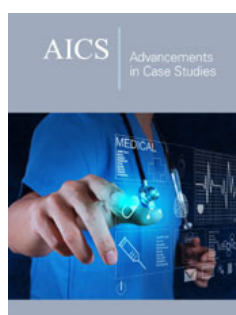


# Abdominal Compartment Syndrome in Child with Acute Viral Hepatitis B: Case Report

Yevtushenko V\*, Kramarov S and Markov A

O Bogomolets National medical university, Ukraine

ISSN: 2639-0531



**\*Corresponding author:** Yevtushenko V, O Bogomolets National medical university, Ukraine

**Submission:**  August 24, 2019

**Published:**  September 11, 2019

Volume 2 - Issue 1

**How to cite this article:** Yevtushenko V\*, Kramarov S, Markov A. Abdominal Compartment Syndrome in Child with Acute Viral Hepatitis B: Case Report. Adv Case Stud.2(1). AICS.000528.2019.

**Copyright@** Yevtushenko V, This article is distributed under the terms of the Creative Commons Attribution 4.0 International License, which permits unrestricted use and redistribution provided that the original author and source are credited.

## Abstract

**Background:** Renal involvement in idiopathic hyper-eosinophilic syndrome is uncommon. The mechanism of kidney damage can be explained as occurring via two distinct pathways: thromboembolic ischemic changes secondary to endocardial disruption mediated by eosinophilic cytotoxicity to the myocardium and direct eosinophilic cytotoxic effect to the kidney.

**Case presentation:** We present a case of a neonate who admitted to our hospital with severe form of acute hepatitis B. During acute period of illness, he was diagnosed with abdominal hypertension and abdominal compartment syndrome. This complication associated with progression of multiorgan failure. Conservative treatment was effective to achieve full recovery.

**Conclusion:** Course of acute viral hepatitis B in children patients may associated with risk of the intra-abdominal hypertension and the abdominal compression syndrome development and requires of abdominal pressure monitoring.

## Introduction

Despite the availability of an effective vaccine, Hepatitis B virus (HBV) infection remains a global public health problem and belongs to the most common cause of acute viral hepatitis. HBV infection prevalence estimates at 3.61% worldwide with highest endemicity in countries of the African region (total 8.83%). It is estimated that globally, about 248 million individuals were HBsAg positive [1]. More often acute hepatitis B presents as a mild or asymptomatic self-limited illness, but also may associated with devastating course with severe liver failure and extrahepatic complications [2].

Abdominal compartment syndrome (ACS) is defined as a sustained intra-abdominal pressure (IAP) associated with new organ dysfunction/failure. ACS and/or intra-abdominal hypertension (IAH) may occur after intra-abdominal events such as surgery, trauma and peritonitis. Its development leads to prolongation of treatment in the ICU and increases the risk of fatal outcome. It is believed that among children who receive therapy in intensive care units, the frequency of ACS fluctuates at the level of 0.6-4.7%, while the mortality rate reaches 40-60% [3,4]. Currently, diseases with a high risk of intra-abdominal hypertension (IAH) and abdominal compartment include trauma, intestinal obstruction, necrotic enterocolitis, abdominal wall defects, diaphragmatic hernia and septic shock with massive intravenous infusion [5]. Also, there are published cases of CAP due to some infection diseases [6,7]. Acute viral hepatitis is associated with factors offers elevation of intraabdominal pressure; however, we didn't find among available literature any cases of ACS associated with acute viral hepatitis.

## Case Description

An 8-month-old male patient presented to our hospital with 3 days of progressive jaundice and weakness. Parents did not report similar symptoms in the past. Child was not vaccinated because of the parents' refusal. During the hospitalization, the patient had jaundice; was conscious; had moderately swollen abdomen; exhibited minor limbs swelling; and had sufficient diuresis (3.91ml/kg/h). Arterial pressure showed of 91/61 mmHg, heart rate-133/min, respiratory rate-28/min, saturation-98%, and body temperature of 37.4 °C. Initial laboratory assessment revealed elevation of total bilirubin at 186.4mkmol/l (direct -134.2mkmol/l), ALT of 1204 IU/l, serum lactate of 4.2mmol/l, intestinal and liver fatty-acid binding protein (I-FABP, L-FABP) of 4.39ng/ml and 255.09ng/ml respectively. Creatinine level, base excess and blood gases was within normal range. On the second day of hospitalization,

