presence of immunoreactive epithelial markers (cytokeratin, epithelial membrane antigen), and ultrastructural elements characteristic of the epithelium (desmosomes, tonofilaments) in mesenchymal areas.[7] Others believe that the cell lines have different origins and that these are two independent malignant tumors invading each other (a collision of tumors).[8] This has caused confusion with the terminology. The WHO defines sarcomatoid carcinoma as a malignant tumor with epithelial and mesenchymal differentiation morphology and/or immunohistochemistry, and specifies in the diagnosis the presence or absence of heterologous elements.[9] Some authors consider carcinosarcoma and sarcomatoid carcinoma separate entities, and reserve the former term for cases with heterologous mesenchymal elements. Others argue that the differential histologic details lack clinical significance, and group all these tumors in one entity called "sarcomatoid carcinoma". This is the current trend reflected in the WHO classification.[10] Radical cystectomy, when feasible, seems to be the choice treatment, but many patients develop local relapses or distant metastases. Adjuvant radiation and chemotherapy have been used with varying degrees of success.[4] Prognosis is unfavorable in most cases, and the 5-year survival is 20%. The most influential prognostic factor is stage.[2] In sum, carcinosarcoma is an aggressive tumor with a complex histology and poor prognosis. The scarcity of published cases makes it difficult to establish a standardized treatment.

REFERENCES

1. Arguelles Salido E, Travado Soria P, Perez Espejo MP, Rodriguez Corchero J, Medina Lopez RA, Pena Outeirino JM. Carcinosarcoma vesical: Análisis de nuestra serie y revisión de la literatura. Actas Urol Esp. 2004;28:262-8.
2. Wick MR, Swanson PE. Carcinosarcomas: current perspectives and an historical review of nosological concepts. Semin Diagn Pathol. 1993;10:118-27.
3. Lopez-Beltran A, Pacelli A, Rothenberg HJ, Wollan PC, Zincke H, Blute ML, et al. Carcinosarcoma and sarcomatoid carcinoma of the bladder: clinicopathological study of 41 cases. J Urol. 1998;159:1497-503.
4. Baschinsky DY, Chen JH, Vadmal MS, Lucas JG, Bahnson RR, Niemann TH. Carcinosarcoma of the urinary bladder--an aggressive tumor with diverse histogenesis. A clinicopathologic study of 4 cases and review of the literature. Arch Pathol Lab Med. 2000;124:1172-8.
5. Perret L, Chaubert P, Hessler D, Guillou L. Primary heterologous carcinosarcoma (metaplastic carcinoma) of the urinary bladder: a clinicopathologic, immunohistochemical, and ultrastructural analysis of eight cases and a review of the literature. Cancer. 1998;82:1535-49.
6. Li Y, Outman JE, Mathur SC. Carcinosarcoma with a large cell neuroendocrine epithelial component: first report of an unusual biphasic tumour of the urinary bladder. J Clin Pathol. 2004;57:318-20.
7. Torenbeek R, Blomjous CE, de Bruin PC, Newling DW, Meijer CJ. Sarcomatoid carcinoma of the urinary bladder. Clinicopathologic analysis of 18 cases with immunohistochemical and electron microscopic findings. Am J Surg Pathol. 1994;18:241-9.
8. Thompson L, Chang B, Barsky SH. Monoclonal origins of malignant mixed tumors (carcinosarcomas). Evidence for a divergent histogenesis. Am J Surg Pathol. 1996;20:277-85.
9. López Beltrán A, Sauter G, Gasser T, Hartmann A, Schmitz-Dräger H. Infiltranting urothelial carcinoma; WHO classification of noninvasive papillary urothelial tumors. World Health Organization classification of tumors. In: Eble JN, Epstein JI, Sesterhenn I, editors. Pathology and genetics of tumors of the urinary system and male genital organs. Lyon: IARCC; 2004.
10. Picazo ML, Regojo RM, Gonzalez-Peramato P. Variantes histológicas del carcinoma urotelial con implicaciones diagnósticas, pronósticas y terapéuticas. Actas Urol Esp. 2007; 31:989-1001.

