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EDİTÖRE MEKTUP / LETTER TO THE EDITOR

Leiomyomatosis peritonealis disseminata mimicking peritoneal carcinomatosis

Peritoneal karsinomatozisi taklit eden dissemine peritoneal leiomyomatozis

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To the Editor,

Leiomyomatosis peritonealis disseminata (LPD) is a very rare benign disease affecting premenopausal women. The first case of LPD was described by Willson and Peale in 1952[1]. It is suspected that this disease originates from a metaplasia of sub-mesothelial multipotent mesenchymal cells[2]. Since female gonadal steroids play an important role in the pathogenesis of LPD, they are usually associated with highly exogenous and endogenous female gonadal steroids[3]. Generally, LPD is characterized by the development of multiple smooth muscle-like nodules in the peritoneal cavity, grossly or radiologically mimicking peritoneal carcinomatosis or disseminated intraabdominal malignancy[4,5]. The definitive treatment of LPD is total abdominal hysterectomy, bilateral salpingoopherectomy and resection of myomatous nodules (debulking) depending on the severity of the disease.

A 39-year-old woman was followed with intramural fibroids and endometrioma in the uterus. Informed consent was obtained from the patient. On magnetic resonance imaging (MRI), a 5x4cm solid lesion in the uterus with hyperintense and heterogeneous signal on T2-weighted sequences (T2W), no diffusion restriction on diffusion-weighted images (DWI), and a 5.5x4cm follicle cyst in the right ovary were observed. One year later, the operation was planned because there was a progression in the myoma size. On preoperative MRI, nodular lesions were observed, hyperintense in T2W, hypointense on T1-weighted sequences (T1W), intensely and heterogeneously enhancing after intravenous contrast injection located in the right adrenal region, adjacent to the liver capsule, adjacent to the uterus, between the intestinal loops and the pouch of Douglas. The lesions with mild diffusion restriction were thought to be compatible with malignancy in the first place. Hysterectomy, excision of the lesion adjacent to the appendix, appendectomy and implant compatible masses in all of the mentioned localizations were excised. LPD or benign leiomyoma metastasis was considered in the pathological examination of the specimen. One-year follow-up CT scan showed no evidence of abdominal recurrence or implantation.

The LPD is a benign and very rare disease which has been reported less than 140 cases[6]. It is mostly asymptomatic; therefore, it is detected while the patient is being evaluated for another reason. It is characterized by the presence of multiple smooth muscle nodules on the peritoneal surface. To date, fewer than 140 cases have been reported in the literature and the estimated prevalence of LPD is <1/1.000.000[7]. The etiology of LPD is unknown. It is considered as the LPD is induced with hormonal or genetic factors during metaplasia of submesothelial mesenchymal cells[2]. Another hypothesis of LPD etiology is the seeding of fibroids

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which can be by morcellation of fibroids during laparoscopic surgery[8]. Thus, the LPD is a multifactorial disease. Also, the gonadal steroids due to pregnancy or long-term oral contraceptive use and the presence of estrogen and progesterone receptors in uterine fibroids might play an important role in the pathogenesis of LPD[6]. The presence of hard-viscous solid lesions on the peritoneum, uterine fundus, ovaries, intestinal mesentery, and omentum are very similar to disseminated abdominal malignancy, metastatic leiomyosarcoma or peritoneal carcinomatosis. This situation might lead surgeons or pathologists to a misdiagnosis. The histopathological features of LPD are sub-peritoneal nodules with typical interlacing of smooth-muscle cells arranged in whorled pattern without atypia, stromal invasion and with no or low mitotic activity. Therefore, the definitive diagnosis of LPD should be done histopathologically[9].

In the presence of LPD without exogenous or increased endogenous exposure, tumors without estrogen or progesterone receptors expression, and postmenopausal women with relatively large sized nodules, malignant transformation has been reported in two out of three cases. Incidentally diagnosed LPD cases have a good prognosis, which often regresses after hormonal stimulation is discontinued. Only some cases require the use of gonadotropin-releasing hormone agonists or surgical castration; however, malignant transformation has a very poor prognosis despite multimodality combination therapy[4].

The patients are followed-up with CT or MRI and CA-125[5]. On computed tomography, it shows as homogeneous or heterogeneous contrasting lobulated hypodense lesions located in the peritoneal cavity and uterus similar attenuation as the soft tissue. MRI is the imaging modality of choice for LPD because of its excellent soft tissue characterization. Typically, lesions appear isointense to skeletal and smooth muscle on T1W and T2W images, with intense contrast enhancement on post-contrast T1W images. Absence of ascites or omental cake support LPD as the most likely diagnosis, especially if the patient has a concomitant fibroid uterus or has had a hysterectomy for a leiomyomatous uterus. Areas of necrosis and bleeding indicate suspicion of malignancy. Areas of flare on GRE images, increased signal intensity on T2W and STIR images suggest necrosis and hemorrhage[10]. On diffusion-weighted images (DWI), malignant leiomyomas may show limited diffusion with apparently reduced diffusion restriction values compared to benign leiomyomas.

LPD is not common. However, it should be considered in a patient with solid nodular lesions on the peritoneum, especially after myomectomy or hysterectomy.

Although the MRI especially DWI secans is useful for the diagnosis of LPD, the diagnosis of LPD should be done histopathologically.

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