small amount of a free fluid in abdominal cavity, gall bladder wall edema, and hepato-splenomegaly were observed. On the third day in the intensive care unit, the patient presented the abdominal bloating, steady decline in the level of consciousness, small moist rales in the lungs, haemorrhagic syndrome (bleedings from the injection sites and haemorrhagic stasis in the stomach), and the diuresis level of 5.4ml/kg/hour. The results of the laboratory examination revealed the progression of liver failure: the increase of the bilirubin level up to 229mkmol/l (direct-118mkmol/l), prothrombin time to 112s (INR of 10.27), ALT of 1204IU/l, decline in protein level to 54g/l, lactate increase to 7.2mmol/l, I-FABP of 28.35ng/ml, L-FABP of 773.05 ng/ml. Due to a steady decline of consciousness level, the patient was transferred on mechanical ventilation.

After the increase of the abdomen volume and the abdominal wall rigidity were observed, the evaluation of intra-abdominal pressure was recommended. Result showed increased pressure to 16mmHg. During adjuvant therapy, maintaining a negative water balance, limiting the volume of enteral nutrition and the usage of prokinetic agents were advised. Despite the intensive care, the state of the patient continued to worsen: on the fourth day, the decline of consciousness increased to coma 2-3; during ophthalmoscopy the swelling of the optic nerve was observed; depressed intestinal peristalsis was observed as well; and delay of bowel movement. From the seventh day after the hospitalization, the intensification of edematous-ascitic syndrome and the impaired consciousness were observed; the diuresis decreased relatively (minimal level to 1.97ml/kg/day); the creatinine level rises from 35.0 to 52.4mmol/l. Intra-abdominal pressure ranged between 12-15mmHg. Since the sixteenth day after hospitalization, a patient's condition started improving: the abdominal strain decreased; peristalsis improved. Since twenty-first day after hospitalization, the consciousness level recovered, and the intra-abdominal pressure became normal again. Laboratory findings at that time showed tendency to normalization of liver tests: ALT of 140 IU/l, total bilirubin of 92.1mkmol/l, serum protein of 62g/l. The patient was discharged in satisfactory condition on the fiftieth day of his hospital stay.

## Discussion

The increase of the intra-abdominal pressure is caused by the increase in the volume of the abdominal organs, the appearance of the free liquid, the diminished abdominal wall compliance as well as the increased pressure in the chest cavity (especially during mechanical ventilation), and the massive infusion therapy [8,9]. In our opinion, the development of the abdominal hypertension and abdominal compartment syndrome in the patient with acute hepatitis was caused by the increase in volume of the parenchymal organs (hepato-splenomegaly), the appearance of ascites, the abdominal swelling, and the inhibition of the gastrointestinal tract motility.

Development of ACS was preceded by gastrointestinal disorders in the form of the absence of peristaltic noises, bloating, constipation, and hemorrhagic stasis of the gastric contents. The damage of the gastrointestinal tract (GIT) in our patient was

not included in the primary lesion. Pathogenesis of the gastrointestinal lesion could involve neuroendocrine disorders, hypoxic and ischemic injuries, the impact of cytokines and other biologically active substances, dysbiosis and side effects of medications etc. [10]. In our patients, the GIT disorders preceded other signs of multiple organs failure, and possibly, stimulated the rise of the abdominal pressure. The accumulation of the free fluids in the physiological cavities (peritoneal and pleural) also belongs to the typical manifestations of intra-abdominal hypertension syndrome and the abdominal compartment syndrome. For instance, there was described direct relationship between the presence of the free fluid in the abdomen and the increased intra-abdominal pressure [11]. The free fluid could be either cause an abdominal compression or appear because of abdominal compression. In our patient the free fluid was found before the symptoms of the abdominal compression were observed. In the patient with the acute hepatitis B, the ascites formation obviously was influenced by the hemodynamic disorders and dysproteinemia.