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High grade dedifferentiated liposarcoma. Case report and review of the literature

Liposarcoma desdiferenciado de alto grado de cordón espermático. A propósito de un caso

Dear Editor,

Paratesticular neoplasms originate in the spermatic cord and the testicular adnexa, and tend to be well differentiated. One third of cases are malignant, and are mostly sarcomas (liposarcoma is one variant).

We present the case of a 61-year-old patient with an unremarkable history who complained of a painless inguinoscrotal mass for three years; the mass was indurated, did not transiluminate, and was independent from the testicle. No lymphadenopathies were palpated. An abdominal pelvic CT scan showed a 5-cm mass pending from the spermatic cord;

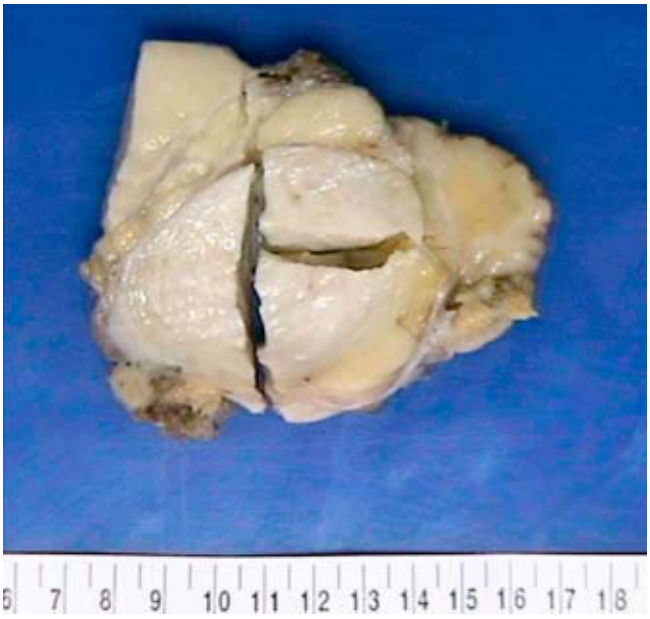

Figure 1 – Removed paratesticular mass; gross appearance.

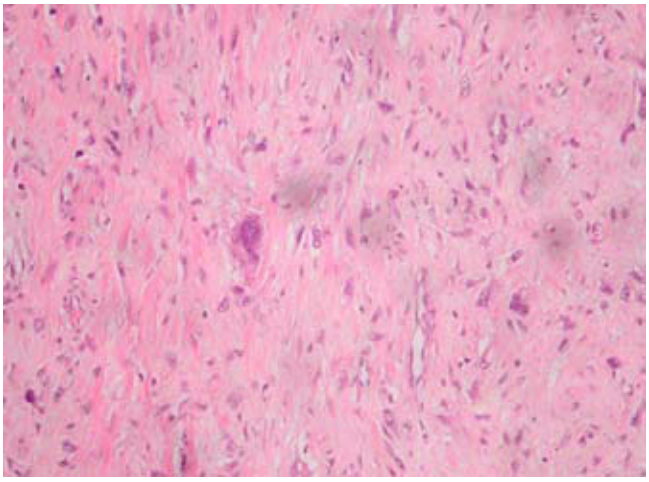

Figure 2 – Hematoxylin-eosin stain.

it was solid, did not uptake contrast, and was well-delimited. No significant inguinal or pelvic lymphadenopathies were observed. The liver and lungs were unremarkable. Tumor markers were: carcinoembryonic antigen 0.8 ng/mL, alpha-fetoprotein 2.6 ng/mL, PSA 0.79 ng/mL. An inguinal radical orchiectomy that included the mass was done.

