Respiratory arrest caused by a large uterine myoma

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Large abdominal masses increase intra-abdominal pressure, thus changing the haemodynamics of the patient by elevating the diaphragm and causing partial occlusion of the inferior vena cava (IVC). Large abdominal masses present many challenges, including life-threatening risks due to severe cardiovascular, pulmonary, and circulatory problems, as well as technical difficulties of surgery and postoperative complications. We report a case of a large pelvic-abdominal myoma with perioperative pulmonary compromise. The goal of this report was to familiarise other surgeons with the alterations in the pathophysiology and management of large abdominal masses.

It is rare to see patients present with exertional dyspnoea caused by a pelvic mass such as uterine myoma without underlying cardio-pulmonary disease. The most common symptom of myoma is menstrual disturbance. However, in cases of large uterine myoma, intra-abdominal pressure (IAP) can increase, which interferes with the pulmonary, renal, splanchnic and cardiovascular systems by elevating and splinting the diaphragm and partially occluding the inferior vena cava (IVC).[1] If untreated, IAP rises and multiple organ failure begins, and may progress to abdominal compartment syndrome (ACS), defined as a sustained IAP of 20 mmHg or higher and associated with new organ dysfunction.[2] Removal of the mass is the treatment of choice for ACS; however, gradual decompression is necessary. We report a case of perioperative respiratory failure precipitated by increased IAP, which was caused by a large abdominopelvic myoma.

Case report

A 42-year-old virgin woman presented to the emergency room for gradually worsening exertional dyspnoea and remarkable abdominal distension. Gradual abdominal distension had been noted by the patient over the past 3 years, but she became symptomatic only a week before admission. Medical and surgical histories were unremarkable. The patient complained of tachypnoea, and her oxygen saturation (SpO2) on room air was 89%. Abdominal examination revealed generalised abdominal distension with a mass located through the entire abdomen. The patient's height was 170 cm, and weight was 55 kg. Laboratory tests indicated hypoxaemia in arterial blood gas analysis (pH 7.43, partial pressure of carbon dioxide (pCO2) 45.5 mmHg, partial pressure of oxygen (pO2) 54.7 mmHg) and mildly elevated liver enzymes (aspartate aminotransferase (AST) 68 IU/L, alanine transferase (ALT) 72 IU/L). Other biochemical parameters, including tumour marker levels (CA 125 15.3 U/mL and CA 19-9 26.82 U/mL) were within normal ranges. Abdominopelvic computed tomography (CT) revealed a large, heterogeneous, multiseptated ovoid pelvic mass measuring 37 × 25 × 12 cm, occupying the pelvis and abdomen up to the diaphragm. It was unclear whether it was from the uterus, adnexa, or another organ (Fig. 1). Chest CT angiogram showed no signs of pulmonary embolism and the echocardiogram revealed normal ejection fraction (EF, 60%) and diastolic function (E/A ratio, 1.33).

To improve her respiratory impairment, 8 L of oxygen was administered in addition to intravenous diuretics. She was admitted to the Gynecologic Cancer Center for further evaluation of the abdominal mass. On the second hospital day, she complained of aggravated tachypnoea (respiratory rate, 40 per minute) and oliguria (urine output ≤20 mL/h). The clinical signs were thought to be suggestive of organ dysfunction due to progressive ACS. The gynaecological oncology team decided to perform exploratory laparotomy. At the time of surgery, we noted a 37 × 25 × 12 cm sized subserosal myoma-like mass arising from the uterus with secondary degeneration. Examination of the frozen section indicated a uterine leiomyoma, not a malignant condition. After ascertaining that the other pelvic organs were grossly normal, myomectomy was performed in view of her nulliparity.

After normal respiratory function was confirmed, she was extubated in the operating room and sent to the intensive care unit for monitoring of signs of multi-organ failure. Her vital signs were stable postoperatively; however, 1 hour later, she was tachyphoeic (respiratory rate 35 per minute) and her SpO2 abruptly dropped to 50%. Because of the possibility of inadequate ventilation, re-intubation with positive pressure ventilation to support adequate respiration was performed. The following day, the patient's urine output and liver enzyme levels returned to normal limits. After respiratory compromise had improved, she was extubated and transferred to the general ward.

