Disseminated Intravascular Coagulation after Myomectomy: A Case Report and Review of Literature

Mohan S Kamath, Mousumi Acharya, Vandana Kamath, TK Aleyamma

ABSTRACT
Hematological complications can complicate the postoperative period following myomectomy. Clinicians should keep such rarer possibilities in mind which will help them identify the complications correctly and manage appropriately. We managed a case of disseminated intravascular coagulation following myomectomy which was promptly diagnosed and managed.

Keywords: Disseminated intravascular coagulation, Myomectomy, Leiomyoma.

INTRODUCTION
Uterine leiomyoma is one of the commonest benign tumors diagnosed in women. For infertile women or those who wish to retain the uterus, conservative surgical intervention in the form of myomectomy, either laparoscopic or open surgery is frequently offered.1

One of the main complications of myomectomy is hemorrhage, which when uncontrolled, may necessitate hysterectomy as a last resort. Though many prophylactic measures, such as tourniquets application, injecting Vasopressin and uterine artery ligation are advocated to reduce intraoperative blood loss, at times blood replacement is required to compensate for the loss during surgery.2

Massive hemorrhage can lead to disseminated intravascular coagulation (DIC) and the sequela (renal failure).3 There are case reports of patients developing hemolytic uremic syndrome and renal failure following uncomplicated myomectomy, which due to similar clinical presentation, could possibly present a diagnostic dilemma for the clinicians.4

We present a rare case of DIC following myomectomy which was managed conservatively accompanied by a review of literature.

CASE REPORT
A 26-year-old unmarried woman, presented to outpatient clinic with history of menorrhagia for 1 year. On clinical evaluation, she was found to have abdominopelvic mass, approximately 14 to 16 weeks size, mobile, and lower borders could not be felt. Her blood test revealed a hemoglobin of 8.3 g/dl, and on transabdominopelvic ultrasound showed a single posterior wall intramural fibroid of 7 × 8 cm size, close to uterine cavity. Her blood counts were within normal limits. During initial evaluation, due to suspected ovarian mass, a CA 125 was sent and the value was found to be 431 IU. The computed tomographic (CT) scan revealed normal ovaries and confirmed the ultrasound findings of fibroid uterus. She was transfused with one unit of red cell concentrate preoperatively, and started on hematinics. A laparoscopic myomectomy was planned after her hemoglobin improved to 10.8 g/dl.

Intraoperatively, during laparoscopic myomectomy, injection vasopressin was injected and horizontal incision over the posterior uterine wall was made. Due to degeneration, the fibroid was soft in consistency making it difficult to identify the right plane for dissection and enucleation. Due to excessive bleeding intraoperatively, the decision for laparotomy was taken. Pfannenstiel incision was given, and uterus was exteriorized. The fibroid was enucleated and dead space obliterated and hemostasis achieved. An indwelling drain was kept. Total surgery time was 90 minutes and estimated blood loss was 900 ml.

Intraoperatively, one unit of blood was transfused and second unit of blood was started in the immediate post-operative period.

Sixth hours postoperatively, her blood pressure was 94/60 mm Hg and pulse rate was 102/min. Wound drain output was 400 ml over 12 hours and urinary output maintained at 50 ml/hr overnight. On the first postoperative day, the general condition of patient worsened and the patient complained of increased anxiety, abdominal discomfort and nausea. Her pallor had increased and the wound site revealed hematoma and ecchymotic changes. The drain wound dressing was also getting soaked. Urgent hemoglobin and coagulation studies were done. The hematocrit was 20% and her coagulation profile revealed disseminated intravascular coagulation picture (Table 1). Since, the urine output was normal, the renal function tests were not ordered. She was immediately resuscitated with blood, fresh frozen plasma (FFP) and platelet concentrates. The coagulation profile repeated after 6 hours
revealed improved blood picture. Clinically the patient stabilized and the wound drain flow reduced. Coagulation profile was repeated every 6 hours till all the parameters returned to normal levels. The patient’s condition improved markedly by second postoperative day and by 6th postoperative day, she was discharged after being advised regarding wound dressing. On 14th postoperative day, the wound had healed well.

**DISCUSSION**

Our case of DIC following myomectomy is not so uncommon but illustrates the importance of prompt identification of hematological complications in the postoperative period, which can markedly reduce the morbidity in such cases.

DIC has been reported as one of the more common hematological complication following myomectomy apart from rarer condition, such as microangiopathic hemolytic anemia, which can cause a diagnostic dilemma. The other differential diagnosis in such scenario is thrombotic thrombocytopenic purpura (TTP).

