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Case report

Uterine leiomyosarcoma as a rare cause of fatal retroperitoneal haemorrhage due to compression and rupture of the renal vein

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ABSTRACT

Abnormal uterine bleeding (AUB) can be caused by uterine tumors, both benign and malignant, although they rarely lead to fatal vascular complications. We present a case of a woman found dead in her home, in which the autopsy highlighted a massive retroperitoneal haemorrhage extending to the left renal lodge and a voluminous uterine tumor of stony consistency and exophytic growth weighing 2250g. The histopathological examination identified the mass as a leiomyosarcoma and demonstrated the presence of a dilation with rupture of the left renal vein, in the absence of neoplastic cells infiltrating the vascular wall.

On these findings, it was concluded that the neoplastic mass had caused external compression of the left renal vein, leading to its rupture and to a fatal retroperitoneal haemorrhage. The case was considered of interest as it involved a peculiar etiopathogenetic mechanism causing a lethal complication from uterine leiomyosarcoma which has not yet been reported in the literature.

1. Introduction

Uterine tumors, both benign and malignant, can cause abnormal uterine bleeding (AUB), rarely leading to fatal vascular complications. They include vascular rupture, thrombosis, and embolism. Tumor cells can invade blood vessels, erode walls, or spread through circulation. Compression of major vessels like the inferior vena cava can lead to edema and thrombosis. This increases the risk of venous insufficiency, pulmonary embolism, and deep vein thrombosis. Uterine leiomyoma has been reported as a rare cause of retroperitoneal haemorrhage[1,2] following the spontaneous rupture of one of its superficial veins.[3] The origin of this complication lies in the congestion of the pelvic venous system, for example due to menstruation, pregnancy, abdominal trauma, intense physical exercise, that lead to an increase in abdominal pressure.[3] Most frequently, AUBs are associated with malignant tumors of the uterus such as cervical cancer, uterine sarcoma or ovarian cancer. Regarding uterine sarcomas, these are rare malignant tumors,[4] which amount to <2 cases/100,000 inhabitants (3–7% of uterine tumors[5] and 1–3% of female genital tract tumors[6]). They originate from the mesenchymal tissues of the uterus, including the endometrial stroma, uterine muscle and supporting tissue.[7] Therefore, proving to be heterogeneous in nature, they are histologically classified into leiomyosarcomas (LMS-60 %), sarcomas of the endometrial stroma (ESS-10-15 %), low-grade (LG) and high-grade (HG) sarcomas, undifferentiated sarcomas (UUS-5-10 %) and adenosarcomas (AS-10 %).[8] Signs and symptoms of these tumors are variable, often overlapping with those of benign tumors. Thus, surgical intervention and histopathological examination are required to define their nature and staging, based on hypercellularity, nuclear atypia, high mitotic rate, infiltrating margins and necrosis. LMS is the most common subtype of uterine sarcoma, constituting 1%–2% of the most invasive uterine neoplasms.[7] It occurs more frequently in peri/postmenopausal women over 40 years of age, with abnormal vaginal bleeding in 56 % of cases, palpable pelvic mass in 54 % and/or pelvic pain in 22 %.[5,9] Other symptoms include abdominal swelling, and in advanced cases, systemic issues like fatigue and weight loss. LMS originates from the smooth muscles of the walls of the uterus, being able to develop internally and/or externally to the uterus, reaching a considerable volume, with average diameters of approximately 10 cm. Only 25 % of cases measure less than 5 cm, although a giant uterine leiomyosarcoma weighing 57 kg has been described in the literature.[4] LMS usually present itself with a soft, fleshy, necrotic-hemorrhagic surface, but can also present a very hardened consistency, when

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calcified.[10] Management of uterine LMS requires surgery, and less frequently radiotherapy and chemotherapy, with early diagnosis critical for prognosis. Symptoms often overlap with uterine fibroids, and MRI is key for diagnosis. Treatment typically involves hysterectomy, with close follow-up due to high recurrence rates. They are associated with an unfavorable outcome due to local and systemic complications after distant spread.[11]

In this paper, the Authors report a unique case of fatal vascular complication caused by rupture due to compression of the left renal vein by a voluminous uterine leiomyosarcoma with exophytic growth, with the onset of massive retroperitoneal haemorrhage.

