An Unsuspected Case of a Cervical Degenerative Leiomyoma, from Mumbai, India

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Abstract Fibroids being the most common benign tumors, present with symptoms depending on their location. Cervical fibroids rarely grow excessively causing pressure symptoms and mimick an ovarian pathology. Also they do cause surgical difficulty in order of their close approximation to bladder and rectum. We present a case of a huge degenerative cervical fibroid not only contributing to a surgical difficulty but also a challenging post operative period.

Keywords Cervical fibroid; Clinical presentation of fibroid; Ischemic hepatitis; Shock marrow

Introduction Fibroids are most common uterine tumors. Cervical fibroids involved with excessive growth, may cause pressure symptoms. The treatment of the symptomatic fibroid is either myomectomy or hysterectomy. In the present case, cervical fibroid mimicking an ovarian tumour, caused clinical dilemma.

Cervical fibroid with excessive growth are uncommon. They give rise to greater surgical difficulty by virtue of their relative inaccessibility and close proximity to the bladder and uterus.

Case Report
A 40 yr old lady noted gradual distention of abdomen over two years. She gave no history of menorrhagia, pain in abdomen, bladder or bowel complaints. She had two full term normal deliveries. On examination there was a mass corresponding to 34 weeks gravid uterus which was smooth, non tender, cystic, non fluctuant and freely mobile. There was no associated ascites or hepato-splenomegaly. On per vaginal examination uterus was normal sized, retroverted, fornices were free and mass could not be felt in the pelvis. A provisional diagnosis of an ovarian tumor was made. Ultrasound revealed a large mass occupying the entire abdomen arising from either the uterus or ovary with mixed echogenicity and with multiple cystic areas.

CA-125 was normal. CT scan demonstrated a large well defined heterogenous predominately cystic mass measuring \(27 \times 23 \times 11.5\) cms arising from the left adnexa. Uterus was normal sized. The mass extended from the pelvis to the abdomen. (Picture 1, 2)
On opening the abdomen a large mass occupied the entire abdomen and it was difficult to identify the UV fold of peritoneum.

An attempt was made to aspirate the contents but no fluid could be aspirated. So the incision was extended and the mass was delivered outside the incision using traction. After exteriorising it was found that the mass was arising from the anterior part of the cervix and the uterus was seen posterior to the mass. The mass was attached to the cervix with a short pedicle without lateral expansion of the cervix. There was a simple 3 cms cyst in the right ovary (Picture 3). A total abdominal hysterectomy with right salpingo-oophorectomy was performed. Estimated intra operative blood loss was around 1000 ml. The mass excised was identified as a leiomyoma with cystic changes through pathology testing.

During the intra operative period she had hypotension when her systolic blood pressure (BP) dropped to 90 mm Hg which was corrected by blood transfusion and colloids. In the immediate post op period she had another episode of hypotension when BP dropped to 90 mm Hg. This was corrected by giving second blood transfusion and colloids.

On post operative day 3 her abdominal girth increased by 3 cms and she complained of giddiness. BP and hemoglobin were normal. An ultrasound showed moderate ascites, which was confirmed by tapping. The following were the lab reports (Table 1).

These lab abnormalities were probably as a result of the ischemic insult to the liver due to hypotension. She was managed conservatively by improving hydration and the abnormal laboratory parameters were restored after 2 weeks. Patient recovered well and was discharged.

Table 1: Laboratory reports

<table>
<thead>
<tr>
<th>Investigations</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total leucocyte count</td>
<td>7,500/cu mm</td>
</tr>
<tr>
<td>Platelet count</td>
<td>35,000 /cu mm</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>2.3 mg%</td>
</tr>
<tr>
<td>Direct bilirubin</td>
<td>1.8 mg%</td>
</tr>
<tr>
<td>SGPT</td>
<td>1,665 IU/L</td>
</tr>
<tr>
<td>SGOT</td>
<td>850 IU/L</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>435 IU/L</td>
</tr>
</tbody>
</table>

Discussion

Uterine leiomyomas are the most common neoplasm’s found in gynecological practice. According to the American College of Obstetrics and Gynecology (ACOG), fibroid occur in approximately 25-50% of all women. Most commonly in those ages, 30-40 yrs and affect African American women in higher numbers than their white counter parts. Cervical fibroids constitute 1-2% of total fibroids and are rare (Rao and Bande, 2005). Cervical leiomyomas in non-pregnant women rarely are of clinical significance and their complications include pressure effects on bladder or urethra, degenerative phenomenon and menorrhagia. (Tok et al., 2006)

Degeneration in fibroids, which occurs secondary to inadequate blood supply, may be hyaline (commonest), myxomatous, cystic, fatty, hemorrhagic or malignant in nature. The type of degenerative change seems to depend on the degree and rapidity of the onset of vascular insufficiency (Rein et al., 2008).

A review of literature suggests that the initiation of leiomyomas involves a multi step cascade of separate tumor initiators and promoters. The initial neoplastic transformation of the normal myocytes involves somatic mutations. Although the initiators of somatic mutations remain unclear. The mitogenic effects of progesterone may enhance the propagation of somatic mutations. Myoma proliferation is the result of clonal expansion and likely involves the complex interactions of estrogen, progesterone and local growth factors. Estrogen and progesterone appear
equally important as promoters of myoma growth (Rein et al., 2008).

The post op dramatic increase in liver enzymes along with thrombocytopenia with no evidence of sepsis in the post op recovery period could be as a result of acute hypotension for prolonged period. A review of literature explained this entity as an ischemic hepatitis and shock marrow.

Ischemic hepatitis is suspected in patients who have risk factors like sickle cells crisis and the following laboratory abnormalities.

Serum aminotransferanses increases dramatically (e.g. 1 000-3 000 IU/L)

LDH increases within hours of ischemia (unlike acute viral hepatitis)

Serum Bilirubin increases modestly, only to $<_4$ times its normal level.

PT/INR increases

Treatment is directed at the cause, aiming to restore hepatic perfusion, particularly by improving cardiac output and reversing any hemodynamic instability. If perfusion is restored aminotransferase decreases over 1-2 weeks. In most cases liver function is restored (Naqvi et al., 2000).

Thrombocytopenia commonly follows non septic shock and is correlated in severity with the degree and duration of hypotension. Significant anemia was less commonly observed probably due to the relatively long circulating life span of RBC’s. Thrombocytopenia is common following shock states unrelated to sepsis and may be related to hypoxic injury to hemopoietic progenitor cells. Platelet production appears to be more sensitive than neutrophil production and severe thrombocytopenia is associated with a poor outcome (Shaffer, 2007).

Injury induced by ischemia and reperfusion results from oxygen deprivation during the ischemic period and cytotoxic events during reperfusion. Following brief periods of ischemia, reperfusion stimulates the production of highly reactive molecule which can induce apoptosis or necrosis. As the ischemic period lengthens oxygen deprivation causes and increasing proportion of the ischemic injury.

Although there is considerable literature documenting association of septic shock with bone marrow failure, very few studies have demonstrated that marrow failure occurs in association with shock due to causes other than infection.

Usually cervical fibroids present with pressure symptoms such as retention of urine or constipation however in some cases they could simulate an ovarian tumor and all possibilities must be born in mind while managing such patients.

References


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Shaffer E.A., 2007, Hepatic ischemia, Merck manual Professional