The main pathophysiological process in DIC is systemic activation of coagulation system and microvascular deposit of fibrin, which results in organ failure. The activation and consumption of coagulation factors manifests clinically in the form of bleeding wound sites and hematoma formation which can have devastating consequences depending upon the site and amount of bleed. Some of the common clinical conditions associated with DIC include sepsis, severe tissue trauma, head injury, malignancies, obstetrical complications (abruptio placentae, amniotic fluid embolism, etc.), reactions to toxin and immunologic causes. In the initial phase of DIC, the coagulation pathway gets activated by release of various tissue factors (e.g. due to trauma, sepsis, etc.) and proinflammatory cytokines (e.g. IL-6) which lead to hypercoagulable phase. There is impairment of natural anticoagulant mechanism which further accentuates the process. Subsequently, due to overutilization of procoagulants and platelets, the hypocoagulable phase manifests clinically in the form of severe bleeding.

The possible mechanism for DIC following myomectomy could be multifactorial. The excessive tissue trauma and bleeding could be possible contributory factor. The uterus is known to be rich in tissue factor and extensive handling of fibroid could lead to massive release of such tissue factors, which when combined with other factors (surgery, administration of colloids and sepsis) could be ideal setting for DIC to set in.

No single coagulation parameter is diagnostic of DIC. Thrombocytopenia or a progressive drop in platelet count is a sensitive test for DIC. Thrombocytopenia is present in 98% of DIC. Prolongation of prothrombin time (PT) and activated partial thromboplastine time (aPTT), elevated International normalized ratio (INR), raised D-dimer and low fibrinogen levels are some of the other abnormalities observed in DIC. However, hypofibrinogenemia has a low sensitivity and is seen in very severe DIC only.

According to the scoring system developed by International Society of Thrombosis and Hemostasis, a score of more than 5 is diagnostic of DIC. This system relies on fibrin degradation product, platelet, D-dimer, PTT and fibrin concentration.

Hemolytic uremic syndrome (HUS) is a triad of microangiopathic anemia, thrombocytopenia and acute renal failure. It is commonly seen in children following gastrointestinal infection (D+HUS). Rarely, it presents without any gastrointestinal infection (D-HUS) and has been associated with various non enteric infections, drugs, malignancies and autoimmune conditions such as scleroderma. The main pathophysiological mechanism is complement dysregulation. The other differential diagnosis of TTP is characterized by deficient ADAMTS13, a metalloproteinase, leading to intravascular platelet aggregation.

DIC and HUS/TTP can be difficult to distinguish. Further in adults, D-HUS and TTP are both very difficult to diagnose clinically and are currently, the term TTP-HUS is often used. In DIC, the coagulation parameters are deranged whereas in HUS/TTP, it is generally normal and can help differentiate between these two clinical entities.

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<thead>
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<th>Table 1: Laboratory findings</th>
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<tr>
<td>Normal range</td>
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<tr>
<td>Hemoglobin (gm/dl)</td>
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<tr>
<td>Hematocrit (%)</td>
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<tr>
<td>Platelet count (×10^9/l)</td>
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<td>Blood picture</td>
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<tr>
<td>PT (sec)</td>
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<td>aPTT (sec)</td>
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<td>INR</td>
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<td>D-dimer (ng/ml)</td>
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<td>Fibrinogen (mg/dl)</td>
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PT: Prothrombin time; aPTT: Activated partial thromboplastin; INR: International normalized ratio
to differentiate DIC from HUS/TTP is important since platelet therapy is generally contraindicated in HUS/TTP. While DIC is treated by correcting the underlying cause and transfusion of appropriate blood products, HUS/TTP is mainly treated by therapeutic plasma exchange, and renal dialysis if required.8

There have been previously reported cases of DIC following uncomplicated myomectomy.7,9 Sack et al postulated extensive myometrial dissection and use of 4% icodextrin as adhesive barrier, with subsequent development of DIC postoperatively.10 Li et al reported a case of DIC following uncomplicated laparoscopic myomectomy and identified the disruption of pseudocapsule, oxytocin injection, morcellation and positive intrabdominal pressure as some of the possible contributory factors for development of the hematological complication.3 Tsimpanakos et al highlighted the diagnostic dilemma following development of intravascular hemolysis and acute renal failure after myomectomy.4 The author described the possible differential diagnosis of D-HUS and DIC and difficulty in differentiating the two in such acute clinical settings.

Though such complications are rare, our case report highlights the need for close monitoring and prompt diagnosis of hematological complications following myomectomy which can possibly limit the morbidity. Unusual bleeding tendency postoperatively and blood tests revealing a drop in hemoglobin and platelet count should alert the clinician and coagulation studies should be initiated on an urgent basis. Further, hematologist inputs should be taken in these cases since failure to identify HUS/TTP could delay potentially life-saving plasma exchange.

REFERENCES


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