



The Giant Intraabdominal Liposarcoma as a Cause of Chronic Intraabdominal Hypertension and Inferior Vena Cava Syndrome

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Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

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Case Study

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ABSTRACT

Aims: We report a case of development of chronic intraabdominal hypertension (IAH) in patient with giant abdominal liposarcoma and finally its presentation as inferior vena cava syndrome.

Presentation of Case: A 77- year old patient presented with history of abdominal enlargement in last three years. The patient had dyspnea, legs edema and poorly tolerated supine position but he was without chest pain and complained on urinary frequency. CT scan of abdominal cavity shows huge tumor formation in front of abdominal cavity, and another one in pelvis, which compressed the bladder and spread into the inguinum. The patient had medical history of hypertension, chronic heart disease and chronic obstructive pulmonary disease (COPD).

Discussion and Conclusion: This case report indicates complex pathophysiology of gradually increased intraabdominal pressure (IAP), which occurs in conditions like morbid obesity, big intraabdominal tumors, pregnancy and ascites development. Since the IAH increases gradually over months or years, the compensatory mechanisms adjust gradually as well, and thus reduce a possibility of acute IAH complications and ACS (abdominal compartment syndrome) development. In our case, patient was presented with symptoms of inferior vena cava syndrome (IVCS) as a result of external compression by a mass on the IVC. This case highlights the importance of understanding of pathophysiological events in chronic IAH as well as in acute IAH.

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ABBREVIATIONS

IAP: Intraabdominal Pressure, IAH: Intraabdominal Hypertension, ACS: Abdominal Compartment Syndrome, IVCS: Inferior Vena Cava Syndrome.

1. INTRODUCTION

Increase in intraabdominal pressure (IAP) may be acute or chronic. Acute elevation of IAP develops within hours or days and may cause a life threatening condition, the abdominal compartment syndrome (ACS) [1]. Ileus, intestine perforation, ruptured abdominal aneurysm, peritonitis, acute pancreatitis, trauma, burns and abdominal wall pathologies such as rectus hematoma are among the causes of acute increase of IAP [2]. Chronic increase in IAP develops over a period of months or years in conditions like pregnancy, ascites development, morbid obesity or growth of large intraabdominal tumor. These processes cause a gradual abdominal wall stretches. Undiagnosed and untreated chronic abdominal hypertension might also progress to ACS. A sustained or repeated pathological elevation in IAP \geq 12 mmHg recorded by minimum of two standardized pressure measurements that are conducted 1-6 hours apart is defined as intraabdominal hypertension (IAH) [1]. The IAH may decrease tissue perfusion and ischemia of intraabdominal organs that requires an urgent abdominal decompression [3]. The chronic elevation of IAP, like the one in the morbidly obese, might contribute to a development of some co-morbid conditions such as arterial hypertension, urinary stress incontinence, lower extremity edema, venous stasis, obstructive sleep apnea and pseudotumor cerebri [4,5]. Inferior vena cava syndrome is caused by compression of the inferior vena cava and may be characterized by abdominal discomfort, abdominal ascites, hepatomegaly, and shortness of breath, increased risk for thrombosis and infection, legs edema [6].

We present a case of chronic abdominal hypertension caused by giant intraabdominal liposarcoma that progresses to vena cava inferior syndrome.

2. PRESENTATION OF CASE

A 77-year-old man with a past medical history of hypertension, chronic heart disease and chronic obstructive pulmonary disease (COPD)

presented to our hospital due to progressive abdominal distension over the last three years. CT scan of abdominal cavity shows huge tumor-like formation with 30 cm diameter in front of abdominal cavity and another one in pelvis with diameter of 12 cm. The last one compressed the bladder and spread into the inguinum. The patient denied any abdominal pain, bowel problems, nausea or vomiting. Surgery was recommended, but the patient refused it.

Three years later he was admitted to hospital with history of one month of dyspnea and legs edema. The patient had no chest pain and poorly tolerated supine position. The abdomen size was comparable to the status before 3 years. Stools were normal, but he complained on urinary frequency. On physical examination he was cachectic, pale, with dyspnoea and with large abdomen (Fig. 1). The heart rate and rhythm were normal, lung auscultation found crepitations and bronchial ronchi.

Abdomen was remarkably distended but with normal bowel sounds. The patient had a remarkable pitting edema of the lower extremities. Blood pressure was 151/97 mmHg, ECG shows sinus rhythm, 97/min, with right bundle branch block. Preoperative laboratory values were E 4.71 ($\times 10^{12}$), HgB 136 g/L, HTC 0.421 L/L, PLT 173 ($\times 10^9$), WBC 7.69 ($\times 10^9$), creatinine 99 μ mol/L, aspartate-aminotransferase 15 U/L, alanine-aminotransferase 11 U/L, alkaline phosphatase 50 U/L, K⁺ 4.8 mmol/L, Na⁺ 142 mmol/L, GUK 5.9 mmol/L, bilirubin 11.7 μ mol/L, urea 8.6 mmol/L, pH 7.43, pCO₂ 5.7 kPa, pO₂ 8.83 kPa, HCO₃ 27.9 mmol/L, tCO₂ 29.2 mmol/L, BE 3.2 mmol/L, SpO₂ 93.6%. The chest X-ray was normal, abdominal CT scan showed significant progression of abdominal tumor which occupied almost the whole abdominal cavity, both intraperitoneal and retroperitoneal spaces. The finding was suspected on liposarcoma. The patient was treated for acute heart failure with modest improvement of symptoms. Thereafter he was transferred to surgical ward. In the preoperative evaluation the heart ultrasound showed normal systolic function with left ventricle ejection fraction of 60%.

