Spontaneous Splenic Laceration - A Rare and Unusual Complication of A Tubo - Ovarian Abscess

Hazarathamma Makam1*; Ami Shukla1; Anil Darbar2

1 Northampton General Hospital NHS Trust, Northampton, United Kingdom.
2 University Hospitals of Leicester NHS Trust, Leicester, United Kingdom.

*Corresponding Author(s): Hazarathamma Makam
Northampton General Hospital NHS Trust, Northampton, United Kingdom.
Email: hazaramakam@gmail.com

Abstract

A Tubo-Ovarian Abscess (TOA) is a complex potentially life-threatening condition associated with an inflammatory mass involving the fallopian tube and ovary as a sequel of Pelvic inflammatory disease (PID), especially in women of the reproductive age. TOA may be associated with sepsis due to a ruptured abscess causing end organ damage and thus requiring aggressive medical intervention. Patients may present with systemic signs of being unwell—consisting of fever, severe constant lower abdominal pain, diarrhoea, systemic inflammatory response syndrome, sepsis, spontaneous visceral rupture and haemorrhagic shock. Although spontaneous visceral/splenic laceration and rupture are very rare, it can be potentially fatal necessitating a splenectomy. Exact aetiology of non-traumatic splenic rupture in TOA remains unclear.

We report a very interesting and unusual case of TOA which was refractory to antimicrobial therapy and complicated by a non-traumatic rupture of the spleen leading to major haemorrhage culminating in a splenectomy. Our case provides an insight into the risk factors that could predispose an individual to TOA and highlights the need for a collaborative multidisciplinary approach in the management of such patients to ensure a favourable outcome.

Keywords: Non traumatic splenic rupture; Splenectomy; Pelvic inflammatory disease.

On examination, her abdomen was diffusely tender and associated with guarding. Speculum examination findings were normal. She couldn’t tolerate bimanual examination due to pain. High vaginal, endocervical and rectal swabs were taken. Her other systemic examination was unremarkable. Urine pregnancy test was negative, C-reactive protein (CRP) was 510 and urine dipstick was normal. Other biochemical and hematological investigations were within normal limits with a Hemoglobin of 140g/l, WCC of 7 x 10^9/l and a serum Creatinine of 125 micromoles/l.

Computerised Tomography (CT) abdomen & pelvis showed IUCD coil insitu in an optimal position, PID with bilateral cystic mass measuring 9-10cms, pyosalpinx and moderate amount of free fluid suggestive of TOA. She commenced on intravenous (IV) fluids, IV Cefuroxime 1.5 g tds, IV Metronidazole 500 mgs tds and oral Doxycycline 100 mg bd. Repeat blood investigations a day later showed an increase in CRP to 648, Creatinine 224 micromoles/l, WCC 8 x 10^9/l, and Hemoglobin135 g/l.

Trans vaginal ultrasound scan (TV USS)confirmed a bilateral TOA measuring approximately 10 cms in size (Figure 1). IUCD was removed within the first 24 hours after admission and sent for culture and sensitivity. Intravenous (IV) Tazocin 4.5 g tds was commenced as per Microbiology advice.

During the subsequent days Gonorrhoea, Chlamydia, Hepatitis B, Syphilis and HIV serology were negative. Blood film for Malaria and sputum for acid fast bacillus were negative.

On day 5, she complained of dyspnoea and bilateral back pain at T10-11 levels. There were no cutaneous changes suggestive of intraperitoneal bleeding. The patient was hemodynamically compensated with tachycardia (a HR of 110-120/ min), BP of 100/ 85 mm hg and a capillary refill time of 3 seconds. In view of a possible Pulmonary Embolism (PE), she was commenced on 40 milligrams of subcutaneous Enoxaparin twice a day and an urgent CT pulmonary angiogram (CTPA) was expedited-which didn’t reveal any thrombus in the pulmonary trunk but revealed a splenic rupture and splenic haematoma (Figure 2), consolidation of right lower lobe of the lung, bilateral pleural effusion and atelectasia. The emergency operating room team, General Surgical Consultant and the on call Gynaecology Consultant were informed. The patient was rushed for an emergency Laparotomy. IV Clindamycin 900 mgs tds and Gentamicin 5mg/kg od for 14 days were started intra-operatively as per Microbiology advice.

Intra-operatively, massive hemoperitoneum was noted due to a ruptured spleen. Splenectomy and drainage of pelvic abscess was performed. Estimated intra operative blood loss was 3 litres requiring a transfusion of 5 units of packed red blood cells and 2 units of fresh frozen plasma. The patient’s cardiovascular state remained labile requiring vasopressor infusion through a Central Venous Catheter (CVC). Drained abscess cavity samples were sent for cytology and cultures. She was transferred to the Intensive Care Unit (ICU) for post-operative invasive monitoring and care. Blood films for electropheresis and serum free light chain were requested to rule out any myeloproliferative disorders whilst the patient was in the ICU. In addition, blood tests had ruled out the possibility of Infectious Mononucleosis.

The patient was transferred back to the Gynecology ward on the third postoperative day from the ICU. Copper IUCD culture, peritoneal fluid cultures and rectal swabs didn’t show any growth. High vaginal swab showed mixed growth. Patient showed a gradual improvement whilst on multiple antibiotics. Repeat CT abdomen pelvis on the seventh postoperative day showed small bilateral pleural effusion, a pelvic cyst of approximately 11cm in size, mesenteric inflammation and small collection around 4cm in size in the right sub hepatic region. Antibiotics were changed to IV Benzylpenicillin 2.4 grams qds and Ciprofloxacin 500mg bd to treat a possible Actinomyces infection as per Microbiologist’s advise and Gentamicin and Clindamycin were stopped. Although Actinomyces was not confirmed on microbiological tests, antibiotics were changed in view of potentially infrequent Actinomyces infection.

