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CASES OF THE ABDOMINAL COMPARTMENT SYNDROME IN CHILDREN WITH INFECTIOUS DISEASES

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ABSTRACT

Currently, the frequency of the intra-abdominal hypertension and the abdominal compartment syndrome (ACS) during the infectious diseases in children remains under researched. Measuring intra-abdominal pressure is generally not included in the routine methods of medical examination, and majority of these cases is not diagnosed. However, the infectious diseases are often accompanied with the processes that stimulate the increase of abdominal pressure. We conducted a retrospective analysis in children with the abdominal compartment syndrome that was treated in the intensive care unit at the Kyiv city municipal children's infection hospital from 2013 to 2015. We report on 2 cases of abdominal compartment syndrome complicated by multiple organ failure. Case 1: An 8-month-old male patient who was hospitalized with severe acute hepatitis B. On third day in the intensive care unit he presented increased intra-abdominal pressure to 16 mm Hg and signs of multiple organ dysfunctions. Case 2: A 10-months-old female patient with encephalitis. Signs of ACS was observed from second day of hospital stay. Both patients was treated with maintaining a negative water balance, limiting the volume of enteral nutrition and the usage of prokinetic agents.

INTRODUCTION

For the last 20 years, the intra-abdominal hypertension syndrome and the abdominal compartment syndrome are being studied extensively. Intra-abdominal hypertension is defined as a steady increase of abdominal pressure. The high pressure in an abdomen, in turn, might cause a cascade of hemodynamic disturbances and a multiple organ dysfunction that is called an abdominal compartment syndrome (Cheatham, 2009). The development of the abdominal compartment syndrome negatively influences the duration of intensive treatment and increases the risks of lethal outcome (Thabet, 2015). Intra-abdominal hypertension and abdominal compartment syndrome might as well complicate the course of various illnesses. The frequency of abdominal compartment syndrome in different categories of patients varies from 1 to 60%. Most observations come from the patients with surgical pathology. However, the cases of the abdominal compartment syndrome development in the patients with sepsis and enterocolitis are also being reported (Newcombe *et al*., 2012; Lee, 2012; Liu *et al*., 2015). Currently, the frequency of the intra-abdominal hypertension and the abdominal compartment syndrome during the infectious diseases in children remains under researched. Measuring intra-abdominal pressure is generally not included in the routine methods of medical examination, and majority of these cases is not diagnosed. However, the infectious diseases are often accompanied with the processes that stimulate the increase of abdominal pressure (for instance, strengthening of capillary leakage, hepato-splenomegaly, ascites, or inflammatory and functional disorders of the gastrointestinal tract etc.). The importance of the timely diagnosis of the intra-abdominal hypertension is related to the necessity to take special measures aimed at the normalization of the abdominal pressure and prevention of multiple organ complications.

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MATERIALS AND METHODS

We conducted a retrospective analysis in children with the abdominal compartment syndrome that was treated in the intensive care unit at the Kyiv city municipal children's infection hospital from 2013 to 2015. The abdominal compartment syndrome was diagnosed based on the criteria set by the World Society of the Abdominal Compartment Syndrome. Intra-abdominal pressure was determined through the presence of the risk factors for development of the intra-abdominal hypertension and the abdominal compartment

syndrome. The intra-abdominal pressure was measured in patients through the catheter inserted in their bladder. The pressure above 10 mm Hg was considered high. The abdominal compartment syndrome was diagnosed based on the steady increase of the abdominal pressure above 10 mm Hg and on the emergence of the organ dysfunctions (Kirkpatrick, 2013). The presence of the organ dysfunction was determined based on the criteria introduced in International pediatric sepsis consensus conference (2005) and on the logistic scale of the organ dysfunction PELOD. The evaluation of the neurological deficit was conducted based on the Glasgow and FOUR scales. Renal dysfunction was diagnosed in children with the increased creatinine level: in children younger than 7 days ≥ 140 μmol/L; in children from 7 days to 1 year of age ≥ 55 μmol/L; in children from 1 to 12 years of age ≥ 100 μmol/L; and in children older than 12 years of age ≥ 140 μmol/L (Leteurtre, 2010), Reduced urine (oliguria) was observed when the diuresis rate was reduced in children to the following levels: rate of urine output < 1 ml/kg/hour for children under 12 months of age; and rate of urine output <0,5 ml/kg/hour for children older than 12 months (Beattie, 2005) The criteria for liver dysfunction was based on the increase of ALT in 2 or more times above the upper limit of the norm or the increase in total bilirubin above 68,4 μmol/L (Goldstein, 2005), Respiratory dysfunction was determined based on the necessity to use ventilatory support for ensuring a sufficient level of saturation (Goldstein, 2005). For the evaluation of the blood pressure the lower limit of the norm was used: for children under 1 year of age – below 75 mm Hg; for children between 1 and 10 years of age – below 85 mm Hg; and for children older than 10 years of age – below 95 mm Hg (Slonim, 2006).

