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Case Report (Pages: 18775-18780)

Abdominal Compartment Syndrome in an Infant with Severe Dengue: A Case Report

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Abstract

Background: Abdominal compartment syndrome can have devastating effects on abdominal visceral organs which eventually can progress to multi-organ dysfunction and death. Various medical and surgical conditions are known to progress to this complication. Abdominal compartment syndrome is now increasingly recognised as a complication of sepsis and Dengue shock syndrome. Life saving measures include reduction of intra-abdominal pressure and adequate support of dysfunctional organs.

Case report: We report the case of a 3-month-old infant with abdominal compartment syndrome due to severe dengue illness with multi organ failure with massive ascites. She had a dramatic recovery following abdominal decompression by therapeutic drainage of 300 ml of ascitic fluid.

Conclusion: The spectrum of abdominal compartment syndrome features may easily be thought to be part of capillary leak syndrome and the diagnosis of ACS could have been missed. Early recognition and aggressive treatment have been shown to significantly improve the outcome.

Key Words: Abdominal compartment syndrome, Abdominal paracentesis, Decreasing intra-abdominal pressure, Severe Dengue.

* Please cite this article as: Kumar KJ, Jagwani H, V G. M, Kumar H C K. Abdominal Compartment Syndrome in an Infant with Severe Dengue: A Case Report. Int J Ped Perspect 2024; 12 (04):18775-18780. DOI: **10.22038/ijp.2024.64274.4878**

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Received date: Feb.08,2022; Accepted date: Apr.21,2024

1- INTRODUCTION

Abdominal Compartment Syndrome (ACS) is Intra-Abdominal Hypertension (IAH) leading on to end-organ dysfunction (1). ACS results in impairment of perfusion to visceral organs in particular to kidneys, restriction of diaphragmatic movement leading to restrictive pulmonary disease and hemodynamic instability due to decreased cardiac preload. Excessive third spacing, fluid overload, and intra-abdominal bleed can contribute to intra-abdominal hypertension (1). The incidence rate of ACS has been reported between 0.6 and 9.8% in critically ill children (2). In a study by Ejike et al., 4.7% of children less than 50 kg on mechanical ventilation and urethral catheter had ACS. Mortality was 50% among those with ACS versus 8.2% without ACS (3). ACS increases the risk of mortality in critically ill children and results in 100% mortality if left untreated (2). Currently, ACS has been increasingly recognised in PICU settings (2). ACS occurs due to a wide variety of medical and surgical causes (2). In ACS, reducing intra-abdominal pressure and supporting dysfunctional organs should be the objectives of therapy (2). Medical management is initiated to achieve these objectives. If medical management fails, timely abdominal decompression is a must to reduce the risk of mortality (2). Percutaneous drainage of peritoneal fluid can prevent the progression of IAH to ACS altogether (2). Only a few case reports of ACS due to severe dengue have been documented (1, 4). We are reporting a 3-month-old infant with ACS due to severe dengue illness and Multi Organ Failure (MOD). She had massive ascites resulting in a tense abdomen, who recovered after draining 300 ml of peritoneal fluid.

2- CASE REPORT

A 3-month-old female infant was brought with a history of fever for 2 days and convulsions for 30 minutes. At admission, the child was actively convulsing and was treated with Injection Lorazepam and Fosphenytoin. There was no history of cough, urinary disturbances, rash, or refusal of feeds. On examination, the child was flushed; and had peripheral cyanosis and cold clammy extremities. Her vital parameters were as follows: Temperature 1010F, RR 42/minute, PR 160/minute, low volume, NIBP 71/47 mm of Hg, CFT > 5 seconds, and SPO2 97% at room air. Her GCS was 9/15 and abdomen distension with hepatomegaly of 6 cm was noted. In view of compensatory shock, the child was given 2 boluses of Ringer lactate 10 ml/kg each and started on Injection Ceftriaxone and Vancomycin. Investigations are depicted in Table 1. Initial chest x-ray was normal and ultrasound showed hepatomegaly, ascites, gallbladder wall edema, and minimal pleural effusion.

