ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 04, pp.49698-49700, April, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

CASE STUDY

PNEUMOPERITONEUM DUE TO RUPTURED SPLENIC ABSCESS

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ARTICLE INFO

Article History:
Received 16th January, 2017
Received in revised form
20th February, 2017
Accepted 22nd March, 2017
Published online 30th April, 2017

Key words:

Spleen, Rupture, Abscess,
Pneumoperitoneum, Peritonitis.

ABSTRACT

We present a case of splenic abscess causing pneumoperitoneum in a patient with diabetes mellitus. The presentation was initially vague and later evolved to an acute abdomen. An X-ray of the chest did not reveal pneumoperitoneum which was later detected by a CT scan. Exploratory laparotomy was performed under a clinical suspicion of hollow viscus perforation. This case report emphasizes that a ruptured abscess should be included in the differential diagnosis of acute abdomen in an immunocompromised patient.

Citation: Lee Fei Yee, AndeeDzulkarnaen Zakaria, Luqman Mazlan and Ismail Sagap, 2017. "Pneumoperitoneum due to ruptured splenic abscess", *International Journal of Current Research*, 9, (04), 49698-49700.

INTRODUCTION

Pneumoperitoneum is commonly caused by perforated hollow viscus. We encountered a case of pneumoperitoneum caused by ruptured splenic abscess. The occurrence of splenic abscess is rare and it is potentially a life threatening disease if left untreated. The vague presentation of the disease often leads to delay in diagnosis. However, the recent advances in radiology able to guide us to early diagnosis and provide a timely intervention.

Case report

A 78-year-old man with multiple comorbidities that include diabetes mellitus, hypertension, ischemic heart disease with congestive heart failure and chronic kidney disease presented with vague symptoms of ruptured splenic abscess. He complained of abdominal discomfort, poor oral intake and intermittent fever for 1 week prior to presentation. He was brought into the emergency department by his family members when he was found to be less responsive at home. He also had 1 episode of vomiting on the same day. Clinically, he was dehydrated, Glasgow coma scale (GCS) on arrival was E4V2M4 and febrile. His blood pressure was high, 190/90mmHg with a heart rate of 100bpm and blood glucose of 30mmol/L. Abdomen was soft and distended but had no signs of peritonism. He was resuscitated in the emergency department with intravenous fluids. His GCS and heart rate responded appropriately. Upon reassessment, we noted tenderness over the epigastric region with voluntary guarding and at the same time he developed coffee ground vomiting. We proceeded with a computed tomography (CT) of the abdomen to rule of perforated gastric ulcer in view of the evolving symptoms. The initial laboratory investigations showed normal white blood cell count of 5.3 x 10^9 per liter (L) but the C-reactive protein was elevated 29mg/L. The kidney function deteriorated and raised amylase level (creatinine 329µmol/L, urea 23mg/dL, serum amylase 500unit/L). The chest x-ray showed blunting of the left costophrenic angle and cardiomegaly. No sub-diaphragmatic air was visualized (Fig. 1). CT abdomen revealed a hypodense collection in the inferior pole of the spleen with multiple air pockets and multiple areas of free gases were seen in the right hypochondrium (Fig. 2). The patient was taken into the operating room for emergency laparotomy for suspected perforated viscus. Much to our surprise; the patient did not have a perforated viscus, but rather a splenic abscess that ruptured causing the pneumoperitoneum. Splenectomy was performed and the purulent collection in the abdominal cavity was irrigated with copious amount of water

