

Progress

The patient had **significant clinical improvement post laparoscopy** and washout, with weaning of vasopressors day one post and she remained afebrile. Day two post-operation with the peritoneal MCS result available, her antibiotics were rationalised to IV ceftriaxone and oral metronidazole. On day five she was deescalated to oral cefuroxime with plans to complete two weeks total oral antibiotics and her renal function had returned to normal. She was discharged on day 12 of admission and was well when reviewed one month after discharge in outpatient clinic.

Discussion and Conclusion

Invasive GAS disease including primary peritonitis, is a lifethreatening condition that is increasing in prevalence in Australia.³ The mechanism of infection in primary GAS peritonitis is not well understood. Haematological spread, translocation from the gastrointestinal tract or a scending infection from the female genitourinary tract have been proposed as likely sources in several large case series. ^{1,4} This patient had a personal history of recent localised GAS infection which is a significant risk factor. ^{1,4}

Primary GAS peritonitis should be considered as a differential in patients with an acute abdomen and shock, particularly after a recent localised GAS infection or exposure. This is pertinent in this case as the addition of clindamycin to her early antibiotic regimen to inhibit GAS toxin production may have been beneficial given her rapid deterioration and multiorgan involvement.

The role of laparoscopy in diagnosis and treatment of this condition is subject to debate, however most literature recommends that laparoscopy and lavage for source control be considered if there is lack of response to antibiotic therapy. ^{4,5} The benefit of this approach is supported in this case study.

Background

Group A Streptococcus (GAS), primarily a pathogen of the skin and upper respiratory tract, is an uncommon but serious cause of infection in the puerperium and following gynaecological procedures. Primary **GAS peritonitis is form of invasive GAS disease with a high mortality rate**, in which GAS infects the peritoneal cavity without a defined intraabdominal source. ¹ This rare pathology **predominantly affects women**, with a significant proportion of patients in other case reports admitted and managed by gynaecology, with many presumptively treated as pelvic inflammatory disease (PID). ¹

Case

A previously healthy premenopausal woman presented with **four days of abdominal pain, fevers and diarrhoea.** She had no abnormal vaginal discharge, and her last normal

menstrual period was three days prior.

She had a long term monogamous female sexual partner, no recent gynaecological procedures and no history of sexually transmitted infections.

She had no significant medical or surgical history. She had a mild penicillin allergy. Her obstetric history was a vaginal birth eight years prior. She did not have an intrauterine device in situ. Of note, she had recently been treated with oral antibiotics for GAS pharyngitis diagnosed on oropharyngeal swab three weeks prior.

Examination Findings

She was febrile (38.6C) with septic shock (HR of 120 and BP 80/50). She appeared pale and unwell with a **peritonitic abdomen**. She had no rash or other localising symptoms. On bimanual examination she was tender in the adnexae. On speculum there was **no abnormal vaginal discharge**. She rapidly deteriorated with septic shock and required vasopressor support in intensive care and developed oliguria.

References

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