CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 29 (2016) 241–244

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Spontaneous rupture of splenic hematoma in a malaria patient: Case report and review of literature

Bassem M. Abou Hussein (General Surgery Specialist)[a,*],
Ali M. Al Ani (General Surgery Senior Specialist)[a],
Omar Al-Mayoofi (Infectious Disease Senior Specialist)[b],
Mahpara Mehraj (General Surgery Resident)[a], Afra A. Joher (General Surgery Resident)[a],
Juanita A. Bonilla (General Surgery Specialist)[a],
Alya S. Al-Mazrouei (General Surgery and Laparoscopy Consultant)[a],
Faisal M. Badri (General Surgery and Laparoscopy Consultant)[a]

[a] General Surgery Department, Rashid Hospital, Dubai Health Authority,Dubai, United Arab Emirates
[b] Infectious Disease Unit, Rashid Hospital, Dubai Health Authority, Dubai, United Arab Emirates

ARTICLE INFO

Article history:
Received 7 August 2016
Received in revised form 4 October 2016
Accepted 5 October 2016
Available online 11 October 2016

Keywords:
Spleen
Rupture
Hematoma
Spontaneous
Malaria

ABSTRACT

INTRODUCTION: Blunt abdominal trauma is the most common cause of splenic rupture. Malaria is the most frequent tropical infectious cause of spontaneous splenic rupture. The exact mechanism is not well-defined.

CASE REPORT: We report a case of thirty-year-old male patient known to have malaria who presented with spontaneous splenic rupture. A trial of conservative treatment failed and splenecomy was done to control bleeding.

CONCLUSION: Spontaneous splenic rupture should be kept in mind in malaria patients presenting with left upper quadrant pain and signs of hypovolemia. Early diagnosis and treatment is essential.

1. Background

Blunt abdominal trauma is the most common cause of splenic rupture. Non-traumatic causes of splenic rupture include infection (Malaria is the most frequent tropical infectious cause), malignancy, vascular, or genetic disorders. The exact mechanism of spleen rupture is poorly defined. It should be kept in mind in patients presenting with clinical picture of splenic rupture and no history of trauma. Conservative treatment can be an option in stable patients while splenectomy remains the treatment of choice in patients with a hemoperitoneum and persistent instability.

2. Case report

Thirty-year –old male Pakistani patient, known to have malaria (plasmodium vivax) on treatment, presented to the emergency room with one-week history of headache associated with generalized body ache and dizziness. The patient reported fever, chills and rigors along with epigastric pain. Epigastric pain was present since one week but increased suddenly in the last twelve hours prior to presentation. This was associated with vomiting, loose stools and dark urine. No other complaints and no history of abdominal trauma. The patient reported history of recent travel to his country three months prior to presentation.

On examination, the patient was sweating profusely, conscious, oriented and jaundiced.

Vitals signs: His temperature (T) was 38 ◦ Celsius, his blood pressure (BP) was 95/62 mmHg, his pulse (P) was 99 beats/min and his respiratory Rate (RR) was 26 breaths/min. His oxygen saturation (SaO2) was 100%.

Abdominal examination revealed a palpable liver and spleen with localized epigastric tenderness.

* Corresponding author.
E-mail addresses: bassem.abouhussein@gmail.com (B.M.A. Hussein), alialani2002@yahoo.com (A.M.A. Ani), ofal-mayoof@dha.gov.ae (O. Al-Mayoofi), paramir24@gmail.com (M. Mehraj), afrahjoher@gmail.com (A.A. Joher), juaangbon@hotmail.com (J.A. Bonilla), mazrouei@bluewin.ch (A.S. Al-Mazrouei), drfbadri@yahoo.com (F.M. Badri).

http://dx.doi.org/10.1016/j.ijscr.2016.10.017

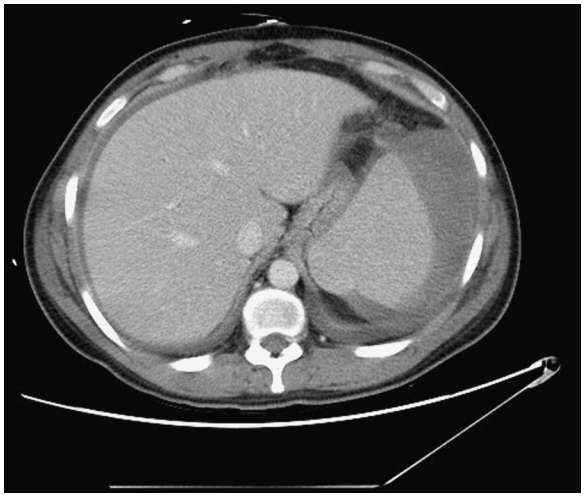

Fig. 1. Computed topography (CT) scan of the abdomen and pelvis with IV contrast showing haemoperitoneum with blood clot adjacent to spleen suggestive of rupture.

