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Non Traumatic Splenic Rupture in A Case of Dengue Fever with Hepatitis, Pancreatitis and Associated With Acute Kidney Injury a Case Report.

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I. Introduction

The spectrum of disease in dengue infection caused by four serotypes (DEN-1 to DEN-4) range from a subclinical or mild illness to a severe form of hemorrhagic fever which may prove fatal [1]. The World Health Organization (WHO) has suggested a revised classification for the illness as dengue (± warning signs) and severe dengue [1]. However, the earlier WHO classification of dengue fever (DF), dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS) is still widely followed in clinical practice [1]. Prior to 1970 only nine countries had experienced dengue hemorrhagic fever [2]. Today, dengue has spread to over 100 countries; this number includes those countries that were free from the disease 20 years ago [2]. Acute abdominal pain is one of the atypical presentations of dengue fever, and spontaneous splenic rupture is a rare complication of dengue fever. DHF and DSS are prevalent among all age groups in northern India and among children in southern India [2]. To date, 11 cases of spontaneous splenic rupture in dengue have been reported in the medical literature [3-12]. We report a non-fatal case of spontaneous splenic rupture in an adult patient with dengue fever with no cutaneous or mucosal bleeding tendencies but with features of hepatits, pancreatitis and acute kidney injury and anaemia due to internal bleed.

II. Case Report

A 25 year old male resident of Jivati village, Chandrapur district of Maharashtra presented with complaints of high grade fever since 10 days breathlessness since 8 days and epigastric pain since 4 days and history of malena with no addictions or co-morbidities. He was investigated in a peripheral hospital and was found to be having dengue IgG positive with viral hepatitis with Acute Kidney injury. On clinical examination his vitals were normal and epigastric tenderness being the only finding on systemic examination. His Laboratory data of the patient in emergency department haemoglobin was 5.4 total leucocyte count was 17,400/platelets 107000/, inr was 1.66, liver function tests-s.bilirubin-4.6,SGOT-750,SGPT-685. Serum creatinine was 1.8. His s.amylase was 132 and s.lipase was 941, s.electrolytes being normal.

Contrast enhanced computed tomography scan was performed which suggested sub-capsular splenic collection,moderate ascites and bilateral minimal basal pleural effusion **(fig 1)**. Ultrasound guided pigtail catheter was inserted which showed hemorrhagic aspirate with only RBC's and no growth on culture. Interventional radiologist opinion taken in view of continuous haemorrhagic drain and fall in haemoglobin they was advised for splenic artery embolization. splenic artery embolization done (fig. 2) His lab parameters improved with conservative management and duration of 12 days. Patient recovered from hepatitis,pancreatitis with normalization of serum creatinine and liver function. His pigtail catheter was removed and he was discharged in a vitally stable state.

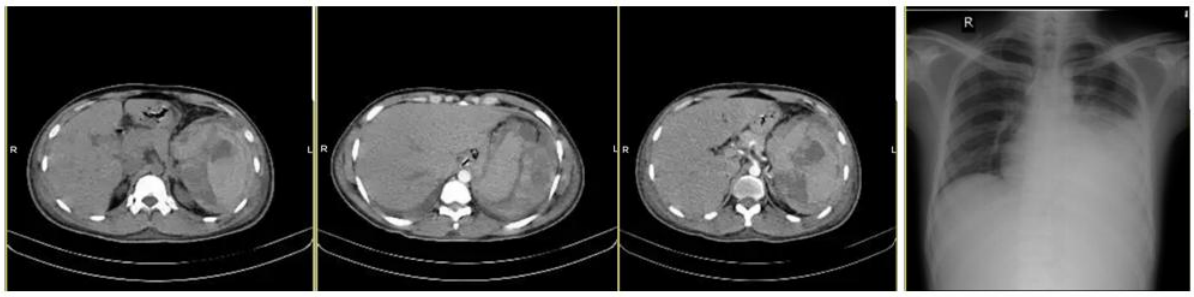
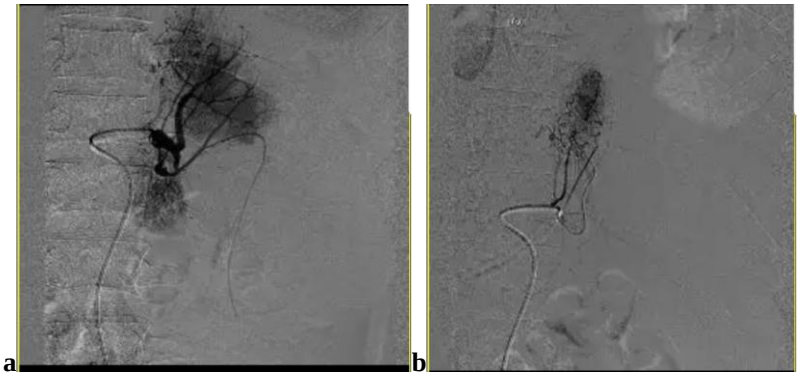