RESULTS

Cases Description

Patient 1

An 8-month-old male patient who was hospitalized with severe acute hepatitis B. During the hospitalization, the patient had jaundice; was conscious; had moderately swollen abdomen; exhibited minor limbs swelling; and had sufficient diuresis. 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, hemorrhagic syndrome (bleedings from the injection sites and hemorrhagic stasis in the stomach), and the diuresis level of 5,4 ml/kg/hour. The results of the laboratory examination revealed the progression of liver failure (the increase of the bilirubinemia up to 229 mkmol/l, prolongation of the prothrombin time to 112 s, decline in protein level to 54 g/l), and the lactate increase to 7,2 mmol/l. 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 16 mm Hg. 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 degrade: 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 the independent defecation was absent. 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 1,97 ml/kg/day); the creatinine level increased to 22,8 mmol/l. Intra-abdominal pressure ranged between 12-15 mm/ Hg. 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. The patient was discharged in satisfactory condition on the fiftieth day of his hospital stay.

Patient 2

A 10-months-old female patient with encephalitis who received inpatient treatment. The patient was hospitalized on the fifth day after disease onset with the observed impaired consciousness (sopor), focal neurological symptoms and intoxication. At the moment of hospitalisation, the parameters of central and peripheral hemodynamics were normal; the effective independent breathing was observed; the diuresis rate was adequate; and the active intestinal peristalsis was present. The laboratory tests indicated minor lymphocytic leukocytosis and increased transaminases level (AST – 110, ALT – 46 IU/l). On the second day after hospitalization, the patient's condition deteriorated significantly: decline of consciousness level reached coma 2 state; inefficient independent breathing was observed; violations of peripheral blood circulation were observed (cold limbs; capillary refill time – to 8 seconds); the abdomen was strained; and the peristalsis was flabby. The patient was transferred on the mechanical ventilation. During the following 24 hours, the diuresis reduced to 1,2 ml/kg/h; peristalsis was not present; the liver and the spleen increased in volume; free fluid appeared in the abdomen; and hemorrhagic stasis was observed in the stomach. Decompensated respiratory acidosis was observed in the patient's blood (pH-7,37, pCO2-46,6 mm Hg). After taking into consideration the strain of abdominal wall, the intra-abdominal pressure was measured. The intra-abdominal pressure was 17 mm Hg. To lower the intra-abdominal pressure, the introduction of fluid to the patient's body was limited; the prokinetics, diuretic drugs and an enteral drainage were used.

The patient's condition stabilized and remained critical for the 12 following days: the consciousness was at the coma 2 level; the signs of microcirculatory disorders were present (prolonged capillary refill time); and inefficient independent breathing was observed. The intra-abdominal pressure ranged within 8-16 mm Hg after the measurements were repeated. From the fourth day in intensive care, the increase in transaminases' level (ALT increased to 92,1 IU/l) and in the level of creatinine (up to 88 μmol/L maximum) was observed; but the diuresis level remained normal. Only from the 13-th day after the start of the treatment, the patient's condition began to improve gradually: the consciousness level was recovered; the hemodynamic stabilized; and biochemical parameters of blood reached the norm. Recovery of the neurological symptoms associated with the respiratory disorders lasted for a considerably long time. The patient was discharged on the 147-th day of her hospital stay with

incomplete recovery and symptoms of neurological deficits. Further rehabilitation treatment was recommended.