On the 11th postoperative day, a TV USS showed pelvic collections around 5-6cms in size (Figure 3). Following a multidisciplinary team meeting (MDT), the plan was to add oral Amoxicillin 500 mgs tds for 7 days, repeat an USS in 5 days time and to radiologically aspirate collections if patient was not improving clinically. The patient continued to show clinical and biochemical signs of improvement with a CRP of 50, WCC 9 x 10^9/l and a serum Creatinine of 130 micromoles/l. A repeat USS of the pelvis had confirmed that there was no change from the previous scan. Therefore, she was discharged home three weeks after the laparotomy with an outpatient follow up (FU). She was discharged on oral Doxycycline 200mg for 2 weeks and then to start Penicillin V as per post-splenectomy protocol. Post -splenectomy vaccines were given before discharge from the hospital.
Outcome and follow-up

On FU 4 weeks after the discharge from the hospital, she was clinically stable with a CRP of 16, Hb-107g/l, WBC 9 x 10^9/l, Platelets 629 x 10^9/l and serum electrophoresis results were unremarkable. USS of the pelvis showed normal anteverted uterus with endometrial thickness of 3mm, 3cm to 4cm size bilateral cystic areas with areas of haemorrhage within. No Doppler flow was seen in the cystic area and no free fluid was seen in the Pouch of Douglas (POD). Histology of the spleen suggested a splenic hilar tear and evidence of focal haemorrhage.

Discussion

TOA is a potentially fatal condition arising as a result of PID, especially in the women of reproductive age (in 17-20% of cases) [1,2] and in nearly 60% of nulliparous women [3]. In post-menopausal women, TOA has been associated with a high risk of malignancy [2]. Risk factors for TOA consist of history of previous PID in approximately 46% of patients [1,4,9], multiple sex partners, presence of an IUCD and failure to use barrier contraceptive. Patients with TOA may present with a constant lower abdomen pain, abnormal vaginal discharge, diarrhoea, dysuria, dyspareunia, irregular vaginal bleeding, signs suggestive of systemic inflammatory response syndrome [7,10]. During the examination, clinicians may elicit guarding, rigidity and cervical motion tenderness [2,8]. TOA could be polymicrobial and thus refractory to antimicrobial therapy. It may be rarely associated with Actinomyces and Tuberculosis. TOA may be complicated by a ruptured abscess, secondary inflammation, damage to the adnexal tissue in women of reproductive age [8] and end organ damage due to sepsis requiring aggressive antimicrobial therapy and surgical intervention in 15% of cases [4].

A high CRP could be a sensitive indicator of TOA [5,6]. CT and MRI may be required when Ultrasonographic findings are inconclusive [8,11]. CT findings for TOA include cystic adnexal masses with internal septations, presence of internal gas bubbles and loss of fat between the planes [12]. A TV scan may demonstrate retrotuereine mass (cystic or solid), free fluid in the cul-de-sac and indistinct fallopian tubes and /or uterine margins [13,14]. Prompt diagnosis and aggressive inpatient medical management with IV antibiotics following a consultation with the Microbiology team for up to 48-72 hours has shown to improve clinical outcome in majority of the patients [15]. Image guided early percutaneous or TV drainage appears to be gaining an interest and may minimise the need for a surgical intervention.

The exact aetiopathogenesis of non-traumatic splenic rupture or laceration in PID remains unclear. Presence of increased laxity of splenic ligament or a wandering spleen [16] and an ascending pelvic infection could be one plausible explanation for a non-traumatic splenic laceration or rupture in TOA. TOA in immunocompromised individuals could potentially predispose to spontaneous non-traumatic rupture of Spleen leading to haemorrhagic shock due to ascending infection in the peritoneal cavity [7]. Approximately 27% of non-traumatic splenic ruptures are associated with Ebstien Barr Virus causing Infectious Mononucleosis, Cytomegalovirus, Tuberculosis, HIV, Malaria, Infective Endocarditis and Typhoid [10]. Patients’ with splenic rupture or laceration may complain of pain the left upper quadrant associated with guarding and rigidity suggestive of peritonism. There may be cutaneous discoulouration of skin in the left upper quadrant and may complain of referred pain in the left shoulder (Kehr sign). Prompt and early diagnosis and multidisciplinary intervention could minimise the morbidity and mortality associated with a splenic rupture.

Conclusion

TOA is a recognised complication of PID which requires prompt diagnosis, early senior MDT involvement, commencement of broad spectrum IV antibiotics and necessary intervention in order to improve the patient outcome. Patients’ refractory to medical management may require an early surgical intervention to minimise the morbidity and mortality. Although various infective conditions have been implicated to predispose an individual to a non-traumatic laceration of the spleen, the exact aetiopathogenesis remains unclear. Splenic laceration is a rare complication with a significant morbidity and mortality and it is imperative for the clinicians to have an index of suspicion about this association in TOA despite the absence of cutaneous or flank discoulouration suggestive of intraperitoneal bleeding. The exact timing of a surgical intervention remains unclear and has to be individualised.

Learning points/Take home messages

- TOA is a potentially life threatening condition associated with sepsis and multi-organ dysfunction.
- Prompt diagnosis and initiation of management through a collaborative multidisciplinary approach is vital in minimising the associated morbidity and mortality due to TOA.
- Splenic rupture requiring splenectomy is a rare complication of TOA and having a high index of suspicion about this association could be vital for the clinicians as it may impact the overall outcome of the patients’

References