1.1. Case report

An 84-year-old woman was found dead in her home where she lived alone and without any family members. The forensic medical inspection was not requested by the judicial authority and no information was known about health condition or when the woman was last seen or heard alive.

To clarify the cause and manner of her death, an autopsy was ordered, performed 6 days after the body was found.

1.2. The autopsy

The corpse was in a good state of preservation and overweight (weight 78.0 kg, length 168 cm - BMI = 27.64 Kg/m^2). On external examination, the body showed a general paleness of the skin, without external signs of injury.

Upon dissection, a massive retroperitoneal haemorrhage extending to the left renal lodge was documented (Fig. 1A-B) beyond an abdominal aortic aneurysm (circumference of 25 cm), without evidence of macroscopic ruptures of the arterial wall. Other findings included pale organs, atherosclerotic arterial disease, pulmonary edema and congestion and heart of increased volume (weight 456 g – longitudinal diameter 14 cm, transverse 15 cm and antero-posterior 3 cm), with undamaged coronary arteries.

The uterus, weighing 2450 g, showed nine pinkish globose formations, with an irregular surface and a stony consistency, the largest of which was 15 cm (longitudinal diameter) x 11 cm (transversal diameter) x 9 cm (antero-posterior diameter). The nine globular formations were connected to each other in a single mass weighing 2250 g and, through a 4.5 cm long peduncle, were connected to the left side of the uterus (Fig. 1C). There was also a mass with exophytic growth, starting from the uterine wall. After cutting the mass, performed only with an electric saw, they all had a yellowish-white internal content, uniformly stratified and of stony consistency (Fig. 1D).

When the uterine cavity was opened, no other masses were documented, nor were secondaries detected in the remaining viscera, similarly to the peritoneal wall. Upon macroscopic exploration, the main venous and arterial branches of the abdominopelvic vascular tree appeared with intact walls, although in the presence of abundant coagulated blood, adherent and difficult to remove near the left renal vein.

1.3. Histological examination

The main viscera, the uterus-neoformation block and the abdominopelvic vascular tree (including the aortic aneurysm and the renal veins and arteries) were removed and subjected to histopathological investigation using post-fixation techniques. From the uterine masses of stony consistency, fragments were obtained which had been preliminarily subjected to decalcification in 14 % hydrochloric acid for approximately 4 months. For the fragments of uterine mass and the left renal vein, serial sections stained with basic H&E were prepared. They have also been carried out Masson's Trichrome staining according to Goldner and Giemsa was also performed for the uterine mass and Weigert's Resorcin-Fuchsin staining for the renal vein.

Microscopic observation in several points of the uterine mass highlighted a richly cellular connective tissue neoplasm with large calcified and sclerotic areas, whose histological morphology showed elongated cells attributable to smooth muscle proliferation. The residual cellular areas located at the margins of the calcified zone showed high cellularity and marked cellular atypia, with a low mitotic index, morphological elements indicative of the diagnosis of leiomyosarcoma (Fig. 2A).