The anatomopathological examination (fig. 1) showed a white-yellowish, multilobular, encapsulated, well-delimited mass that measured 9×7×7 cm. The microscopic report (fig. 2) described a malignant mesenchymal lesion with several differentiation patterns including lipomatous areas with lipoblasts and an elevated Ki-67 labeling index. There were areas with fibrosarcomatous, myxofibrosarcomatous, neural, and leiomyosarcomatous appearance. It was positive for actin, desmin, vimentin, and S-100. The surgical borders were negative. All this led to the diagnosis of high-grade dedifferentiated spermatic cord liposarcoma. Twenty-one months after surgery, the patient is asymptomatic and has not experienced local or distant relapse.

Paratesticular tumors are the most common neoplasms derived from the spermatic cord. One third are malignant, the most frequent being sarcomas.[1]

Liposarcoma is a soft tissue malignant tumor that develops in adulthood (between the fifth and seventh decade of life), most commonly in the retroperitoneum. It accounts for 16-18% of soft tissue sarcomas, and includes five histologic subtypes: well differentiated, lipoblastic, fibroblastic, myxoid/round cell, and pleomorphic.[2] Only 4% of liposarcomas present with metastases at the time of diagnosis. Paratesticular liposarcoma represents 3-7% of paratesticular sarcomas.[3,4] Although paratesticular liposarcomas tend to be well-differentiated, occasionaly there is a process of dedifferentiation or progression to a higher grade.[2,5]

Dedifferentiated liposarcomas are defined as primary or recurring tumors with a well-differentiated liposarcoma that is associated with, or progresses to, high-grade non-lipogenic sarcoma. The presence of atypical cells, hyperchromatic nuclei, lipoblasts, and fibrous septa distinguish them from lipomas. This type of dedifferentiated tumor, in turn, may present a great variety of histological patterns, the most common being medium-high-grade myxofibrosarcoma. Immunohistochemical analysis is essential to reach a diagnosis; protein S-100, desmin, vimentin, and protein CD34 are expressed. This type of tumor tends to be located in the retroperitoneum, extremities, spermatic cord, and rarely in the head or neck.[2]

This type of tumor presents as an irregular inguinoscrotal mass that does not transiluminate; it is occasionally associated with reactive hydrocele.[2] Differential diagnosis must be made with inguinal hernia, cyst in the cord, hematocele, hydrocele, tubercular orchiepididymitis, and testicular tumors. Echographically it appears as a solid, hyperechoic and heterogeneous lesion. CT shows a septated lesion that is hypointense compared to the subcutaneous fat, and permits to find pelvic or retroperitoneal lymphadenopathies.[6] The study can be completed with a chest X-ray and an inguinoscrotal MRI.

Due to the rarity of these tumors,[7] information about treatment and survival is based only on retrospective reviews of small series of patients. The only consensus is on the importance of removing all the affected tissue (radical orchiectomy with ample local resection of the surrounding soft tissue), as one of the most important prognostic factors is the existence of negative surgical margins.[8] Lymphadenectomy has not shown to improve survival.[4]

Dedifferentiated liposarcomas have a poorer prognosis than well-differentiated ones, but are less aggressive than high-grade sarcomas. Local recurrence can develop in the scrotum, the inguinal area, or the pelvis. Due to the lack of adequate studies, the role of radiation and chemotherapy is still controversial.[8]

Positive margins, inguinal location, nuclear grade and degree of differentiation are associated with the risk of recurrence or progression.[8] Overall 5-year survival of liposarcomas is 70%. The mean disease-free time is 36 months (6 months-16 years).[4] According to published series, 19.4% of

cases suffer local recurrence, 11.1% distant metastasis, and 5.5% pelvic nodal relapse.[8]