The fact that intra-abdominal hypertension might cause multiple organs disorders suggests that its timely correction is needed. The methods for treating the intra-abdominal hypertension syndrome and abdominal compartment syndrome include both surgical and therapeutic methods [12]. Among therapeutic methods mostly recommended appropriate sedation and analgesia to improve the compliance of the abdominal wall; the reduction of the intestinal contents through drainage and prokinetics; the removal of the free fluid from the abdominal and pleural cavities; water balance correction; application of diuretics and colloid solutions; and the optimization of the respiratory support [13]. This patient received nasogastric and rectal drainage, early enteral nutrition, adequate sedation, corrected fluid therapy and were provided with negative fluid balance. Treatment was sufficient to achieve full recovery.

## Conclusion

Thus, course of acute viral hepatitis B in children patients may be associated with conditions that may provoke development of the intra-abdominal hypertension and the abdominal compression. When the risk factors are observed, it is crucial to monitor the abdominal pressure and to correct the hypertension in a timely manner to prevent the development of the multiple organ disorders.

## References

1. Schweitzer A, Horn J, Mikolajczyk RT, Krause G, Ott JJ (2015) Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013. *Lancet* 386(10003): 1546-1555.
2. Lampertico P, Agarwal K, Berg T, Buti M, Janssen H LA, et al. (2017) EASL 2017 Clinical Practice Guidelines on the management of hepatitis B virus infection. *J Hepatol* 67(2): 370-398.
3. Divarci E, Karapinar B, Yalaz M, Ergun O, Celik A (2016) Incidence and prognosis of intraabdominal hypertension and abdominal compartment syndrome in children. *J Pediatr Surg* 51(3): 503-507.
4. Arabadzhiyev GM, Tzaneva VG, Peeva KG (2015) Intra-abdominal hypertension in the ICU-a prospective epidemiological study. *Clujul Med* 88(2): 188-195.

5. Rogers WK, Garcia L (2018) Intraabdominal hypertension, abdominal compartment syndrome, and the open abdomen. *Chest* 153(1): 238-250.
6. Lee SL, Johnsen H, Applebaum H (2012) Cytomegalovirus enterocolitis presenting as abdominal compartment syndrome in a premature neonate. *World J Pediatr* 8(1): 80-82.
7. Liu MH, Lloyd SJ A, Gause C, Seifarth F, Deross A (2015) Abdominal compartment syndrome associated with Norovirus infection. *J Pediatr Surg Case Reports* 6: 20-23.
8. Lee RK (2012) Intra-abdominal hypertension and abdominal compartment syndrome: a comprehensive overview. *Crit Care Nurse* 32(1): 19-31.
9. Hunt L, Frost Sa, Hillman K, Newton PJ, Davidson PM (2014) Management of intra-abdominal hypertension and abdominal compartment syndrome: a review. *J Trauma Manag Outcomes* 8(1): 1-8.
10. Stefaniak J, Baron DM, Metnitz PGH, Kramer L (2010) Disturbances of gastrointestinal motility in intensive care units. *Anesthesiol Intensivmed Notfallmed Schmerzther* 45(11-12): 696-706.
11. Akhobadze GR, Chkhaidze MG, Kanjaradze D V, Tsirkvadze IB, Ukleba VA (2011) Identification, management and complications of intra-abdominal hypertension and abdominal compartment syndrome in neonatal intensive care unit (a single centre retrospective analysis). *Georgian Med News* 192: 58-64.
12. Rastogi P, Iyer D, Aneman A, D Amours S (2014) Intra-abdominal hypertension and abdominal compartment syndrome: pathophysiological and non-operative management. *Minerva Anesthesiol* 80(8): 922-932.
13. Doctor A, Zimmerman J, Agus M, Rajasekaran S, Wardenburg JB, et al. (2017) Pediatric multiple organ dysfunction syndrome: promising therapies. *Pediatr Crit Care Med* 18(3 Suppl 1): S67.

For possible submissions Click below:

[Submit Article](#)