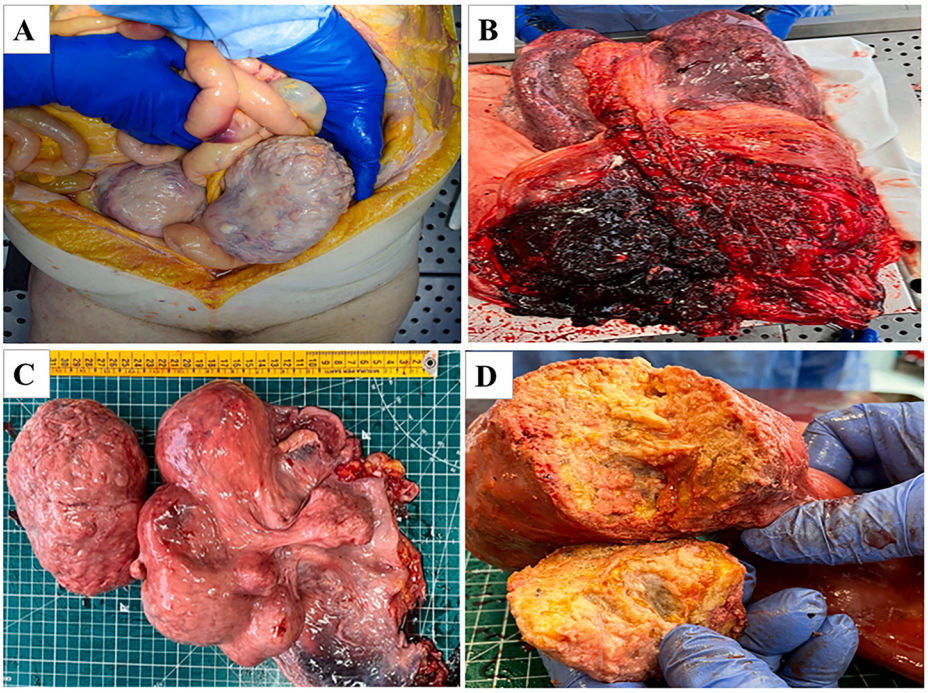

Fig. 1. In A, exploration of the pelvic excavation with evidence of a voluminous uterine mass; in B, massive retroperitoneal haemorrhage *in situ* and after evisceration; in C, macroscopic views of the uterus-ovarian-neoformation block after evisceration; in D, larger mass (15x11 × 9 cm) in its entirety after resection and cut.

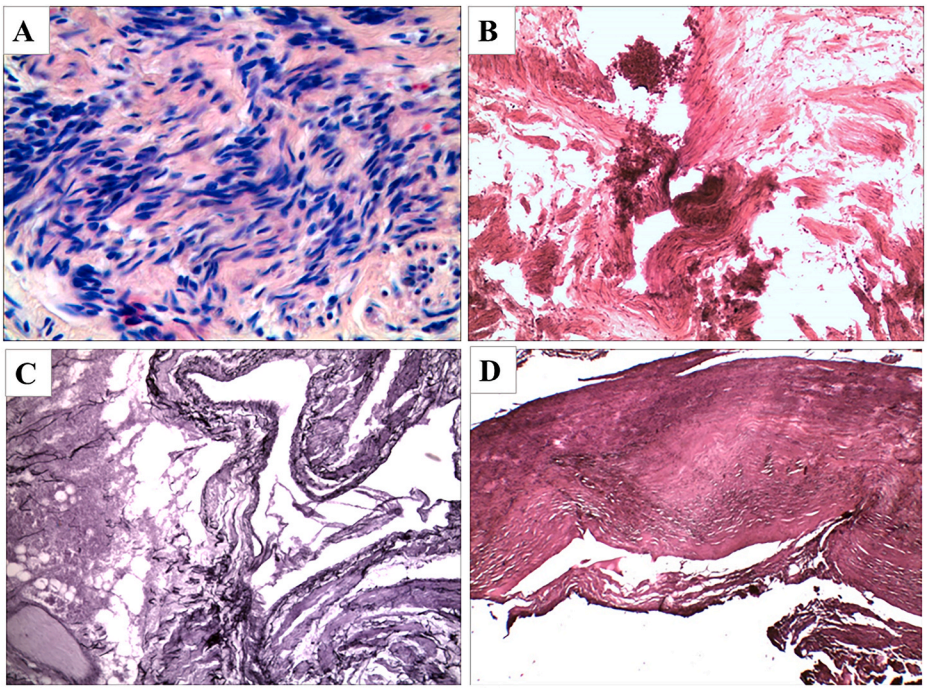

Fig. 2. In A, microscopic view of the neoplastic proliferation with high cellularity and histological features of smooth muscle cells with marked cellular atypia (Giemsa, 400x); in B and C, microscopic views of the left renal vein with evidence of the full-thickness interrupted muscle wall (B, H&E 100x) and of the portion immediately before the rupture (C, Resorcin Fuchsin of Weigert 50x), in which the wall of the renal vein appears dilated and fragmented with large empty spaces, fragmentation of the elastic fibers and disordered arrangement of both isolated and combined bundles, next to an extensive acute hemorrhagic infiltrate; in D, microscopic view of the aortic aneurysm wall completely sclerotized with area of dissociation and microcalcifications, without evidence of wall laceration or blood infiltration (H&E, 32x).