Laboratory investigations:

White Blood Cell (WBC) = 6900/uL, hemoglobin (Hgb) = 12.5 g/dL,

Creatinine (Cr) = 1 mg/dL, Prcoalcitonin (PCT) = 36 ng/ml and Lactic acid = 7.7 mmol/L

Blood film showed vivax malaria (ring forms).

After adequate resuscitation, the patient was stabilized with vital signs: BP = 118/81 mmHg and P = 84 beats/min and was shifted to radiology department where a computed topography (CT) scan of the abdomen and pelvis with IV contrast was done and showed:

- Haemoperitoneum with blood clot adjacent to spleen suggestive of rupture with no active extravasation (Fig. 1).
- Bilateral perinephric fluid and mild to moderate pleural effusion.

The patient remained stable so he was admitted to the surgical intensive care unit (ICU) in a trial of conservative treatment. On the second day, the patient complained of increased pain severity in the left upper quadrant; he become unstable with a BP = 75/41 mmHg and P = 110beats/min with sudden drop of hemoglobin (Hgb = 7.8 g/dL), so he was transfused by packed red blood cells, fresh frozen plasma and platelets in addition to intravenous fluid resuscitation and was shifted to the operating theatre for exploration. A pneumococcal vaccine was administered before shifting to the theatre.

On the operating table, the patient developed a cardiopulmonary arrest so cardiopulmonary resuscitation was done and the patient picked up. Exploratory laparotomy showed a large amount of blood (1.5 liters) and a grossly enlarged spleen with a ruptured large subcapsular hematoma (Fig. 2). The hemoperitoneum was cleared and splenectomy was done. No other pathology was found.

The patient was further managed in the surgical ICU. Acute renal failure was noticed and required haemodialysis. Blood film showed no malaria parasite after 3 days of anti-malarial treatment (Quinine and Clindamycin) and continued for seven days where he recovered with gradual improvement in his renal and hepatic function with gradual increase of hemoglobin over the following days. He was then extubated and transferred to the general ward where he received meningococcal & H. influenza vaccine prior to discharge and is followed up in the infectious disease outpatient clinic to continue anti-malarial treatment plan.

Histopathologic examination of the spleen showed splenomegaly (its weight is 492 g) with hemorrhage/hematoma and capsular rupture. Microscopically hemorrhage was confirmed together with slight red pulp expansion (Fig. 3) and foci of brown/black pigment in macrophages (Fig. 4). Giemsa stain was performed but plasmodia were not seen.

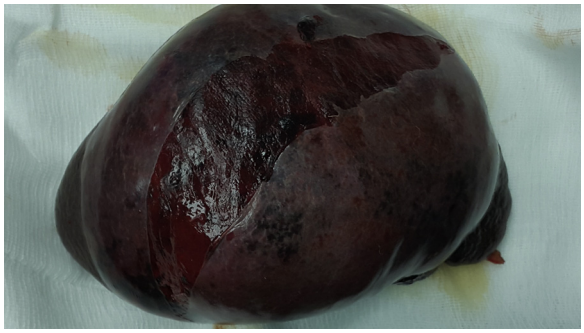

Fig. 2. Intraoperative finding of a grossly enlarged spleen with a ruptured large subcapsular hematoma.

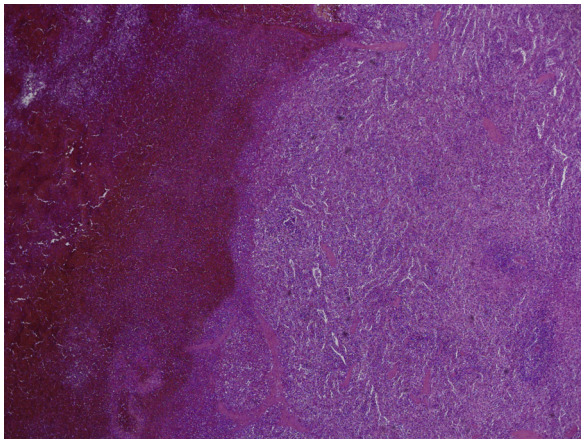

Fig. 3. Histopathology: Spleen showed hematoma/hemorrhage (left) with slight red pulp expansion (right). H&E (Taken by Dr Manal Abdulrahim).