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 pressured in the chest cavity (especially during mechanical ventilation), and the massive infusion therapy (Lee, 2012; Hunt, 2014). 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. The factors that caused the increase in the intra-abdominal pressure, and consequently, an abdominal compartment syndrome in the second patient were capillary leak syndrome related to hypoxia and acidosis, inhibition of intestinal motility, and mechanical ventilation. An important issue is the early diagnosis of the intra-abdominal hypertension that allows timely to correct treatment and prevent multiple organ disorders. Among the early symptoms of the abdominal hypertension often reported in the literature is the abdominal distension. When studying the abdominal compartment syndrome in children, Thabet *et al*. (2016) noticed that the abdominal distension is an independent predictor of the abdominal hypertension (Thabet, 2015). In both our patients, an acute increase of the abdominal volume and the abdominal wall tension were observed among other symptoms before the appearance of the abdominal compartment syndrome, and were the main indication for measuring the abdominal pressure when the patient's condition deteriorated. In the patient with encephalitis, the abdominal strain became noticeable a few hours before his condition deteriorated sharply. In turn, in the patient with hepatitis, the abdominal distension was noticed already during hospitalization, and the first signs of the multiple organ failure appeared two days after.

Without the adequate correction, the steady increase of the intra-abdominal pressure causes the development of the pathological changes in the form of multiple organ failure. For instance, renal dysfunction is often being reported among the frequent and early symptoms of the abdominal compartment syndrome. Although it is not entirely clear in which way the damaging of kidneys happens, the direct compression of the renal parenchyma and the increased pressure in the renal veins are often mentioned among the possible causes for diuresis rate and glomerular filtration reduction. However, the influence of the prerenal disorders, reperfusion lesions and ischemic edema, endocrine dysfunction and the cytokine activity could not be excluded (Mohmand *et al*., 2011). Zhang and Liu (2015) when investigating the abdominal compartment syndrome in 60 patients found a direct relationship between the level of the intra-abdominal pressure and the rate of diuresis, and the renal biochemical markers (creatinine). The signs of oliguria were observed when the abdominal pressure increased to 2.31±0.4 mPa (approximately 17,3 mm Hg); the appearance of anuria and creatinine elevation were observed when the abdominal pressure reached to 3.11±0.5 mPa (approximately 23,3 mm Hg) (Zhang, 2015). In both patients that we treated, the signs of kidney dysfunction were observed in the form of the increase in creatinine level. However, in neither of the two cases, a significant decrease in the rate of urination (oliguria or anuria) was observed. One of the possible explanations for the absence of direct relationship between the biochemical markers and the diuresis in our patients could be related to the introduction of the corrective options (such as infusion, sympathomimetics or diuretics) at the early stages of the kidney dysfunction. In both patients, the gastrointestinal disorders in the form of the absence of peristaltic noises, bloating, constipation, and hemorrhagic stasis of the gastric contents were observed. The damage of the gastrointestinal tract (GIT) in our patients was mediated and was not included in the primary lesion. Pathogenesis of the gastro-intestinal 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. (Stefaniak, 2010). 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, Akhobadze *et al*. (2011) found a direct relationship between the presence of the free fluid in the abdomen and the increased intra-abdominal pressure (Akhobadze, 2011). The free fluid could be either cause an abdominal compression, or appear because of abdominal compression. In the patients that we treated, the free fluid was found before the symptoms of the abdominal compression were observed. In patient with encephalitis, the most probable reason for free fluids appearance, in our opinion, was capillary leak related to the hypoxia and acidosis. In the patient with the acute hepatitis B, the ascites formation 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 (Rastogi, 2016). The experts from the World Society of the Abdominal Compartment Syndrome recommend the following therapeutic methods for the normalization of the intra-abdominal pressure: the 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 (http://www.wsacs.org/education/algorithms.html). In the patients that we treated, the treatment of the causative factor (antimicrobial and anti-inflammatory therapy) was conducted and the therapeutic means for the reduction of the abdominal pressure were applied. In particular, both patients received nasogastric and rectal drainage, early enteral nutrition, adequate sedation, corrected fluid therapy and were provided with negative fluid balance. In the patient with encephalitis, the measurement of the abdominal pressure and the diagnostics of the abdominal hypertension were conducted when the progressive multiple organ disorder was present, and the development of the abdominal compression could not be prevented and stopped by the set of the corrective measures. In the patient with hepatitis, the correction of the abdominal pressure began before the appearance of the severe hemodynamic disturbances, stopped the progression of the

circulatory disorders and helped to achieve the stabilization of the patient's condition. Thus, in children patients, the severe forms of the infectious diseases could cause the conditions for the 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 organs disorders.

Conclusions

- The abdominal compartment syndrome might complicate severe infectious diseases in children.
- Monitoring the intra-abdominal pressure is a necessary instrument for examining patients when the risk factors for the development of the abdominal compartment syndrome are present.
- Timely diagnostics of the abdominal hypertension and the abdominal compression is required for the correction of the treatment methods and to decrease the probability of the complications and fatal outcome.

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