Microscopic observation of serial sections of the left renal vein showed an acute hemorrhagic infiltrate invading and dissociating the muscle wall, communicating with the bleeding present outside the vascular wall (Fig. 2B). Within the area of dissociated muscles, the elastic fibers appeared fragmented, irregularly arranged and with small residues of elastic fibers separated from the wall and incorporated into the hemorrhagic infiltrate dissociating the muscle wall. (Fig. 2C). No invasion by tumor cells was found inside the vessels and in the thickness of the wall. The microscopic examination conducted on the aortic aneurysm did not highlight any area of laceration of the walls or blood infiltration, therefore appearing intact (Fig. 2D). Similarly, the left renal artery also appeared intact.

At the end of all the investigations, the concordance of the autopsy and laboratory findings made it possible to identify the cause of death as massive retroperitoneal haemorrhage due to rupture of the left renal vein arising from the compression by a voluminous uterine leiomyosarcoma with exophytic growth.

2. Discussion

The abdominopelvic venous vascular system may present pathological alterations, with vessels affected by dilations resulting from stenosis, occlusions or compressions that could conduct to ruptures.[12] These situations constitute a serious risk to life, causing haemorrhages, also with lethal haemodynamic alterations. According to the literature, among the main causes underlying these conditions there are vascular malformations and arteriovenous fistulas. Regarding the hypothesis of vascular malformations, in this case no evidence emerged; furthermore, they typically fit into the framework of genetic syndromes (such as the Klippel-Trenaunay-Weber syndrome) typically characterized by a vast procession of further viscero-skeletal anomalies, absent in the present case.[13,14] Nor was there any evidence of duplicated or abnormal renal veins. However, as regards arteriovenous fistulas, the literature[15] describes the possibility of forming a fistula connecting the aorta and the left renal vein (ALRVAF), generally deriving from the rupture and confluence of an aortic aneurysm in the vein left renal. In this case, the autopsy finding of an abdominal aortic aneurysm initially gave rise to the suspicion that it might be an arteriovenous fistula with consequent bleeding from the ruptured renal vein due to the increased blood pressure inside the vessel. However, macroscopic and microscopic examinations of the vascular tree did not reveal the presence of any continuity in the wall of the abdominal aorta nor, in particular, of the aneurysm. For this reason, the hypothesis that the origin of the retroperitoneal haemorrhage was a fistulous communication between the aneurysm and the left renal vein has not found any foundation.

Other known causes of rupture of the left renal vein include direct trauma to the kidney, upper urinary tract or abdomen, particularly high-energy blows. The rupture may be primary (directly caused by the trauma) or secondary to external crushing or compression. In addition, blunt or penetrating trauma (e.g. from car accidents, sports injuries, gunshot or stab wounds) can also damage the renal vessel. Surgery on the kidney or in the pelvic area (e.g. nephrectomy, tumor resection, gynecological or proctological procedures) can also damage the renal veins. A rupture can also be caused by postoperative scarring, which impairs the resistance of the renal vein 16,17. In the present case, there was no possible traumatic cause (no soft tissue or skeletal injury) to justify the massive retroperitoneal haemorrhage, nor any evidence of previous or recent abdominal or pelvic surgery.

In addition, infections and chronic inflammation can also increase the risk of renal vein rupture, especially if they cause thrombosis or vascular inflammation. These diseases include acute or chronic pyelonephritis, renal vein thrombosis and vasculitis. In addition, blood disorders that affect the blood's ability to clot can promote the formation of clots or bleeding in the renal vein. These disorders include nephrotic syndrome and the use of anticoagulant medication.[17] There was no evidence that problems of this nature could have been present in this case,

in particular there was no evidence of an inflammatory infiltrate in the thickness of the vessel wall and no other signs of infection.