Fig 1 - Contrast enhanced computed tomography scan was performed which suggested sub-capsular splenic collection,moderate ascites and X Ray chest suggestive of left sided pleural effusion.



a **b**

fig 2- a) pre- embolization and **b)**post- embolization of splenic artery

Serial laboratory results of the patient during hospitalization

CBC investigations on Cell counter with PS

INVST. DATE	Hb%	Total RBC Count	HCT	MCHC	MCV	MCH	Total WBC Count	Total Platelet Count	Monocytes	Granulocytes	Lymphocytes	RDW	Eosinophils	Basophils
27/10/2019 07:48 pm	5.4	1.91	16.4	33.2	86	28.6	17400	1.07	03	70	25	13.1	02	00
29/10/2019 09:10 am	5.7	1.98	17	33.4	86	28.8	13000	0.75	03	70	25	12.7	02	00
31/10/2019 11:00 pm	10.3	3.57	31.4	32.7	88	28.8	20300	1.04	03	83	12	18	02	00
01/11/2019 12:02 pm	10.3	3.61	31.8	32.5	88	28.6	19600	1.02	06	80	12	18.4	02	00
01/11/2019 04:05 pm	10.4	3.72	32.2	32.4	87	28.1	23000	1.29	02	78	18	18.5	02	00
02/11/2019 08:14 pm	10	3.53	31.1	32.2	88	28.3	17400	1.69	04	74	18	18.1	04	00
03/11/2019 06:33 pm	10.5	3.81	33.1	31.6	87	27.4	15800	3.62	02	78	18	18.4	02	00
04/11/2019 07:58 pm	10.4	3.67	32.8	31.5	90	28.2	13700	4.23	03	75	20	18	02	00

INVST. DATE	Amylase
27/10/2019 07:15 pm	172
30/10/2019 07:06 am	273
02/11/2019 05:42 pm	444
04/11/2019 07:52 pm	115

INVST. DATE	Lipase
27/10/2019 07:15 pm	941
30/10/2019 07:06 am	1616
02/11/2019 06:58 pm	3196
04/11/2019 07:52 pm	283

KFT

INVST. DATE	Urea	Creatinine	Sodium (Na+)	Potassium (k+)
27/10/2019 07:15 pm	101	2.1	136	4.3
28/10/2019 06:28 am	99	2.1	140	3.7
29/10/2019 06:41 am	75	1.8	142	3.7
30/10/2019 07:06 am	50	1.3	141	3.6
31/10/2019 02:41 pm	41	1.2	144	4.3
02/11/2019 05:42 pm	27	0.7	140	3.4
08/11/2019 07:07 am	24	0.6	137	3.0
11/11/2019 01:57 pm	16	0.5	133	3.3

LFT

INVST. DATE	Alkaline Phosphatase	ALT(SGPT)	Total Protein	Albumin	Total Bilirubin	BC Bilirubin Conjugated	BU Bilirubin Unconjugated	Globulin (Calculated Parameter)	AST (SGOT)
27/10/2019 07:15 pm	55	750	4.3	1.8	2.8	2.3	0.5	2.5	
27/10/2019 08:03 pm									1692
29/10/2019 06:41 am	43	685	5.1	2.1	4.6	3.4	1.2	3.0	750
31/10/2019 02:41 pm	45	412	6.1	2.6	5.4	4.0	1.4	3.5	468
02/11/2019 05:42 pm	38	222	5.5	2.3	7.0	5.6	1.4	3.2	150
03/11/2019 04:40 pm	50	203	6.1	2.6	8.3	6.7	1.6	3.5	146
04/11/2019 07:52 pm	39	153	5.7	2.4	7.4	5.8	1.6	3.3	126

III. Discussion

Splenic rupture occurs secondary to abdominal trauma or due to non-traumatic causes [13,14]. Non-traumatic splenic rupture (NTSR) may be either pathologic or spontaneous. The term pathological splenic rupture is applied when it occurs in a diseased spleen (with abnormal histology) while spontaneous splenic rupture refers to one occurring in a histologically normal spleen [13]. NTSR can occur in infections, malignancies, and connective tissue disorders [13].

