

DISSEMINATED PERITONEAL LEIOMYOMATOSIS

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A 42-year-old, gravida 3, para 2, patient with a history of an abortion and a previous cesarean section presented to our outpatient clinic with a complaint of menometrorrhagia for the last 6 months. Her vital findings were: temperature, 36.5°C; blood pressure, 120/80 mmHg; pulse, 84 bpm; respiration, 10/minute. Her pelvic examination revealed a globally enlarged uterus, approximately the size of a 14-week pregnancy. Ovaries were also enlarged. Ultrasonography showed a uterus 100 × 135 mm with multiple myomas, the largest being about 5 cm diameter. Douglas pouch was free of any fluid, and the adnexal lodges were evaluated as normal. Other systemic examinations and laboratory findings were normal. The patient, who had been using oral contraceptives for the last 10 years, was offered a diagnostic uterine curettage which revealed a secretory endometrium. She was then advised to have an elective abdominal hysterectomy, taking into consideration her complaints and her age. Intraoperative evaluation of the patient revealed multiple myomatosis of the uterus with exophytically expanding myomas over the peritoneum and the omentum. After hysterectomy, a midline dissection of the uterus revealed exophytic nodules originating from the myometrium, as well as a 4-cm intramural myoma (Figures 1 and 2). The frozen section pathology report confirmed leiomyomatosis. The patient underwent a total abdominal hysterectomy and bilateral salpingo-oophorectomy, with total omentectomy.

In the pathologic examination, sections of peritoneal biopsy specimens showed tumoral nodules with bundles crossing over each other and mesenchymal proliferation resulting in nodular structures on the peritoneum (Figure 3A). Bundles of spindle cells with no neoplastic proliferation, atypia or mitosis were seen (Figure 3B).

She was discharged on postoperative day 3 without any complications. Postoperative thorax tomography did not show any metastatic foci.

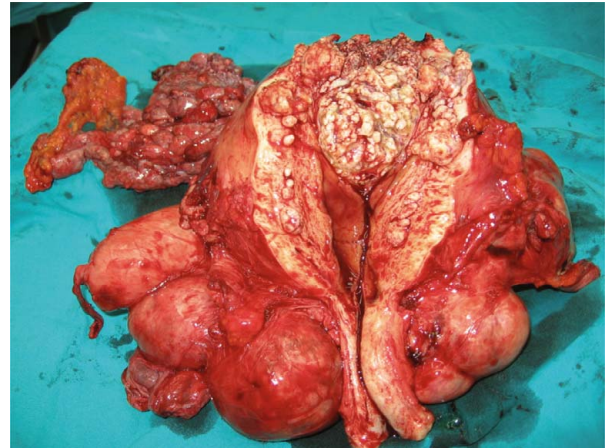


Figure 1. Uterus with multiple myomas.



Figure 2. Exophytic appearance of leiomyomatosis.

Leiomyomatosis peritonealis disseminata (LPD) was first reported by Willson and Peale in 1952 [1]. The exact etiology is not known, but it is thought to originate from metaplasia of the submesothelial multipotential mesenchymal cells [2]. States of prolonged estrogenic conditions, such as long-term oral contraceptive use, pregnancy, granulosa cell tumor and endometriosis, have all been implicated in the etiology [3–5]. In our case, there was a history of long-term oral contraceptive use, but it should be kept in mind that disseminated peritoneal leiomyomatosis can be seen in patients without long-term oral contraceptive use [6].



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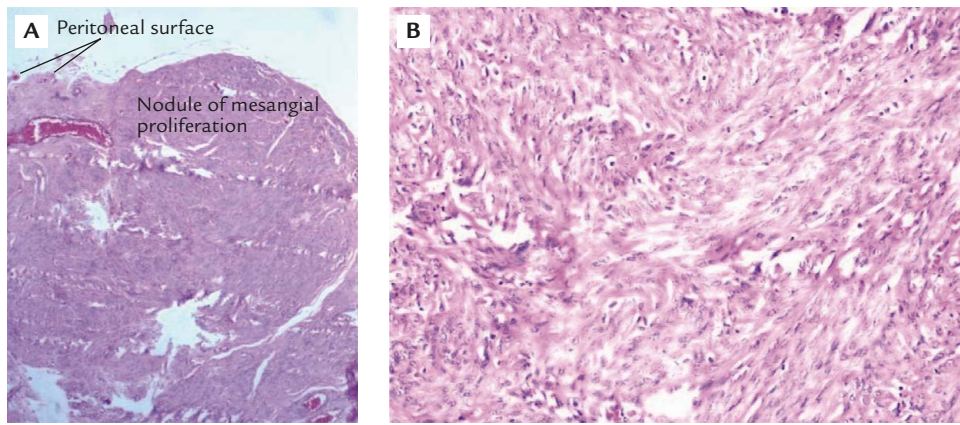


Figure 3. (A) Mesenchymal proliferation forming nodular structures on peritoneum. (B) Bundles of spindle cells with no neoplastic proliferation, atypia or mitosis.

When the literature was reviewed, it was seen that LPD often stays asymptomatic but is sometimes diagnosed after complaints such as lower abdominal pain and menorrhagia. It is also encountered sporadically as pelvic peritoneal or abdominal nodules during laparotomies performed for other reasons.

LPD may be misdiagnosed as a disseminated malignancy due to its macroscopic appearance with nodular implants [7]. Based on examination of a frozen section, this case was reported as a benign lesion.

There is no consensus established on the treatment of those patients. There have been six sarcomatous changes reported in the literature [8]. Even though inevitable hormonal inactivation in menopause would cause regression of these lesions, the sarcomatous changes mentioned in the literature and the reported high recurrence rates are reasons for the surgical treatment of patients without fertility expectancy [9].

In young patients with fertility expectancy, the use of gonadotropin-releasing hormone (GnRH) agonists, such as danazol and megestrol acetate, has been suggested [10–12].

Two of the three patients with diffuse leiomyomatosis and wishing to retain their fertility were treated with GnRH analogs and myomectomy. The other patient was treated with a GnRH analog only. The patient treated with a GnRH analog alone conceived, and delivery was performed by emergency cesarean section followed by a cesarean hysterectomy at the 34th week of her pregnancy because of heavy vaginal bleeding. The other two, who were treated with medical therapy in addition to surgical therapy, failed to conceive. Medical treatment should therefore be considered in young patients with a fertility expectancy [13].

In another case, a 30-year-old nulliparous patient was diagnosed with a pelvic mass, and during the operation, LPD was revealed. Since she wished to retain her fertility, conservative surgery was performed and she was

subsequently given danazol. This patient subsequently delivered twice by cesarean section, and no sign of LPD was encountered during these operations [11].

Uterine artery embolization with preservation of the uterus and ovaries could be another treatment option for LPD in patients desiring future pregnancies [14]. The surgical and medical treatment options should definitely take into account the fertility status and desire of the patients. In our case, the lack of desire for future fertility and the disseminated nature of the disease with many omental implants influenced our decision towards a surgical approach, in an attempt to reduce the estrogenic stimulus. Generally, LPD has a good prognosis, even though some sarcomatous degenerative cases have been reported. With hormonal inactivation and removal of the tumoral mass, LPD can be presumed to regress, and additional treatment may not be necessary [15].

Conversely, malignant cases do not have a good prognosis, and early stage death has been reported [8].

In conclusion, every patient should be evaluated and treated individually, taking into consideration the desire for fertility and the severity of her symptoms.

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