Finally, the literature reports that diseases that lead to increased venous pressure in the renal system or in the large veins (e.g. inferior vena cava) can cause renal vein rupture. These include portal hypertension, venous stasis or other central venous abnormalities, such as compression by enlarged lymph nodes or tumors.[12] The literature reports in particular that pelvic tumors, especially when reaching large dimensions, can compress venous vessels, including the renal vein, inducing an increase in blood pressure and focal or diffuse vascular dilatation which can evolve into sudden rupture.[18] In 85 % of cases of dilated venous vessels due to compression from pelvic neoplasia, the origin is located in the venous vessels that supply the neoplastic masses, with an increase in pressure upstream, which can be reflected in the renal veins. The latter, especially in large tumors, can be directly compressed from the outside.[19–26] This has been reported to occur in malignant tumors such as mesenteric, gestational trophoblastic, ovarian and uterine tumors.[18] For malignant tumors, in addition to a compressive mechanism of the blood vessel, there can also be an infiltration of the vascular wall by the neoplastic cells, with weakening of the same and predisposition to rupture. Compression of the venous vessels can also occur in the case of benign neoplasms - typically uterine with exophytic growth - for which there is only a compressive and non-infiltrative mechanism, which can however lead to ruptures of venous vessels with fatal consequences. This is precisely what was reported by Fassina et al.[27] who presented a peculiar case concerning a 52-year-old woman found dead in her home in a pool of blood. The external examination revealed a wound of 1 cm in diameter in the medial portion of the right leg, which on dissection turned out to be a lesion of a superficial varicosity in continuation with the great saphenous vein. Furthermore, a subversion of the uterus was found due to the presence of a tumor, characterized histologically and immunohistochemically as a leiomyoma. Under these conditions, the authors concluded that the compression of the pelvic veins by the uterine neoplastic mass obstructed the venous return, leading to an increase in venous pressure in the lower limbs, which resulted in the rupture of a superficial venous varicosity and the occurrence of a fatal haemorrhage. After appropriate evaluation and integration of the autopsy and laboratory data, we considered it very likely that an etiopathogenetic mechanism similar to that reported by Fassina et al.[27] also occurred in the present case, albeit in a different venous district. In fact, a voluminous uterine neoplastic mass of a leiomyosarcoma with exophytic growth was found in the pelvis. This mass was found to be almost fixed, both due to its large size and the short peduncle that connected it to the uterus. It also had a very hard consistency, as it was calcified and could not be imprinted by other neighboring anatomical structures. It was therefore quite capable of exerting effective pressure on the left renal vein and compressing it, especially since it was lying on top of it. In addition, the left renal vein is often deeper than the right renal vein, making its compression more likely and more significant in the presence of tumors that invade or compress surrounding structures.[28]

Since the recognition of abnormal hemodynamics facilitates the understanding of pathophysiologic processes,[12] it seems appropriate to consider the following. When blood flow is mechanically impeded, it changes from laminar to turbulent flow and, according to Bernoulli and Poiseuille's laws of hydrodynamics, is characterised by reduced velocity and a consequent increase in wall pressure.[29] An exogenous tumor compression, as the one we strongly suspect, represents an event that would be able to cause hemodynamic abnormalities with a significant increase in pressure on the vessel walls upstream of the compression point itself, as already described in the literature.[24] If compression persists and venous pressure continues to rise, a condition known as venous hypertension develops, which can damage the renal vein wall. This can lead to damage to the endothelium and disruption of the vascular barrier, possibly resulting in rupture and retroperitoneal haemorrhage. We believe that the rupture of a renal vein (with a known diameter of about 1 cm[28]) after such an increase in pressure can fully explain the severe bleeding observed macroscopically, which was also topographically consistent with the left renal vein (it did not run along the median line).