After induction of anaesthesia the IAP was measured using the transvesical technique with installation 25 ml of normal saline and zero point at mid-axillary line in supine position. The IAP value was 14 mmHg, MAP (mean arterial pressure) 106 mmHg, APP (abdominal perfusion pressure, MAP-IAP) 92 mmHg and CVP (central venous pressure) was 6 mmHg. Laparotomy was performed and first appeared grossly, well limited, solid consistency tumor measured 30x15 cm (Fig. 2). The tumor originated from the root of the small bowel mesentery. After the exploration and its removal, the other tumor mass with the same size and consistency appeared. There were also smaller ones with dimensions from 5 to 10 cm, which had infiltrated into the mesentery, pelvic wall and small intestine. Consequently the small intestine was resected. The total tumor's

weight was 20 kg (Fig. 3). The surgery passed uneventfully.

After the operation the hemodynamic parameters were measured including MAP, IAP, APP and CVP for two days every four hours, and FG (renal filtration gradient), creatinine, bilirubin, blood glucose, electrolytes and blood gases once daily. Two hours after the operation the patient respired spontaneously and MAP was 80 mmHg, IAP 15, 5 mmHg and APP 64, 5 mmHg, FG 49 mmHg, but on second day those parameters improved: MAP 113 mmHg, IAP 4 mmHg, APP 109 mmHg and FG 104 mmHg. Blood gases such as pO_2 was 9.5 kPa on second day after operation and SpO_2 94%, pH and other data did not change. The patient was discharged from ICU in good condition.



Fig. 1. Patient with huge abdominal mass at operating table, anaesthesia induction in progress



Fig. 2. Operative findings: The two parts of giant intraabdominal liposarcoma



Fig. 3. The total resected tumor mass

3. DISCUSSION

Liposarcoma mostly occurs in the deep soft tissue of extremities and in the retroperitoneum. It is the most common soft tissue sarcoma and accounts for 20% of all mesenchymal tumors. Liposarcoma arising from the mesentery of the bowel is a rare lesion. Some of the most common presenting symptoms of primary mesenteric liposarcoma include: increasing abdominal girth, weight loss, abdominal pain, abdominal discomfort with meals, and the presence of a freely moveable abdominal mass or masses. The pathohistological analysis confirmed the diagnosis of liposarcoma.

The patient developed the IAH due to gigantic liposarcoma with a gradual development of chronic intraabdominal hypertension and finally presented with inferior vena cava syndrome that urge surgical intervention. The chronic intraabdominal hypertension usually is connected with obesity, pregnancy and ascites formation and may be caused by malignancies. It is well known that morbid obesity is correlated with some co-morbidities including systemic hypertension, diabetes mellitus type II, urinary stress incontinence, obstructive sleep apnea and lower extremity edema. One of the presumed mechanisms for the development of obesity - related co-morbidities is a chronically elevated intraabdominal pressure [4,5]. Becerril and co-authors found correlation between pregnant preeclamptic patients and intraabdominal hypertension [7]. Paperini described two cases of Meigs' syndrome and development of chronic intraabdominal hypertension [8].

In our case the patient suffers from abdominal distension, shortness of breath and dyspnoea,

legs swelling and urinary frequency (just as the patients in Paperini's cases), and was hypertensive. At slowly increasing intraabdominal volume the IAP is maintained in normal range because of compliance of elastic abdominal wall which adapts and stretches gradually. Further increase of abdominal cavity causes abdominal wall stiffness with reduction of compliance and development of IAH.

In chronic IAH stepwise development of IAH is presented with symptoms of mechanical pressure of huge abdominal mass on intraabdominal vascular structures, diaphragm and bladder. Since the IAH increase gradually over months or years, the compensatory mechanisms adjust gradually as well, and thus reduce a possibility of acute IAH complications and the ACS (abdominal compartment syndrome) development. It seems that gradual elevation of IAP is better tolerated than the acute ones.

The major communication among the tissues within the human body is maintained via blood circulation, and lymph drainage of fluid from tissue into venous system. IAH can negatively affect the function of organs inside and outside the abdominal cavity, for example cranial displacement of the diaphragm increase intrathoracic pressure resulting in a reduction in the intrathoracic volume and an increase in the intrathoracic pressure, causing dyspnea.

Elevated pressure in the abdominal cavity compresses the vena cava, reducing blood flow through the vena cava to the heart. This drop in venous return (reduced preload) leads to a reduction in cardiac output. As the cardiac output reduces, the body responds by vasoconstriction

(increased SVR or afterload) to maintain an adequate central blood pressure. Chronic elevation of intraabdominal pressure reduces venous blood flow and compromises lymph drainage which leads to leg swelling and pitting edema. Because of direct compression of tumor mass on the bladder, its compliance is diminished and consequence is urinary frequency.

In our patient the complex multiorgan dysfunction symptoms were originally interpreted to be due to a heart failure. However, the intraabdominal hypertension caused by huge abdominal mass proved to be the primary cause, as evidenced by the postsurgical withdrawal of symptoms. The symptoms of chronic IAH suggest the need for timely surgical intervention and reduction of abdominal volume regardless IAP value.

4. CONCLUSION

In our case, patient was presented with symptoms of inferior vena cava syndrome and this is a unique presentation of chronic intraabdominal hypertension that leads to inferior vena cava syndrome because of a gradual disease development. This case highlights the importance of understanding of complex pathophysiological events in chronic IAH as well as in acute IAH/ACS.

CONSENT

The author declares that a written informed consent was obtained from the patient for the publication of this case report and accompanying images.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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