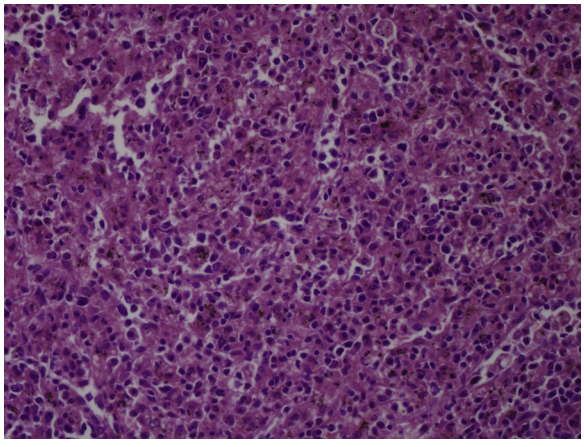

Fig. 4. Histopathology: High power view showing brown/black pigment in macrophages in the splenic tissue. H&E. (Taken by Dr Manal Abdulrahim).

3. Discussion

Blunt abdominal trauma is the most common cause of splenic rupture. Non-traumatic splenic rupture is rare and occurs in a diseased spleen. Among the non-traumatic causes, hematological

malignancy is the leading cause of splenic rupture; others being infection, vascular, genetic or hematological disorders [1].

Malaria is the most frequent tropical infectious cause of spontaneous splenic rupture [2]. Most of the cases of spontaneous splenic rupture in malaria occur during acute infection and are associated with *Plasmodium vivax*, although there have been rare cases associated with other

Plasmodium species [3]. Review articles have reported only 22 malaria cases with spontaneous splenic rupture in the English language literature since 1960. The predominant plasmodium species in these cases were *P.vivax* (15 patients) [4].

Although spleen enlargement is a very common feature of malaria, the incidence of spontaneous splenic rupture is not well known; it ranges from 0% to 2% [5]. The exact mechanism of spleen rupture is poorly defined. However, two mechanisms have been implicated in the process. The first mechanism happens in acute malaria and is a result of activated lymphatic tissue of the organ and marked stasis in the splenic sinuses caused by deformed parasite containing erythrocytes with altered surface characteristics. The second mechanism happens in acute malaria and is a result of compression of the spleen by abdominal musculature during physiological activities such as sneezing, coughing, defecation, and sitting up or turning in bed [6,3].

When examining a non-trauma patient with abdominal pain in the left upper quadrant, the doctor has to bear the diagnosis of a non-traumatic rupture of spleen in mind. Typically, patients present with hypovolemic shock and signs of peritonitis [7]. Diagnosis should always be suspected in a patient from an endemic region who presents with rapid onset of hypotension and severe left hypochondrial pain [8].

Abdominal ultrasonography, computerized tomography (CT) scan, and arteriography can provide confirmation of the diagnosis in a hemodynamically stable patient [9]. CT has replaced angiography as the preferred diagnostic tool and can clearly show splenic hematoma or rupture. CT has a sensitivity, and specificity of at least 95% in detecting splenic injury [10].

CT scan is useful in diagnosis and monitoring a patient in whom conservative management of splenic rupture is considered [11].

Historically, the treatment of choice for all kinds of splenic rupture used to be splenectomy; however, nowadays it remains subject to debate. Transcatheter splenic artery embolization has a major role in the management of traumatic splenic injuries [12] and can be used in selective cases of non-traumatic rupture. The therapy of choice can vary between patients depending on grade of splenic rupture, hemodynamic instability, availability of endovascular treatment and preference of the treating physician. Treatment should be focused on preserving splenic tissue [13]. Splenectomy remains the treatment of choice in patients with a hemoperitoneum and persistent instability [14].

In our case, our patient was started with conservative management as he had stable vital signs. One day later, he developed hypotension and his hemoglobin dropped, he was then resuscitated but he did not respond, which prompted us to take him for surgical intervention immediately.

