



# Atraumatic Splenic Rupture Secondary to the Use of Anticonvulsants: A Case Report from Libya

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## Case Report

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## Abstract

Atraumatic/spontaneous splenic rupture (ASR/SSR), is defined as its rupture without an obvious cause. In addition to trauma, splenic rupture can be caused by medical conditions like infections, malignancies, the use of some medications, after colonoscopy among others. It is a rare condition accounting to about 0.1- 0.5% of the admissions. Our case had ASR but did not show any symptoms or signs of peritoneal irritation or shock that make it has some peculiarities.

**Keywords:** Atraumatic Splenic Rupture; Anticonvulsant

## Introduction

Atraumatic/spontaneous splenic rupture (ASR/SSR), is defined as its rupture without an obvious cause. In addition to trauma, splenic rupture can be caused by medical conditions like infections, malignancies, the use of some medications, after colonoscopy among others [1-3]. Here, we report a case of ASR arising in a patient who is using anti-epileptic drugs since long time that can be the indirect cause of this problem.

## Case Report

A 34 years old Libyan, was taken to the ER in a private clinic with a history of high fever (40°C) for seven days associated with headache and unwell being. He denied other symptoms like abdominal or chest pain, cough, urine symptoms, blurred vision, fainting or palpitations. He denied skin rash sore throat or recent trauma.

Many years ago, he had a head trauma during a car accident that was complicated by secondary epilepsy, being

well controlled by Carbamazepine and phenobarbital, not having any fits since nine months back. On examination, the patient was conscious, febrile (38.8 C), PR:101/min, having a good volume pulse, Sat O<sub>2</sub>: 97% on room air, blood pressure of 126/66. He was pale, but not jaundiced, no enlarged lymph nodes, and no skin rash.

His cardiac and chest examination was unremarkable. His abdominal examination showed no tenderness or guarding, his liver and spleen were not palpable and his bowel sounds were audible. His CNS examination did not show any meningeal signs or focal neurological deficit; A laboratory work up was done (Table 1):

His hemoglobin was dropping quickly so an abdominal ultrasound was requested and revealed fatty liver stage III, perisplenic fluid and free dense fluid in the peritoneal cavity suggestive of blood. A CT scan done in the same day that revealed mild hepatomegaly and splenomegaly, perisplenic free fluid and a moderate amount of dense fluid in the pelvic cavity, features that suggest splenic rupture. A surgical

opinion was requested, that advised for close observation. We made ready three units of blood for possible transfusion but were not needed.

Upon admission, his complete blood count showed thrombocytopenia ( $103000/\text{mm}^3$ ), and in spite of a normal leukocyte count his blood film showed absolute neutrophilia (81%) and 15% of lymphocytes with heavy toxic granules and a lot of large platelets. A search for malaria parasites was negative and there were no atypical lymphocytes. The CRP was high (198mg %) and a viral screen for HBV, HCV and HIV was negative. His liver transaminases were raised but his bilirubin was normal. All the other routine investigations were within the normal range except for a high triglyceride level.

Serologic investigations of infectious mononucleosis (IM) were as follows: EBV VCA IgG antibodies 180.7 U/ml (non-reactive <20), EBV VCA IgM antibodies: 9.1 U/ml (non-reactive <20), and EBV ZEBRA IgM: 5U/ml (non-reactive <20U/ml).

Based on the history, laboratory and radiologic findings, the diagnosis of splenic rupture was done and because of the high fever and the high CRP, that can be explained by secondary peritonitis, the patient was started on meropenem. Doxycycline was also added empirically because rickettsial infections are endemic in Libya during these summer months and it can be complicated by splenic rupture.

The patient started to improve in the 2<sup>nd</sup>-3<sup>rd</sup> day with less fever and headache and better general condition and his laboratory workup became better (Table 1). A repeat CT scan 3 days later did not show any worsening of the bleeding compared to the previous one. The patient was discharged after four days of admission, to complete his antibiotic treatment at home. Ten days later, he came for follow up reporting no symptoms and having CBC almost normal (Table 1). His abdominal ultrasound was repeated and revealed mild hepatosplenomegally but no more free fluid in the splenic area or in the peritoneum.

Date	Hb	Platelets	WBC	CRP	INR	ALT	AST	Sodium
08/10/23	10,6	103000	6500	198	1,03	121,27	146,85	127
08/10/23 (9 hours later)	10,2	89000	4900	-	-	-	-	-
09/10/23	9,9	128000	6100	-	-	-	-	-
10/10/23	-	-	-	112,74	-	-	-	-
11/11/23	-	-	-	65,48	-	-	-	-
21-10-2023	13,7	136000	4780	5,32	-	40	36	-

**Table 1:** Laboratory findings of the patient during admission and discharge.

## Discussion

Splenic rupture can be secondary to some conditions like infectious mononucleosis (IM), trauma, colonoscopy, in malignant diseases like lymphoma or after the use of some medications like anticoagulants [1,2,4].

ASR is rare being reported in 0.1-0.5% of the cases [5]. It is usually associated with some symptoms like left upper quadrant abdominal pain that radiates to the left shoulder (Kehr's sign) that can be caused by peritoneal irritation, or symptoms of hypotension like dizziness, faintness, confusion and blurred vision, caused by the bleeding [6].

What makes our case different from the cases reported in the literature is the presence of fever and the absence of local abdominal signs of ruptured spleen together with absence of important signs of bleeding, this is in contrast to reports in

the literature [6] that mentioned these features in such cases.

Our case is also different because of the possible association of ASR with the use of anti-epileptic drugs which has never been mentioned in the literature. In this case, fever could be caused by secondary bacterial infection of the blood in the peritoneal cavity but even so the patient did show any signs of peritoneal irritation. This is quite logical since the patients fever disappeared after 48 hours of ABT use and his CRP declined very quickly.

The abdominal ultrasound was mainly requested, because of the rapid drop in his haemoglobin, which revealed the possibility of splenic rupture that was confirmed by the CT scan report. As we have mentioned the cause of this incident, could be a consequence of hepatosplenomegally and abnormal function of the liver that can be caused by the use of anti-epileptics [7]. The decline of his platelet count

could be caused by consumption in the clotted blood around the spleen and in the peritoneal cavity.

### Conclusion

ASR in this case could be due to a mild splenomegally induced by chronic use of anti-epileptic drugs. To the best of our knowledge, this is the 1st case in the medical literature that documented the association of carbamazepine/phenobarbital use with ASR.

### Consent Form

A verbal consent was taken from the patient and the paper was authorized from the ethical committee of the clinic.

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