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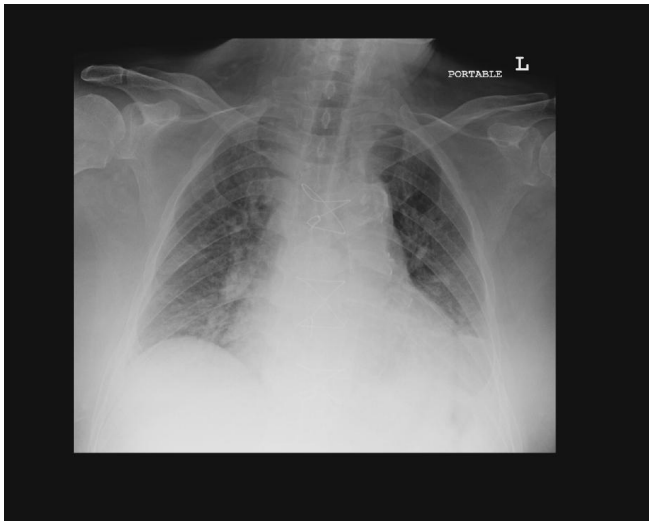
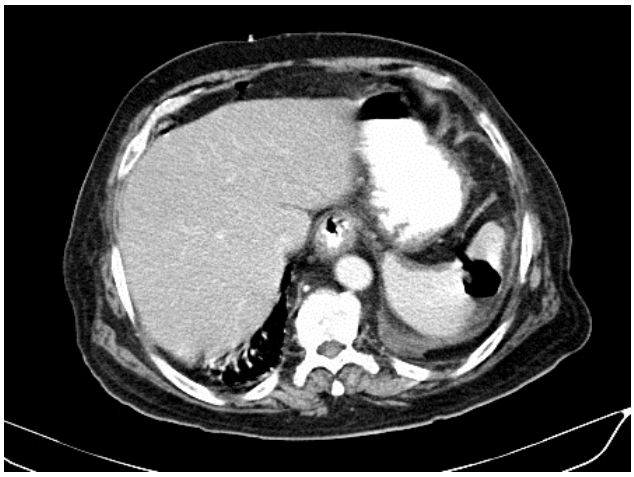


Figure 1. The chest x-ray demonstrate blunting of the left costophrenic angle and cardiomegaly. No sub-diaphragmatic air was visualized



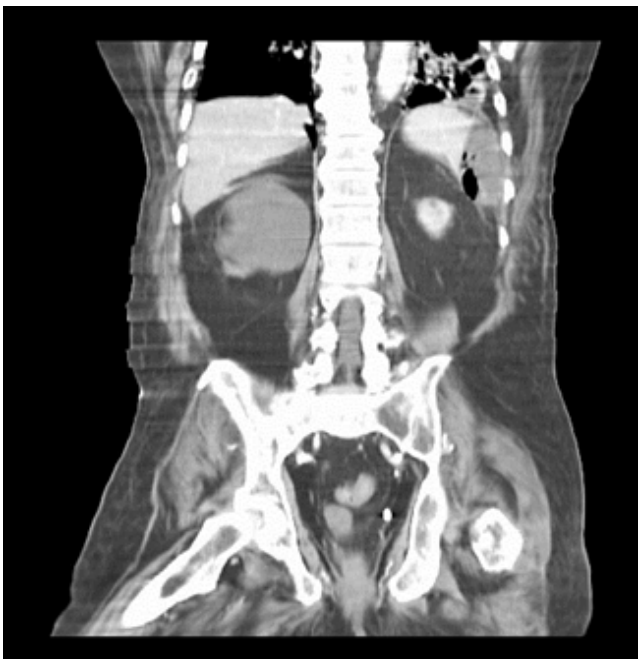


Figure 2. CT scan revealed irregular hypodense collection in the inferior pole of then spleen with multiple air pockets (blue arrow). Bubbles of free gasses seen at the right hypochondrium region (red arrow). Incidental finding of a well-defined exophytic cystic lesion in the upper pole of the right kidney (grey arrow). Left lung basal pleural effusion with emphysematous changes

(Fig. 3). The operation was uneventful. Microbiological culture of the splenic abscess grew Proteus species and the blood cultures were negative. The patient underwent a full work-up looking for possible sources of the splenic abscess. A transthoracic echocardiogram was performed but was negative for any masses, thrombus or vegetation. Macroscopic examination of the spleen revealed an irregular cavity at the mid-pole measuring 70x25x20mm with slough covering the surface. Microscopically there were multiple necrotic centers with the splenic capsule diffusely infiltrated with neutrophils. No evidence of granulomatous inflammation or malignancy. Postoperatively, he was taken to the intensive care unit (ICU) for post-operative care. He was treated for sepsis and broad spectrum antibiotic was initiated. He was extubated on postoperative day 7, however his general condition deteriorated. He developed cardiogenic shock due to non-ST elevation myocardial infarction and the renal function worsened and required dialysis. Unfortunately, he succumbed to death.

