second year, and posteriorly only when indicated.

The patient remained free of disease until October 2001, when a left supraclavicular lymphadenopathy was noted. In addition, CT revealed a 9-cm longitudinal retroperitoneal mass adhered to the abdominal aorta, though no other lesions were observed.

The neck lymph node biopsy study indicated seminoma.

The serum tumor markers proved negative. Treatment was provided in the form of 4 cycles of 20 mg/m^2 of cisplatin via the intravenous route for 5 days, and 100 mg/m^2 of etoposide via the intravenous route on a daily basis for 5 days, repeated every three weeks.

Repeat disease staging after 4 chemotherapy cycles revealed complete remission.

On occasion of the last follow-up visit in June 2009, the patient remained well and without disease relapse.

The introduction of cisplatin-based chemotherapy for the management of GCTs in the late 1970s spectacularly improved the results obtained in patients with tumors of this kind.

However, 10-30% of all patients suffer relapse after initial treatment. Relapse tends to occur in the first two years after treatment, and most such cases are resolved with rescue therapy. Recently, a limited number of patients (about 1-4%) have been found to develop late disease relapse[1-5].

The most common location of relapse is the retroperitoneum, though there have been descriptions of late GCT relapse in the chest, neck, pelvis, brain and liver[3-4].

Baniel et al.[1] reported complete response in only 17 of 65 patients (26%) subjected to chemotherapy, and only two of these individuals were healed with chemotherapy alone. In contrast, 11 of 16 patients (69%) subjected to surgery alone remained disease-free after a median follow-up of 14 months. The authors concluded that surgery is the treatment of choice, and that chemotherapy plays only a marginal role in the management of late disease relapse.

In the analysis published by Dieckmann et al.[6], chemotherapy alone was used to treat 46% of the patients, while 34% received combination surgery and chemotherapy. Eighty-four percent of the patients achieved complete remission after treatment.

Gerl et al.[2] reviewed a series of 25 patients with late relapse and reported a disease-free survival rate of 36% after a median follow-up of 38 months. A total of 20 males underwent chemotherapy for late relapse.

Most of the published series show surgical resection to be associated with improved results, though only modest success has been recorded with chemotherapy alone.

We consider that chemotherapy can be used to treat patients that have not received such therapy before, without the need for surgery, if complete remission is achieved.

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Solitary fibrous tumor of the prostate

Tumor fibroso solitario de la próstata

To the Editor,

A 65-year-old patient presented with urinary obstruction problems for the previous 6 months. Rectal digital exploration of the prostate revealed a gland of fibroelastic consistency, without nodules. The serum total prostate specific antigen (PSA) concentration was 7.52 ng/ml, with a free PSA level of 2.61 ng/ml, and a ratio of 0.35. The abdominal and pelvic ultrasound study confirmed prostate gland enlargement. The clinical diagnosis was grade I-II benign prostate hyperplasia. Suprapubic prostatectomy was carried out based on the

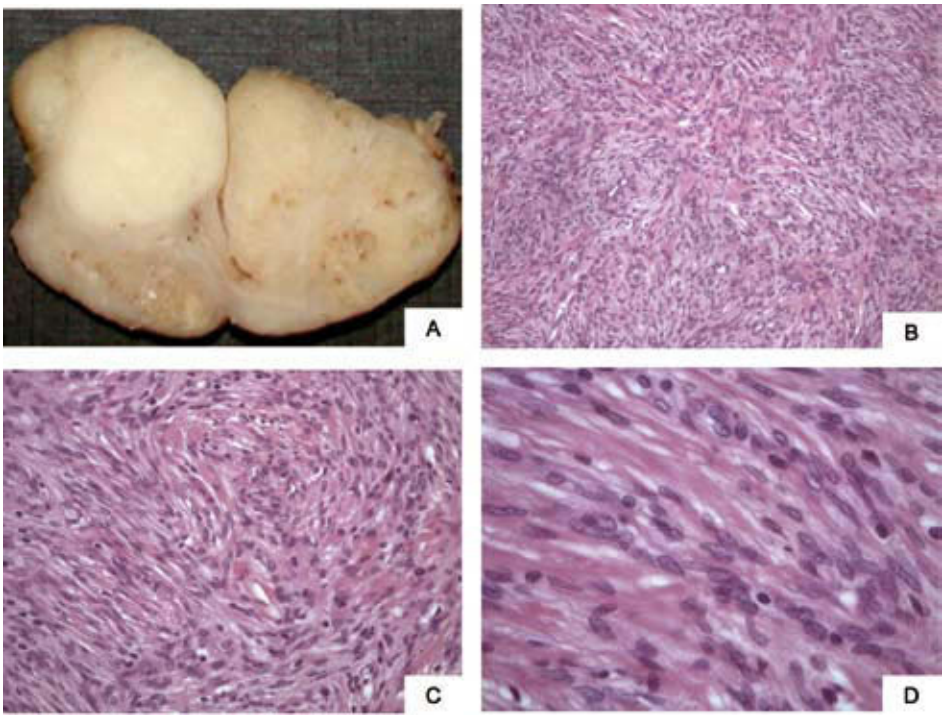

Figure 1 – (A) Macroscopic appearance of solitary fibrous tumor (SFT) of the prostate gland. A well defined tumor is observed, without hemorrhage or necrosis (compare with the prostate tissue adjacent to the tumor). (B) Characteristic histological appearance of SFT, showing hypercellular areas (hematoxylin-eosin [HE] x40). (C) Vascularization consists mainly of small capillaries (HE, x100). (D) The tumor is characterized by a proliferation of randomly distributed fusiform cells with focal intercellular collagen deposits (HE, x400).