The final pathology indicated a 6.54 kg uterine leiomyoma with hydropic change. She was discharged on postoperative day 6 with no pulmonary symptoms. The postoperative process was uneventful. The patient remains well 31 months postoperatively.

Discussion

Uterine myomas are benign, monoclonal tumours of the smooth muscle of the myometrium. Uterine myomas occur in approximately...
Pronounced rib flaring and attenuated diaphragm and abdominal total lung capacity, leading to tachypnoea and arterial hypoxaemia. The mass reduces functional residual capacity, vital capacity, and in the present case. Elevation and splinting of the diaphragm by 3 years. IAH may have caused preoperative pulmonary compromise. In our case, gradual growth of the uterine myoma occurred over 3 years. A large abdominal mass induced an increase in IAP that resulted in changes in the haemodynamics of the patient. A large intra-abdominal mass may cause immobility, breathlessness, and the inability to lie supine. Increased IAP can cause partial occlusion of the IVC, impaired venous return, and decreased cardiac output. Chronic vena cava compression produces venous stasis and dilated superficial abdominal veins, so-called caput medusa. Our patient experienced immobility and breathlessness but had normal cardiac output on echocardiography.

Intra-abdominal hypertension (IAH) is defined as a sustained or repeated pathological elevation of IAP to 12 mmHg or higher. IAH is diagnosed with a combination of clinical findings and monitoring of bladder pressure, but the diagnosis is often based only on clinical examination. Chronic IAH involves the insidious development of increased pressure over months (such as that during pregnancy) or years (such as a large intra-abdominal tumour or ascites). In our case, gradual growth of the uterine myoma occurred over 3 years. IAH may have caused preoperative pulmonary compromise in the present case. Elevation and splinting of the diaphragm by the mass reduces functional residual capacity, vital capacity, and total lung capacity, leading to tachypnoea and arterial hypoxaemia. Pronounced rib flaring and attenuated diaphragm and abdominal muscles have also been observed. Increased IAP may affect the viscoelastic properties of the thoraco-abdominal region, elevating chest wall resistance, decreasing chest wall compliance and inducing pulmonary oedema.

Undiagnosed and untreated chronic increased IAP can progress to an acute condition, ACS. ACS is defined as a sustained IAP of 20 mmHg or higher and is associated with new organ dysfunction. In the present case, respiratory distress, hypoxaemia, decreased urine output, and elevated liver enzyme levels were observed, that could be signs of multiple organ dysfunction, suggestive of ACS. ACS can be treated with abdominal decompression. Surgical removal of the mass was attempted in the present case; however, the diaphragm was lax, and the abdominal wall was weak after the removal of the myoma, thus resulting in pulmonary compromise after surgery. Another possible mechanism for postoperative pulmonary compromise is the sudden expansion of chronically collapsed lungs after removal of the large abdominal mass, causing re-expansion pulmonary oedema (RPE).

There is no standard method to prevent RPE but re-expanding the lung very slowly with a low tidal volume may be helpful. Any patient admitted for the removal of a large abdominal mass should undergo cardiac and pulmonary assessments. During the operation, placing the patient in the reverse Trendelenburg position with a tilt to the left side and continuous monitoring of blood gases may avert the supine hypotensive syndrome and prevent further respiratory difficulties. Gradual decompression of the mass reduces supine hypotension, and increases vital capacity. If the large mass is cystic, gradual decompression of the cystic fluid by paracentesis preoperatively and intraoperatively may prevent the phenomenon of splanchnic shock, which occurs when the compressed IVC is suddenly released. In case of a large, solid abdominal mass compressing the IVC, rolling the mass gradually off the IVC may be the optimal surgical technique. Delayed extubation is sometimes recommended to prevent pulmonary compromise. If extubation had been delayed in the present case to support the lax diaphragm and weak respiratory muscles, postoperative pulmonary compromise could have been prevented.

Uterine myomas are frequently encountered in the gynaecological department. Most clinicians perform surgery without any preparation; however, a chronic large abdominal myoma can alter the patient’s haemodynamics peri-operatively. We make the point that large abdominal masses, regardless of whether they are benign or malignant, can increase the risk of IAH and progression to ACS. Furthermore, surgeons should be familiar with the alterations in physiology and management of large abdominal mass.

References


Fig. 1. Abdominopelvic CT imaging of the patient; coronal view image depicting a 47 × 25 × 12 cm heterogeneous, ovoid mass.