An initial diagnosis of dengue shock /Sepsis shock with meningitis was considered. In the light of thrombocytopenia and deranged coagulation profile, the child was transfused with 2 units of platelets and FFP each. As she continued to have cold peripheries with low pulse volume and prolonged CFT, dopamine infusion was started. However, there was no improvement, and hence adrenaline infusion was added. After 24 hours, her platelet count was 8000/mm3 PT/INR 22.6/2.9. Hence, 2 more units of platelets and FFP were transfused. On the 3rd day of admission, her peripheries were warm, blood pressure improved, and hence inotropes were tapered and stopped. On the 4th day, she developed respiratory distress and increased abdominal distension. Her chest x-ray revealed ARDS and the child was put on non-invasive ventilation (nasal CPAP) along with Lasix infusion. On the 6th day, abdominal girth further increased by 2 cm, and her liver enzymes were as follows: SGOT 1445

U/L, SGPT 1438 U/L, and serum albumin 2.8 g/dl. Sonography revealed significantly altered liver texture with periportal cuffing along with gross ascites. The child was started on albumin infusion 1 gm/kg over 6 hours and N-Acetyl cysteine infusion for 2 days. Anticonvulsants were changed from Injection of Fosphenytoin to Levetiracetam. She had serial chest x-rays and abdominal ultrasounds to monitor the progress which revealed increasing ascites. On the 7th day of admission, the abdomen was tense requiring medical decompression measures like continuous gastric aspiration, placement of flatus tube, and diuretics (Spironolactone). Administration of FFP and albumin bolstered the treatment. However, these measures were unsuccessful, and her condition worsened leading to Abdominal Compartment Syndrome (ACS). On the 10th day of admission, she was in respiratory embarrassment with a tense shining abdomen. Budd Chiari syndrome was ruled out by a hepatic vein Doppler study. In view of the tense abdomen, transfusion of platelets [platelet count 29000/mm3] and albumin infusion were done followed by abdominal paracentesis, done by inserting intravenous cannula at the right iliac fossa under aseptic precautions. Around 300 ml of ascitic fluid was drained slowly while monitoring the vitals. The rate of drainage was controlled to avoid rapid decompression. Following abdominal paracentesis, there was a dramatic turnaround of the clinical condition. Her abdominal distension improved over the next 3 days and the child was discharged after a week.

3- DISCUSSION

Our 3-months-old infant with severe dengue in shock with MOD (encephalitis, ARDS, hepatic failure, myocarditis, bleeding tendencies, and sepsis) was managed according to WHO fluid protocol, noninvasive ventilator support, hepatic support, and inotropes. She developed a tense abdomen due to ascites on the seventh day leading to respiratory embarrassment. She was diagnosed to be suffering from abdominal compartment syndrome (ACS). After draining 300 ml of ascitic fluid, her distension decreased over 3 days and she was discharged. Timely relieving the intra-abdominal pressure led to dramatic improvement in the clinical condition. ACS is defined as abdominal distention with intra-abdominal pressure (IAP) > 15 mm Hg, accompanied by at least two of the following: oliguria or anuria; respiratory decompensation; hypotension or shock; metabolic acidosis (4). Based on the pathophysiology of elevated IAP and the predisposing factors, ACS can be grouped into four major categories (1). Firstly, ACS due to decreased abdominal wall compliance, e.g., tight closure of the abdomen following abdominal surgery (2). Secondly, ACS due to increased intraluminal contents, e.g. fluid in the intestines as seen in Hirschsprung's disease (3). Thirdly, ACS due to increased intra-abdominal contents, e.g., intra-abdominal space-occupying lesions such as intraperitoneal fluid and intra-abdominal haemorrhages (4). Finally, ACS due to capillary leak syndrome/fluid resuscitation, e.g., Aggressive fluid resuscitation, especially with crystalloid solutions in the critically ill patients or diseases associated with capillary leak syndrome (2). The case which we have presented here belongs to the fourth category.

Out of 1762 patients admitted over 5 years, ten patients (0.6%) had a total of 15 episodes of ACS in a study by Raphael et al. (5). Kamath et al. reported 3 children with ACS out of 109 severe dengue patients admitted in PICU and it was attributed to refractory shock. Two of them recovered following the release of intra-abdominal pressure by peritoneal dialysis while the third patient died (4).

Table-1: Investigations

Investigations	1st day	2nd day	3th day	4th day	6th day	8th day	9th day	11th day	14th day
Hemoglobin [g/dl]	8.9	6.6	7.1	6.2	9.5	7.8	10.3	9.2	9.1
PCV (%)	27	19		8100	28	24	30.5	29.5	29
TLC [cells/mm^3]	8730	5800						13500	14460
Platelet count [cells/mm^3]	9000	8000	53000	34000	37000	32000	29000	56000	95000
CRP [U/L]	4.1			48					
Serum bilirubin [mg/dl]/Direct	0.15					3.2/1.	3.7/2		
SGOT(U/L)	243				1443	604	377	182	
SGPT(U/L)	54				1438	907	919	542	
PT/INR	14.2/1.7	22.6/2.9	14.2/3.2	27/3.4	21/2.82			29/3.3	14/2.2
APTT	102/3.8	103/3.08	79/2.39	74/2.6	54/1.8			78/2.7	44/1.36
Serum Albumin [gm/dl]	2.8				2.89	2.91	3.02		
ABG P^H P_{CO2} P_{O2} BE-	7.28 26 56 -12	7.29 19 106 -16		7.4 37 116 -1					

Dengue NS1 antigen test by ELISA positive.

