Giant peritoneal loose body in a patient with end-stage renal disease

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Abstract
A 72-year-old male with end-stage renal disease underwent a computed tomography scan to assess renal function. An oval-shaped mass, 50 mm × 60 mm in size, was discovered incidentally in his recto-vesical pouch. Because it was suspected to be a teratoma, which could be an impediment for future renal transplantation, surgery was performed. It revealed a giant peritoneal loose body, a rare entity, that has not been reported before in patients with renal chronic insufficiency.

Keywords
Appendix epiploica, peritoneal loose body, radiology, surgery

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Introduction
Peritoneal loose bodies (PLBs) are rare entities found incidentally during routine examinations or as a result of complications that intervene by their extrinsic compression. Their pathophysiology is thought to derive from torsion, infarction, and calcification of appendices epiploicae, although other origins have been reported (calcified fibromyoma, auto amputated adnexa, calcified extra uterine pregnancy). In rare instances, they can grow to more than 5 cm in size due to protein absorption from peritoneal serum, in which case they are called giant PLBs. We report an unusual case of giant PLB discovered during imagistic explorations in a 72-year-old male proposed for kidney transplantation due to chronic kidney disease.

Case presentation
A 72-year-old male was referred to our unit because of an incidental computed tomography finding of a solid, well-defined mass, 58/65/48 mm in size, with central calcification of 23/27 mm, located in the pelvic cavity, between the sigmoid colon and the urinary bladder, thus highly suggestive of teratoma (Figure 1). The patient was diagnosed with stage V chronic renal disease, supposed to occur due to acute tubular necrosis a year before consultation and has since received hemodialysis three times weekly. Due to the above imagistic findings, kidney transplantation was temporarily excluded from the possible therapeutic management of his kidney disease.

His case history revealed associated obesity (body mass index of 37 kg/m²) and hypertension. On physical examination, no abnormality was found. Colonoscopy revealed grade II hemorrhoids, otherwise—normal colonic mucosa. Tumor markers were within normal ranges. Biochemical analyses showed elevated triglycerides, total cholesterol level, urea, and creatinine. Exploratory laparotomy was performed with discovery of a white, oval-shaped PLB, 5.8 cm × 6.5 cm in size, situated in the recto-vesical pouch, with a smooth, hard cartilage surface (Figure 2). The cross section revealed a central calcified area (Figure 3). Postoperative recovery was uneventful.

On histopathologic examination, the lesion consisted of lamellar connective tissue with diffuse calcifications (Figure 4).

The patient was discharged from the hospital on day 5 after surgery.

Discussion
PLBs or so-called peritoneal mice have not been reported in patients with renal chronic insufficiency. They are defined as bodies that developed free from the lining of the abdomen,
resembling the loose bodies found in joints. PLBs’ size usually ranges from 5 to 25 mm, and they generally do not cause any symptom. Few cases have been reported with a diameter of more than 5 cm, the biggest one measuring 95 mm × 86 mm. In 1863, Virchow proposed the theory of PLBs’ origin: obesity or infection can trigger an increase in the amount of fat in appendices epiploicae. This can lead to saponification and calcification of the fat and therefore to progressive obstruction of the blood vessels of the pedicle. When the vascular obstruction is complete, appendix epiploica suffers infarction and falls into the peritoneal cavity. Nevertheless, Patterson suggested that torsion and inflammation are the main factors for ischemia and detachment of appendices epiploicae. Our case supports Virchow’s theory, the patient suffering from severe obesity.

We believe that the PLB in our case grew to its dimensions because the peritoneal fluid was rich in proteins. Han et al. suggested that the peritoneal membrane permeability in patients with end-stage renal disease may be altered because of peritoneal lymphatic channel obstruction. In nephrogenic ascites cases, he described the peritoneal fluid to be high in protein content. In our case, we assume that due to end-stage renal disease, the patient’s peritoneal serum contains a higher amount of proteins than in cases without renal insufficiency, thus favoring protein deposition on PLB and its faster growth.

Other possible etiologies include auto amputated adnexa, calcified extra uterine pregnancy, omentum, auto
amputated subserosal uterine leiomyoma, and fat tissue in the pancreas.

Symptoms in small PLBs are typically absent, and they are usually discovered incidentally at laparotomies or during imagistic analysis. On the contrary, giant PLBs may show with acute urinary retention, constipation or, even, acute intestinal obstruction due to extrinsic compression. To differentiate them from other conditions and to evaluate PLBs preoperatively, a computed tomography (CT) scan or magnetic resonance imaging can be used. CT imaging often shows a concentric or oval-shaped, well-defined mass with central calcification, surrounded by peripheral soft tissue. It is especially useful in discerning PLB from teratoma and fibroma, in which situations contrast enhancement is achieved on CT. In patients with cancer history, the radiologist may proceed with more elaborate imagistic techniques. Allam et al. reported the use of positron emission tomography with 2-deoxy-2-[fluorine-18] fluoro-d-glucose integrated with computed tomography (18F-FDG PET/CT) in differentiating PLB from metastatic malignancy.

