Uterine myomatosis and portal vein thrombosis: a rare association

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ABSTRACT

Uterine leiomyomas are the most common pelvic tumors in women and occur in 20–30% of women over 30 years of age. Many complications are seen with fibroid. We report a rare case of a large uterine leiomyoma associated with portal vein thrombosis. 50-year patient presented lower abdomen swelling associated with pain and breathlessness, diagnosed as multiple fibroids. She had massive splenomegaly. Abdomen Doppler revealed splenomegaly with thrombosis of portal, splenic and superior mesenteric vein. Total abdominal hysterectomy with bilateral salpingo-oophorectomy was done. Iron deficiency anaemia with thrombocytosis caused by fibroid was the identified cause for portal vein thrombosis.

Keywords: Uterine myomatosis, Portal vein, Thrombosis, Iron deficiency anaemia

INTRODUCTION

Uterine leiomyomas are the most common pelvic tumors in women and occur in 20-30% of women over 30 years of age. Many complications are seen with fibroid. Those to be mentioned are torsion of a subserosal pedunculated leiomyoma, urinary retention, hemorrhage and thromboembolism. Thrombosis and subsequent embolic phenomenon due to large uterine fibroids is a very rare complication. Literature has quoted case reports were pelvic vein thrombosis has been encountered in patients with large uterine fibroids. Various causes and pathogenesis have been discussed as the contributing factors for development of thrombosis in the pelvic veins. But there has been no such report of an association of uterine fibroid with portal vein thrombosis. We present a rare case of a large uterine leiomyoma associated with portal vein thrombosis. Consent has been obtained from the patient to publish the details in a case report.

CASE REPORT

A 50-year-old female patient presented to the gynaecology OPD with complaints of Swelling and pain in the lower abdomen for past 5 months and amenorrhea for 2 months. Swelling was gradually increasing in size and was associated with pain radiating to the back and loin. It was also associated with breathlessness, loss of appetite and loss of weight. On physical examination, she was thin built with a body mass index of 19.5 kg/m², body temperature 37°C, blood pressure 100/70 mmHg, heart rate 84 beats per min and respiratory rate of 24 breaths per min with clear lung sounds. Examination of the abdomen revealed a mass around 26 weeks size of gravid uterus, irregularly enlarged and non-tender. The largest myoma was 8 × 8 cm. Multiple subserous fibroids were palpated, each measuring 4–5 cm. Apart from the fibroid she also had dilated veins in the flanks and massive splenomegaly which was palpated 6cm below the costal margin. So an initial working diagnosis of uterine myomatosis with splenomegaly was made and was planned to evaluate for the causes of splenomegaly before further intervention.

Hematologic testing revealed hemoglobin of 8.9 g/dL, platelets - 5,40,000/mm³ and peripheral smear was microcytic hypochromic anaemia with thrombocytosis. Coagulation profile was unremarkable with a prothrombin time INR of 1.5. Renal function (Blood urea - 25mg/dl, Serum creatinine - 0.9mg/dl) and liver
association of fibroid?

Why do we report this as a rare association of fibroid? Here we will have to answer two questions. One, if fibroid is the reason behind the thrombosis in the portal system, what is the mechanism behind it? Two, why isn’t the portal system thrombosis (extra-hepatic portal vein obstruction) a co- incidental finding rather than an association of fibroid?

The authors have made an attempt to answer the above two questions. Thrombus formation is governed by the principles of the Virchow’s triad namely, hemostasis, endothelial injury and hypercoagulability. When considering the mechanism in large fibroid uterus, it is attributed to stasis of blood in pelvic veins due to obstruction. What is the attributable cause in our patient? It has been reported that iron deficiency anaemia is associated with thrombosis of cerebral veins and venous sinuses. Our patient had iron deficiency anaemia with thrombocytosis. This is one main attributable cause. Iron acts as a controller of thrombopoisis and regulates the level of platelets. In iron deficiency the level of megakaryocytes increase thereby increasing the platelet level, thus resulting in a hypercoagulable state. Sometimes even thrombocytopenia can be present due to other consumption of platelets. Iron deficiency also induces a hypercoagulable state by altering pattern of blood flow. The microcytosis resulting from iron deficiency causes reduced red cell deformability and increased viscosity, which contributes to thrombosis in a negative-pressure environment, as is found in veins. Further poor oxygenation due to reduced hemoglobin levels lead to metabolic stress and endothelial damage.

The portal vein forms at the junction of the splenic vein and the superior mesenteric vein behind the pancreatic head. Thrombosis of the portal vein can occur due to hepatic causes like cirrhosis or malignancies or extra hepatic causes. In our patient it was extra hepatic portal vein obstruction. Extrinsic obstructions and inherited hypercoagulable states have been ruled out in our patient (normal ultrasound and coagulation study). Further reduction in the size of splenomegaly after removal of fibroid also suggests an association between them. Hence we conclude that this appears to be a de-novo presentation of portal vein thrombosis associated with uterine myomatosis due to the presence of iron deficiency anaemia with thrombocytosis.

As this the first reported case we have tried to draw an association between fibroid uterus and portal vein thrombosis. Meticulous search to look for such complications in future can further enlighten us for the betterment of health care services to patients.

CONCLUSION

Venous thrombosis in patients with fibroid though a rare complication has now been reported in many cases. So importance should be given to search for such
complications, especially in patients with large fibroids in atypical sites also.

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**REFERENCES**