Throughout the course serial Kidney function tests, blood sugar and serum calcium levels were monitored.

On the 4^{th} day of admission:-LDH 4015 U/L [Normal 230-460U/L], Troponin-T=0.195ng/ml [Normal 0.0001-0.349], CK-MB=14.39 [Normal 1-7ng/ml].

Ascitic fluid analysis revealed 40 cells/mm^3, most of them were lymphocytes, occasional neutrophils, protein 1.82 gm/dl, sugar 100 g/dl, SAAG=1.1 and LDH=309 U/L.

d- Dimer test 6.39 µg/mL [Normal < 0.5 µg/mL], Lactate 60 U/L [Normal 4.5-20].

Echocardiography normal and Blood culture sterile

PCV (Packed cell volume), TLC (Total leucocyte count), CRP (C-reactive protein) ,SGOT (serum glutamic-oxaloacetic transaminase), SGPT (Serum Glutamic Pyruvic Transaminase), PT (prothrombin time), APTT (Activated Partial Thromboplastin Time), ABG (Arterial blood gases), LDH (Lactate dehydrogenase), Creatine kinase-MB (CK-MB) , SAAG (Serum Ascites Albumin Gradient)

Gala et al. reported the case of a 14-year-old child with dengue shock syndrome who required aggressive fluid resuscitation, developed abdominal compartment syndrome, and responded well to abdominal paracentesis (6). Ghosh et al. reported ACS in a patient of DHF due to spontaneous rectus sheath haematoma causing external compression (7). A study in Pakistan reported that out of 875 dengue patients, abdominal compartment syndrome was seen in 2 patients (0.23%) (8).

ACS has been increasingly recognized following sepsis and dengue shock syndrome (6). In dengue shock syndrome,

fluid leakage from the intravascular compartment particularly after massive fluid resuscitation, leads to ACS due to fluid accumulation in serous cavities (6). Shock and oliguria can be further exacerbated by ACS leading to more fluid administration and increase in IAP (9). ACS can have deleterious effects on various organ systems like decreased cardiac contractility, increased afterload, increased ventilation requirement, and decreased GI perfusion and oliguria (6). Relieving high intra-abdominal pressure leads to dramatic improvement in clinical conditions (6). The intravesical method is the gold standard for measuring IAP (2). Even though we did not measure intra-abdominal pressure, tense abdomen with gross ascites and associated symptoms like severe shock, liver cell failure, ventilatory requirements and dramatic improvement after abdominal paracentesis draining suggested the diagnosis of ACS.

IAH and ACS can cause significant morbidity and mortality (2). Early recognition and treatments have shown to significantly improve morbidity and mortality (2). ACS due to dengue has been underrecognized and infrequently reported (9) Therefore, monitoring of IAP is necessary in dengue cases who are receiving more than 30 ml/kg in the first 2–3 hours; it can definitely help earlier recognition of IAP (9). All PICU children should undergo early screening for ACS (2). After percutaneous drainage of peritoneal fluid, resolution of symptoms was noted in all patients, but with variations, with respect to clinical parameters and measurements (2). Out of 10 PICU patients with ACS, there was a decrease in IAP and ventilatory parameters and an increase in urine output rapidly after PD in 5 patients and decompressive laparotomy in 2 patients (2). Management includes treatment of fluid overload with diuretics or renal replacement, as well as gastrointestinal decompression through nasogastric tube and flatus tube (1). Refractory ACS caused by ascites requires careful ultrasound guided abdominal paracentesis. Thrombocytopenia and coagulation disorders must be corrected prior to the procedure (10). Persistent increased abdominal pressure will require peritoneal drainage (1). Our child did not respond to diuretics or nasogastric compression but recovered after ascitic fluid drainage. When medical and less invasive therapies fail to respond, surgical decompressive laparotomy with open abdomen management is the treatment of choice for ACS (2).

4- CONCLUSION

In our case (severe dengue with MOD with gross ascites), the spectrum of features may easily be thought to be part of critically unstable DSS associated with capillary leak syndrome and the diagnosis of ACS could have been missed. Early recognition and aggressive treatment have been shown to significantly improve the outcome. All PICU children should undergo screening for early detection of ACS.

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