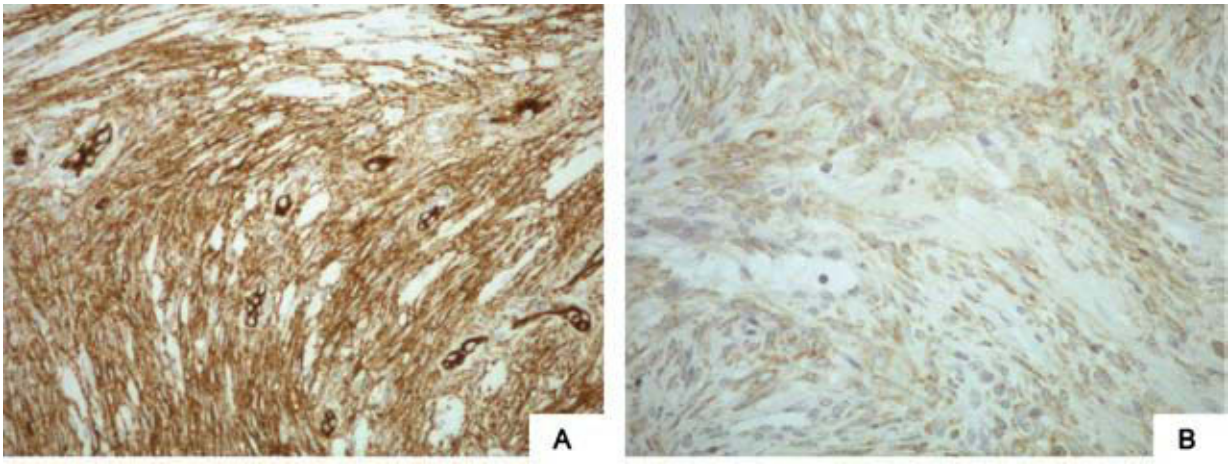

Figure 2 – Immunohistochemical studies. (A), (B) and (C) CD34, BCL-2 and CD99 positivity in the cytoplasm and at plasma membrane level of the tumor cells (diaminobenzidine [DAB], x200). (D) Nuclear expression of Ki-67 (DAB, x200).

Millin technique. The surgical piece was sent for histological evaluation. The adenectomy specimen was fixed in buffered 10% formalin solution and processed as usual for histological and immunohistological study. Macroscopically, the piece was irregular and measured 80 x 65 x 24 mm in size, of a pinkish color with brown areas. Upon sectioning, the surface was found to be multinodular, with a well defined nodule and presenting a whitish-gray, homogeneous appearance.

No bleeding or areas of necrosis were observe (fig. 1). Histologically, the tumor consisted of randomly distributed short fusiform cells, with a rounded or ovoid nucleus, homogeneously distributed chromatin, and an inconspicuous nucleolus (fig. 1). Vascularization consisted mainly of small capillaries. The tumor periphery in turn showed the normal prostate component entrapped by the mesenchymal proliferation. No mitotic figures, bleeding, myxoid changes or areas of necrosis were observed. The neoplastic cells proved immunohistochemically positive for CD34, CD99 and oncoprotein BCl-2 (fig. 2). The cell proliferation rate as determined by the nuclear expression of Ki-67, was 17%.

Solitary fibrous tumor (SFT) is an infrequent prostate neoplasm, with at least 20 cases documented in the literature to date[1]. When the prostate gland is affected, the clinical presentation of SFT is variable, depending mainly on the size of the tumor and the involvement of adjacent structures[2,3]. The previously reported cases presented with lower urinary tract symptoms ranging from dysuria to acute urinary retention[2-4]. Our patient showed no specific symptoms, probably due to the central location of the tumor. As a result of the variability of SFT in the histological study, a range of other tumors must be included in the differential diagnosis. Thus, in the prostate gland, SFT must be distinguished from a series of benign and malignant mesenchymal lesions, though the two most important tumors (considering the prognostic implications) are sarcomatoid carcinoma and gastrointestinal stromal tumor (GIST). Generally, sarcomatoid carcinoma shows atypical histological features, with broad spectrum cytokeratin expression[3]. In turn, GIST expresses c-Kit, which serves to distinguish it from SFT[5].

The origin of prostate SFT is not unknown. Different tissues have been found to contain cells similar to CD34-positive fibroblasts in the mesenchymal component, particularly around vascular and neural structures, etc.[6] A number of CD34-positive lesions, such as dermatofibrosarcoma *protuberans* in the skin[7] or intestinal fibroid polyps[8], develop in locations where there are normal populations of cells similar to CD34-positive fibroblasts. Taking these observations into account, it has been suggested that these lesions may develop from cells similar to CD34-positive fibroblasts. Curiously, in coincidence with our own case, the previous reports describe intense CD34 expression[1-4]. This suggests that the fusiform proliferative component of these tumors develops from CD34-positive interstitial cells in a way similar to the situation suggested for other CD34-positive lesions in other locations.

In conclusion, we have presented a typical case of prostate gland SFT. Local surgery often suffices as treatment. Probably, the histogenetic origin in the prostate is represented by the CD34 fibroblastic cells.

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