REFERENCES

1. Coleman J, Brennan MF, Alektiar K, Russo P. Adult spermatic cord sarcomas: management and results. Ann Surg Oncol. 2003;10:669-75.
2. Peyrí E, Urban A, Martínez M, Sanmarti B. Liposarcoma dediferenciado del cordón espermático: degeneración de un lipoma previo. Actas Urol Esp. 2003;27:383-6.
3. Schwartz SL, Swierzewski III SJ, Sondak VK, Grossman HB. Liposarcoma of the spermatic cord: report of 6 cases and review of the literature. J Urol. 1995;153:154-7.
4. Soler J, Zuluaga A, Hidalgo M. Liposarcoma de cordón espermático: aportación de un nuevo caso y revisión de la literatura. Actas Urol Esp. 1999;23:447-54.
5. Henricks WH, Chu YC, Goldblum JR, Weiss SW. Dedifferentiated liposarcoma: a clinicopathological analysis of 155 cases with a proposal for an expanded definition of dedifferentiation. Am J Surg Pathol. 1997;21:271-81.
6. Calahorra F, Pérez C, Ramos A. Liposarcoma paratesticular del cordón espermático. Actas Urol Esp. 1990;3:202-4.
7. Garcia Morua A, Lozano Salinas JF, Valdes Sepulveda F. Liposarcoma of the espermatic cord: our experience and review of the literature. Actas Urol Esp. 2009;33:811-5.
8. Ballo MT, Zagars GK, Pisters PW. Spermatic cord sarcoma: outcome, patterns of failure and management. J Urol. 2001; 166:1306-10.

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Post-renal transplantation intermittent anuria secondary to calcified granuloma of the neomeatus

Anuria intermitente postrasplante renal secundaria a granuloma calcificado del neomeato

Dear Editor,

We report the case of a patient presenting with early ureteral obstruction secondary to a calcified granuloma in the ureterovesical junction after a right heterotopic renal transplantation. This granuloma variant has not been described in literature.

A 34-year-old female with end-stage chronic renal failure secondary to systemic lupus erythematosus diagnosed in 1990 was admitted in the hemodialysis program in the year 2000. She had a history of hypersensitization, hyperparathyroidism secondary to chronic renal failure on treatment with cinacalcet (Mimpara) and calcitriol (Rocaltrol), and HT on treatment with atenolol, doxazocin, and amlodipine.

She was admitted in March 2008 for related living donor renal transplantation (husband). The donor underwent an uneventful laparoscopic left nephrectomy, and a single artery and vein kidney and a well vascularized ureter of the proper length were obtained. As part of the desensitization protocol, the patient received two doses of rituximab, six sessions of pre-transplant immunoadsorption, and three doses of monoclonal gamma globulin and induction with thymoglobulin. Post-surgical immunosuppressor treatment consisted of mycophenolate sodium 750 mg p.o/12 h, tacrolimus 6 mg p.o/12 h, and diminishing regimen of prednisone down to 20 mg p.o/d.

A heterotopic transplantation in the right renal fossa was performed, with termino-lateral anastomosis of the renal vein to the external iliac vein and termino-lateral anastomosis of the renal artery to the external iliac artery with continuous prolene 6 and 7/0, respectively; there was adequate perfusion after declamping, and no complications. A Politano-Leadbetter ureterovesical implantation was done, anchoring the distal end of the ureter with three loose poliglecaprone (Monocryl) 6/0 sutures, with immediate onset of diuresis. Warm ischemia time was 2 min, and total ischemia time was 50 min. A postoperative renal doppler ultrasound was normal, and an isotope renography showed adequate uptake. The patient's kidney function improved; her creatinine level was 0.8 mg/dL when she was released.

The patient was readmitted 14 days later for pain at the level of the graft, anuria, and declining renal function, with a change in creatinine from 0.8 to 2.3 mg/dL. An abdominal ultrasound reported pyeloureteral ectasia up to the ureterovesical anastomosis, where a hyperechogenic image compatible with lithiasis was found. Diuresis was spontaneously reinitiated, and creatinine dropped to 0.5 mg/dL. An abdominal CT reported pyeloureteral ectasia in the kidney graft and ureteral dilatation with an intravesical calcified mass in the area of the ureteral anastomosis of the graft, which could correspond to a hematoma or a calcified