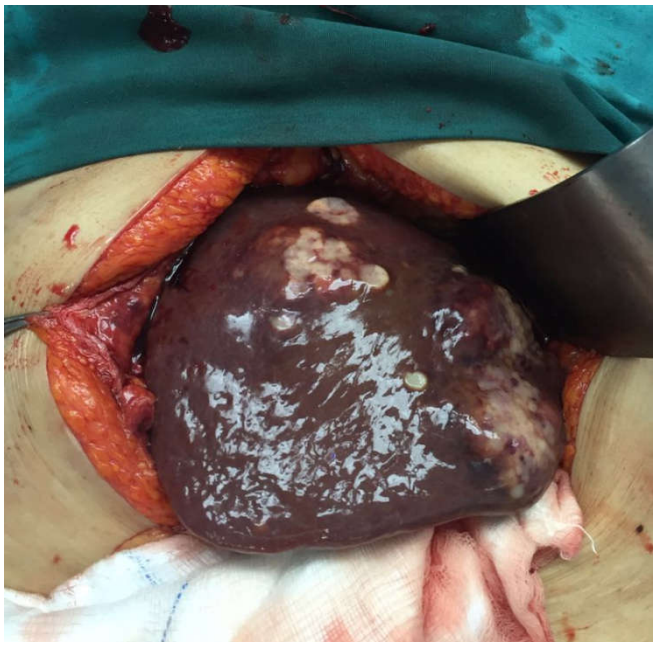


Figure 3. Photography of the spleen shows multiple abscesses

DISCUSSION

The incidence of splenic abscess reported was 0.14-0.7% in autopsy series. (Lee *et al*., 2011; Ferraioli *et al*., 2009) However, in the past decades the incidence has increased from the rising number of immunocompromised patients within the general population due to diabetes, malignancy, use of chemotherapy or immunosuppression drugs and acquired immune deficiency syndrome. (Farres *et al*., 2004; Al-Salam *et al*., 1998) Several other predisposing factors identified include pyogenic infection, splenic trauma, haemoglobinopathies and contiguous disease processes extending to the spleen (Chang *et al*., 2006). The overall mortality in patients with splenic abscess is about 12%, but in case of ruptured splenic abscess and generalized peritonitis may be as high as 20-55%. (Ooi and Leong, 1997) Classical clinical manifestations of splenic abscess are fever and left upper quadrant pain with or without splenomegaly are only present in one-third of cases. Other clinical spectrum ranging from no symptoms to events such as nausea, vomiting, weight loss and poor appetite which are not specific to splenic abscess. (Ferraioli *et al*., 2009; Chang *et al*.,

2006) Clinically, this patient did not have a classical presentation. His presentation was vague and deceiving despite having CT imaging performed. The coffee ground vomitus led us to wonder if this could be a case of perforated gastric ulcer. It has been reported that majority of patients have leukocytosis, however this is not the case in our patient most probably because he is in aimmunocompromised state which is one of the predisposing factor of splenic abscess. (Tung *et al*., 2006) Splenic abscesses are mostly polymicrobial and some are caused by gas-producing organisms. The most common organisms are Streptococus sp. and E.Coli. (Lee *et al*., 2011) Fungal and mycobacterium infection was also reported in recent literature. (Chang *et al*., 2006) In our case, the microorganism isolated from the pus of the splenic abscess was a gas-forming gram negative bacilli, proteus species. When the abscess ruptures, gas liberated from the fermentation by the gas-forming organism forms pneumoperitoneum as seen in our patient. (Chang *et al*., 2006; Ishigami *et al*., 2003) The diagnosis of splenic abscess has been facilitated by the advances of imaging techniques over the years. Screening by X-ray of the chest and abdomen may be used although the diagnostic value is low. The most frequently described features are left pleural effusion, elevation of the left diaphragm and in some cases pneumoperitoneum. Computed tomography and ultrasonography are the studies of choice, the former being the gold standard for its high sensitivity. In our case, CT scan provided important information about the cause of our patient's pneumoperitoneum, as we had assumed it was due to a perforated ulcer. (Chang *et al*., 2006; Ooi and Leong, 1997)

The spleen is important for immunologic function, and a splenectomy will lead to an increased morbidity rate with the risk of post-splenectomy infections. Antibiotics and splenectomy remain as the standard of care in cases of ruptured abscess. (Chang *et al*., 2006) However, image-guided percutaneous drainage is also a therapeutic option in selected cases. (Tung *et al*., 2006) The outcome of splenic abscess is usually favorable in early intervention; however the ultimate prognosis depends on the underlying process predisposing the patient to the development of splenic infection. Our patient isimmunocompromised and the abscess has ruptured causing peritonitis and septicemia post splenectomy. Mortality rate for this patient can potentially reach 100%. (Al-Salam *et al*., 1998)

Conclusion

In conclusion, although ruptured splenic abscess is a rare cause of acute abdomen, it should be considered in the differential diagnosis, especially in an immunocompromised patient. We emphasize that clinicians should have a high index of suspicion in order to achieve an early diagnosis and timely appropriate management for a better prognosis.

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