Infections associated with NTSR include infectious mononucleosis, malaria, typhoid, varicella, infective endocarditis, Q-fever, influenza, aspergillosis and dengue [13,14,15].

Splenic rupture associated with dengue can occur in both uncomplicated dengue fever (which lacks features of severe bleeding and plasma leak) and complicated dengue hemorrhagic fever. Though hypothesized to be due to a combination of coagulation factors and severe thrombocytopenia, the mechanism of splenic rupture in dengue is not clear [12]. Of the 11 dengue cases describing splenic rupture, 4 occurred in uncomplicated dengue fever and 7 occurred in DHF of which 8 (out of 11) patients survived. [3-12]. The survival observed in these cases should be attributed to timely diagnosis and management rather than the natural course of splenic rupture, which is expected to be poor if the diagnosis is missed. Unlike the situation in earlier reports, our case highlights the occurrence of splenic rupture in the viremic phase of dengue before the development of antibodies, as illustrated by NS1 dengue antigen detection with no dengue IgM antibodies in the admission sample.

Abdominal pain is the main presenting symptom of splenic rupture in dengue fever. Given that acalculous cholecystitis is a known complication of dengue fever presenting with abdominal pain, which is often treated conservatively, it is possible that clinicians may overlook splenic rupture except when it is specifically sought. Hypotension may be the only clinical clue indicating a possible splenic rupture in a patient with suspected dengue presenting with abdominal pain. This scenario can be complicated with the fact that hypotension is frequently associated with DHF, which may mislead the clinician. It may be prudent to consider that abdominal imaging focussing on splenic rupture is mandatory in a patient with suspected dengue fever presenting with abdominal pain and hypotension, irrespective of the presence of signs of plasma leak. Furthermore, patients presenting with abdominal pain in dengue should undergo careful evaluation since inadvertent surgery may prove fatal [17].

Infectious mononucleosis (IM) is the protean disease affecting the reticuloendothelial system leading to splenic rupture. First described in 1941 by King, the prevalence of splenic rupture in proven infectious mononucleosis is about 0.1 to 0.5% [15]. IM is associated with infiltration of lymphocytes in the splenic pulp, supporting trabeculae, blood vessels, and even the splenic capsule [16], which leads to fragmentation of the splenic architecture facilitating spontaneous rupture.

Splenic rupture is a well-known complication of malaria (more frequent in Plasmodium vivax than in Plasmodium falciparum infection), with 64 cases

reported by the year 1948 [16]. The mechanisms of splenic rupture in malaria include rapid hyperplasia and stretching of the splenic parenchyma and capsule along with occurrence of small infarctions and haemorrhages making the spleen more vulnerable for rupture [16]. Though reports of splenic rupture in typhoid date back to 1898, it is far less common today [18].

Pathologic splenic rupture can occur in malignancies which include haematological conditions such as

acute myeloid leukemia, chronic lymphoid leukemia and non-hodgkins lymphoma [13]. Splenic rupture can also occur due to splenic metastasis from choriocarcinoma, malignant melanoma, teratoma, carcinoma of lung, liver, stomach, rectum and urinary bladder [13]. Rheumatoid arthritis, systemic lupus erythematosis, polyarteritis nodosa and Wegener's granulomatosis are connective tissue disorders reported to be associated with splenic rupture [13].

Splenic rupture, either pathological or spontaneous, can be associated with a spectrum of causes. Splenic rupture is a rare, but potentially fatal complication of dengue fever which should be suspected when a patient presents with abdominal pain and hypotension. Dengue and malaria can present with hypotension and they are unique causes of splenic rupture, which may delay the diagnosis of this complication. Our case highlights the fact that splenic rupture in dengue can occur during the viremic phase of the illness before the development of dengue antibodies.

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