Establishing differential diagnosis with teratomas, desmoid tumors, rhabdomyomas, ovarian metastases, fibromas, echinococcal cysts, tuberculosis, foreign body granulomas, urinary stones, gallstones, appendicitis, or calcified lymph nodes is important, because it guides the surgeon in choosing the most appropriate treatment. Small asymptomatic PLB can be left untreated, while PLBs bigger than 5 cm are prone to cause chronic abdominal pain or other clinical manifestations and are removed surgically. In our case, a definite diagnosis could not be established preoperatively and, even if it was asymptomatic, in order to meet renal transplantation criteria and to exclude a potential malignancy, PLB’s removal was preferred.

Conclusion

To meet eligibility criteria for renal transplantation, a thorough clinical and paraclinical examination is performed to exclude the presence of malignity in end-stage renal disease patients. Existence of PLB, though rare, may become an impediment in achieving the management of renal chronic failure. Its surgical removal is recommended.

Declaration of conflicting interests

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Ethical approval

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Informed consent

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References

1. Sewkani A, Jain A, Maudar KK, et al. “Boiled egg” in the peritoneal cavity—a giant peritoneal loose body in a 64-year-old man: a case report. J Med Case Rep 2011; 5: 297.
2. Lee KH, Song MJ and Park EK. Giant peritoneal loose body formation due to adnexal torsion. J Minim Invasive Gynecol 2016; 24(2): 189.
3. Sahadev R and Naggappa PK. A case of peritoneal free floating calcified fibromyoma. J Clin Diagn Res 2014; 8(5): ND03–ND04.
4. Suganuma I, MorI T and Takahara T. Autoamputation of a pedunculated, subserosal uterine leiomyoma presenting as a giant peritoneal loose body. Arch Gynecol Obstet 2015; 291(4): 951–953.
5. Cooke WR and Kirk E. Medically managed tubal ectopic pregnancy presenting as a peritoneal loose body. BMJ Case Rep 2015; 2015: bcr2015211871.
6. Zhang, Ling YZ, Cui MM, et al. Giant peritoneal loose body in the pelvic cavity confirmed by laparoscopic exploration: a case report and review of the literature. World J Surg Oncol 2015; 13: 118.
7. Hedawoo JB and Wagh A. Giant peritoneal loose body in a patient with haemorrhoids. Trop Gastroenterol 2010; 31(2): 132–133.
8. Kosam S, Kujur P and Mire V. “Peritoneal mice” a peritoneal loose body in pelvic cavity of 70 years old man, an incidental finding—a case report. Int J Sci Res 2017; 6(1): 2366–2369.
9. Mohri T, Kato T and Suzuki H. A giant peritoneal loose body: report of a case. Am Surg 2007; 73(9): 895–896.
10. Makineni H, Thejeswi P, Prabhu S, et al. Giant peritoneal loose body: a case report and review of literature. J Clin Diagn Res 2014; 8(1): 187–188.
11. Patterson DC. Appendices epiploicae. N Engl J Med 1933; 209: 1255–1259.
12. Han SB, Reynolds TB and Fong TL. Nephrogenic ascites. Analysis of 16 cases and review of the literature. Medicine 1998; 77(4): 233–245.
13. Abbey RK and Chandel UK. Large intraperitoneal loose body causing strangury. J Surg Pak 2012; 17(4): 172–173.
14. Gayer G and Petrovitch I. CT diagnosis of a large peritoneal loose body: a case report and review of the literature. Br J Radiol 2011; 84: e83–e85.
15. Allam T, Muzaffar R, Nguyen N, et al. Peritoneal mouse as detected on 18F-FDG PET-CT. Front Oncol 2013; 3: 83.
16. Takayama S, Sakamoto M and Takeyama H. Clinical challenges and images in GI. Image 1: huge peritoneal loose body in the pelvic cavity. Gastroenterology 2009; 136(2): 404, 730.
17. Jang JT, Kang HJ, Yoon JY, et al. Giant peritoneal loose body in the pelvic cavity. J Korean Soc Coloproctol 2012; 28(2): 108–110.
18. Kim HS, Sung JY, Park WS, et al. A giant peritoneal loose body. Korean J Pathol 2013; 47(4): 378–382.