Spontaneous splenic rupture is a fatal complication of malaria, which requires critical decision making in its management. Most malarial splenic ruptures do heal; hence, attempts at splenic salvage should be the aim in their management. [15].

4. Conclusion

Despite the fact that spontaneous splenic rupture is an extremely rare condition; a high index of suspicion should be kept in mind while facing patients with malignancy, infection, vascular, or genetic disorders who present with abdominal pain or signs of hypovolemic shock. Early diagnosis and treatment is crucial in decreasing morbidity and mortality. Treatment of spontaneous splenic rupture can be conservative or surgical depending on the general condition of the patient.

Conflict of interest disclosure

The authors declare that no conflict of interest exists.

Statement of informed consent

Informed consent was obtained from the patient included in the study.

Author contribution

Bassem Abou Hussein: Author.
Ali Al Ani: Co-Author.
Omar Al-Mayoofi: Co-Author.
Mahpara Mehraj: Co-Author.
Afra A. Joher Co-Author.
Juanita A. Bonilla: Discussion the case.
Alya Al-Mazroui: Contributor.
Faisal Al-badri: Contributor.

Ethical approval

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Approval was given by consenting the patient.

Acknowlegments

Dr Manal Mohammmad Abdulrazzaq Abdulrahim, Consultant Histopathologist- Dubai Health Authority for her kind help and assistance in pathology reports and photos.

References

[1] N. Gupta, P. Lal, A. Vindal, N.S. Hadke, N. Khurana, Spontaneous rupture of malarial spleen presenting as hemoperitoneum: a case report, Vector Borne Dis. 47 (2010) 119–120.
[2] B.C. Jiménez, M. Navarro, H. Huerga, R. López-Vélez, Spontaneous splenic rupture due to *Plasmodium vivax* in a traveler: case report and review, J. Travel Med. 14 (2007) 188–191.
[3] M.F. Ozsoy, O. Oncul, Z. Pekkafali, A. Pahsa, O.S. Yenen, Splenic complications in malaria: report of two cases from Turkey, J. Med. Microbiol. 53 (2004) 1255–1258.
[4] M.I. Patel, Spontaneous rupture of a malarial spleen, Med. J. Aust. 159 (11–12) (1993) 836–837.
[5] F.M. Jacobs, et al., Spontaneous rupture of the spleen during malaria: a conservative treatment approach may Be appropriate, Clin. Infect. Dis. 40 (12) (2005) 1858–1859.
[6] P. Bonnard, J.B. Guiard-Schmid, M. Develoux, W. Rozenbaum, G. Pialoux, Splenic infarction during acute malaria, Trans. R. Soc. Trop. Med. Hyg. 99 (2005) 82–86.
[7] E. Wehbe, S. Raffi, D. Osborne, Spontaneous splenic rupture precipitated by cough: a case report and a review of the literature, Scan. J. Gastroenterol. 43 (2008) 634–637.
[8] Dayal. Madhukar, Non-Traumatic rupture of malarial spleen, Trop. Gastroenterol. 34 (2) (2013) 191–193.
[9] S.R. Schackford, M. Molin, Management of splenic injuries, Surg. Clin. N. Am. 70 (1990) 595–620.
[10] R.B. Jeffrey, F.C. Laing, M.P. Federle, Goodman PC. computed tomography of trauma, Radiology 141 (3) (1981) 729–732.

[11] M. Correia, et al., Spontaneous rupture of a malarial spleen – a case report and review of literature, The Internet J. Sur. 23 (2) (2009).
[12] Antony Raikhlin, Mark Otto Baerlocher, R. Murray Asch, Andy Myers, Imaging and transcatheter arterial embolization for traumatic splenic injuries:review of the literature, Can. J. Surg. 51 (December (6)) (2008) 464–472.
[13] Michiel Leijnen, Wobbe O. de Steur, Wouter Brekelmans, Andy da Costa, Non-traumatic rupture of the spleen: an atypical presentation of the acute abdomen, Abdom. Surg. (2011).
[14] C.T. Hamel, J. Blum, F. Harder, T. Kocher, Nonoperative treatment of splenic rupture in malaria tropica: a review of literature and case report, Acta Trop. 82 (1) (2002) 1–5.
[15] L.F. Tauro, R. Maroli, C.R. D'Souza, B.R. Hegde, S.R. Shetty, D. Shenoy, Spontaneous rupture of the malarial spleen, Saudi J. Gastroenterol. 13 (2007) 163–167.