CASE REPORT

Splenic necrosis requiring ultrasound-guided drainage following meningococcal septicaemia

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Abstract

Splenic necrosis is an extremely rare complication in the context of meningococcal septicaemia and disseminated intravascular coagulopathy. We present the case of a previously healthy 22-year-old male who was diagnosed and treated for meningococcal septicaemia. He represented 4 days following discharge with significant splenic necrosis and associated abscess formation despite previously unremarkable imaging on his first admission. The splenic collection was successfully treated with ultrasound-guided percutaneous drainage. We discuss the leading causes of atraumatic splenic infarction and the recent shift towards treating splenic necrosis with minimally invasive procedure.

INTRODUCTION

Neisseria meningitidis is an aerobic gram negative diplococci and commensal bacterium of the human nasopharynx [1]. This is acquired through direct physical contact or prolonged close contact by droplet transmission. Nasopharyngeal carriage rates of *N. meningitidis* without invasive disease is estimated between 10–35% across developed countries [1]. Immunosuppressive factors such as human immunodeficiency virus (HIV) may increase the risk of progression to an active disease state of meningococcal septicaemia. Meningococcal septicaemia is a devastating disease resulting in fatal outcomes without urgent medical intervention. Recent literature reports the mortality rate of meningococcal disease between 10% and 15% of cases [2].

CASE REPORT

A 22-year-old male was admitted to our rural hospital with one-day history of sudden onset fevers, headache and generalized myalgia as well as several days of non-productive cough. He denied photophobia, neck stiffness, urinary and bowel symptoms. He reported to be recently well before presentation. His only medical background was Q fever.

On examination, the patient was febrile and tachycardic with no increased work of breathing. His chest was clear on auscultation and abdomen was soft and non-tender. The rest of the examination was unremarkable with no localizing sign of infection. The patient deteriorated overnight with progressive onset of severe epigastric pain, vomiting and persisting fevers. His abdomen was soft with generalized tenderness across the epigastrium. The patient developed septic shock and urgently transferred by an aeromedical retrieval service to the intensive care unit of the closest regional hospital. Noradrenaline infusion and intravenous piperacillin-tazobactam were commenced on arrival.

Blood tests revealed ischaemic hepatitis (ALP 85 U/l, GGT 77 U/l, ALT, 126 U/l, AST 337 U/l), acute kidney injury (Creatinine 124 μmol/l, urea 8.0 mmol/l) and disseminated intravascular coagulopathy (DIC) (INR 2.9, prothrombin time 33 seconds, activated partial thromboplastin time 53 seconds, fibrinogen 2.8 g/l, D-dimer 115.24). Screening for hepatitis B, hepatitis C and HIV were negative. Lumbar puncture was not

performed at our rural hospital due to the lack of on-site pathology laboratory to process this investigation. Abdominal ultrasound and contrast enhanced CT abdomen/pelvis did not demonstrate any pathology.

Three days following initial presentation, blood culture revealed *N. meningitidis*. A seven-day course of intravenous benzylpenicillin was completed for meningococcal septicaemia. The patient clinically improved and discharged from hospital after 10 days.

The patient represented to our hospital 4 days later with mild intermittent left upper quadrant abdominal pain. He was otherwise systemically well. The left upper quadrant was tender on palpation, however the remaining examination was unremarkable and he was haemodynamically stable.

The spleen was grossly abnormal on abdominal ultrasound with significant fluid replacement of the normal splenic tissue consistent with splenic necrosis (Fig. 1). CT abdomen/pelvis reported extensive splenic necrosis measuring 11.5 cm × 8.3 cm × 13.8 cm with no normal splenic tissue on imaging (Fig. 2). There was associated abscess formation contained inside an expanded splenic capsule (Fig. 3). The associated vessels were of normal appearance without any evidence of thrombus.

After excluding a splenic artery aneurysm on CT angiogram, the treating team decided for percutaneous drainage of the splenic abscess. With the patient under conscious sedation, an 8 French percutaneous catheter (Cook Medical, Bloomington, IN, USA) was successfully inserted under ultrasound and 550 ml of thick altered blood was drained. There was no growth from this collection. The patient recovered well from the procedure and he was discharged 3 days later. The patient will receive the post-splenectomy vaccines and antibiotics prophylaxis as per the Australian clinical guidelines.

DISCUSSION

Patients with meningococcal disease predominantly first present with prodromal symptoms consistent with an upper respiratory tract infection. This is followed by rapid deterioration including sudden onset nausea and vomiting, fevers, arthralgia, myalgia, petechial rash and drowsiness [3].

Inside the blood stream, *N. meningitidis* undergoes rapid bacterial proliferation with consequent production of lipopolysaccharides (LPS). LPS is critical in driving the systemic inflammatory response and circulating levels of this endotoxin is associated with the mortality rate in meningococcal septicaemia [1, 3]. Inflammatory mediators released secondary to LPS activate various pathways such as the complement system and the coagulation cascade. This ultimately contributes to septic shock and DIC. Multiple organ dysfunction syndrome from widespread thrombosis throughout the respective microcirculation is a significant determinant of mortality in meningococcal septicaemia [4].