Histologically, an obvious rupture site on the left renal vein, which appeared dilated upstream of it, was directly confirmed. At the site of maximal dilatation, there was marked rarefaction of the elastic fibres, but further upstream there was also evidence of chronic changes with fragmentation of the elastic fibres, a consequence that can occur as a result of vascular degeneration that may be caused by mechanical stimuli such as dilatation.[30] In addition, the section upstream of the vein appeared strongly tortuous, which can have two consequences: blood flow is already slowed by even greater pressure on the walls, and the dilatations are less noticeable precisely because they are very tortuous. In the present case, there was no evidence of collateral circulation. However, it should be noted that these are more difficult to assess in women, as retrograde blood flow into the ovarian vein can only occasionally lead to varicosities of the ovarian and parametrial venous plexus.[12] These findings are not easy to detect, especially in a uterus such as the one examined, which is characterised by anatomical abnormalities associated with the tumor.

Furthermore, no tumor cells were found infiltrating the vessel wall. Thus, all the findings were coherent and highly consistent, leading to the diagnosis that the large neoplastic mass had caused external compression of the left renal vein, leading to its rupture after dissociation and fragmentation of the venous vessel wall, resulting in fatal retroperitoneal haemorrhage.

This case is also important because it shows that in cases of massive bleeding associated with an abdominal aortic aneurysm, the mechanism of cause and effect is not always obvious and that the specific causes need to be investigated. Precisely because it has been shown that examination of the renal veins can play a crucial role for forensic purposes, forensic pathologists need to be aware of this and know how to proceed rigorously. In summary, a procedural protocol could consist of the following points. After incision of the abdominal wall and opening of the abdominal cavity, the renal veins behind the transverse colon and the mesenteric arteries that are in contact with the inferior vena cava must be searched for. After identifying landmarks such as the aorta and inferior vena cava, the surrounding soft tissues must be carefully removed to identify their course from the origin to the renal hilum. They must then be examined for abnormalities such as dilatation, thrombosis, signs of inflammation or tumors. If you want to examine their integrity macroscopically *in situ*, you can inject saline while clamping the other vessels. In cases where small vessels are involved that cannot be assessed macroscopically, this method is obviously not effective. Also, in cases of massive bleeding with clots, the *in situ* examination is very complex and it is better to remove the entire abdominal vascular block to keep the relationships between the different anatomical structures. The specimen can then be examined with targeted histological analyses after appropriate fixation in formalin.

3. Conclusion

The case was considered of interest due to the rarity of the neoplasm involved, its size and the resulting fatal complication, which to date has not yet been reported in the literature. Unfortunately, the absence of family members and health information does not allow for further clinical considerations but it is clear that the severity of the disease found is the result of a neglected and untreated disease. This case demonstrates that similar scenarios are still possible today despite the growing importance of prevention campaigns. In this context, the anatomo-pathological results observed are crucial as, by highlighting a peculiar etiopathogenetic mechanism of lethal complication, they enrich the knowledge on uterine leiomyosarcoma.

Consent to participate

The authors declared that all the investigations were carried out accordingly to the Italian Law.

Ethics approval

This article does not contain any studies with living human participants performed by any of the authors. All the studies conducted followed the guidelines provided by Legislation and the National Bioethical Committee and guidelines by Helsinki Declaration. Moreover, the subject involved in this study underwent a judicial autopsy at the Institute of Legal Medicine of Milan in order to identify the cause of death. Data collecting, sampling and subsequent forensic analysis were authorized by the public prosecutor.

Consent for publication

All the authors agree for publication. In this case, the person lived alone and had no family. We anonymised the woman's personal information as much as possible and avoided photographs of her face. Publication of data is allowed when the forensic case has been closed, but the anonymity of the subject must be guaranteed.

Availability of data and material

All the data have been reported in the manuscript.

Code availability

(software application or custom code) Not applicable.

Authors' contribution

GL and ST equally contributed to this work. They devised the project and the main conceptual idea of the article, collected data, drafted the manuscript and performed literature research. SA and GG contributed to the investigation and methodology; GG also contributed to review and edit the manuscript. RZ guarantor of the project and directed the study, devised the main conceptual idea of the article.

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Conflicts of interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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