Splenic infarction secondary to DIC in meningococcal septicaemia is an extremely rare complication. As there was extensive splenic necrosis, as opposed to segmental infarction, we believe the splenic artery or vein became significantly occluded before dividing into its branches during the pro-coagulant phase of the systemic inflammatory response.

Splenic infarction may be classified as traumatic or atraumatic in nature. Infection, haematological and non-haematological malignancies represented the three most common underlying aetiologies of atraumatic splenic infarction following two recent systematic reviews of over 600 and 800 patients respectively [5, 6]. Malaria, infectious mononucleosis and cytomegalovirus are the
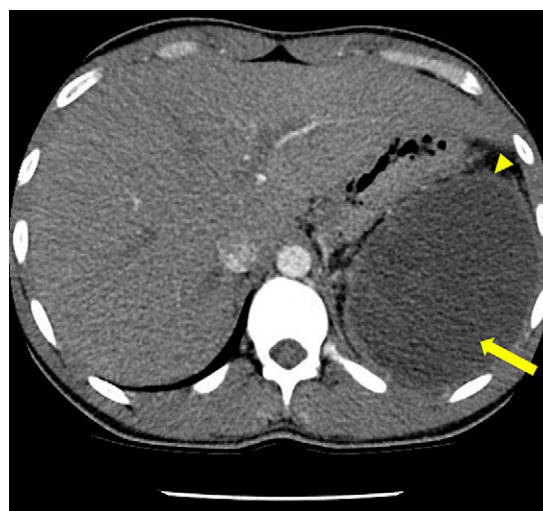

Figure 2: Extensive splenic necrosis with associated abscess formation on the axial section of CT abdomen/pelvis (block arrow). The splenic collection was contained inside an expanded splenic capsule (arrowhead). There was no free fluid inside the abdominal cavity.

Figure 1: Abdominal ultrasound performed on re-presentation revealed a grossly abnormal spleen (block arrow) with complete fluid replacement of the splenic tissue.
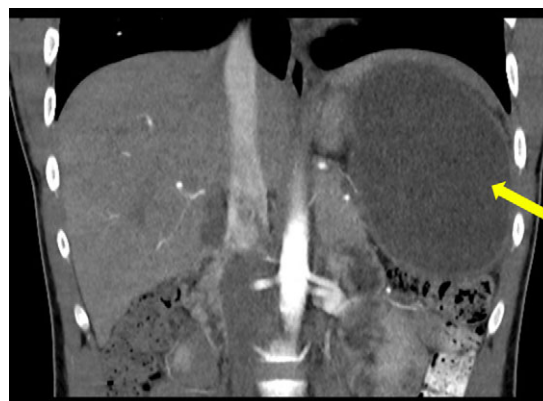

Figure 3: Coronal section of the CT angiogram providing another view of the splenic collection (block arrow). No residual enhancing splenic tissue was present. There was no splenic artery aneurysm.

most common infectious causes. Splenomegaly, age greater than 40 years of age and presence of neoplastic disease were recognized as the strongest risk factors for mortality in the second review [6].

Patients with splenic infarction classically present with a triad of acute left upper quadrant pain, palpable swelling over the affected region and fevers. CT abdomen/pelvis with contrast is the preferred imaging modality, however diagnosis may require surgical intervention. On imaging, as the infarction matures the spleen may undergo one of the following: resolution, contraction of the splenic capsule, scarring or liquefaction [7].

Antibiotics therapy with or without splenectomy has been the traditional management of splenic abscess, an uncommon but potentially life threatening complication of splenic infarction. However, recently ultrasound- or CT-guided percutaneous drainage is regarded as a safe, effective and less invasive alternative [8, 9]. The following pathology results are recommended before proceeding: minimum platelet count greater than 50 000/μl, INR less than 1.2 and activated partial thromboplastin time between 20–33 seconds [8, 10]. The presence of unilocular splenic collection without an internal septa, a discrete wall surrounding the collection and liquefied content are indications for percutaneous intervention [8]. Advantages of ultrasound include the absence of procedural radiation, providing real time guidance of the catheter during its placement and detecting any loculation or septations inside the splenic architecture to determine the likelihood of successful procedural outcome [10]. Periprocedural complication rate is estimated between 2% and 4% including splenic haemorrhage, pneumothorax, pleural effusion and visceral perforation [8]. Splenectomy is associated with a higher post-operative complication rate between 17% and 27%, predominantly due to increased risk of intra-abdominal spillage, resulting in systemic infection, and venous thromboembolism [8, 10, 11].

CONFLICT OF INTEREST STATEMENT

No conflicts of interest.

FUNDING

ACKNOWLEDGEMENTS:

ETHICAL APPROVAL

No approval was required.

CONSENT

Patient's consent was obtained for this report.

GUARANTOR

Kosuke Kato.

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