**The sick breast lobe has a testicular counterpart**

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**Introduction**

It has been hypothesized that ductal carcinoma in situ (DCIS) of the breast is a lobar disease, meaning that some breast lobes are prone to develop cancer while others are not (1). This “sick breast lobe” theory states that the involved lobe(s) are genetically misconstructed from birth and that further accumulation of genetic changes during the following decades ultimately leads to DCIS presenting in a specific pattern that can be explained by accepting this hypothesis.

The “sick breast lobe” theory does not only propose a mechanism of DCIS development, it also has implications regarding the diagnosis and treatment of DCIS (2).

Until now, the “sick lobe” theory only applies to the breast and there is no data indicating that a similar phenomenon might occur in other organs that show a lobar/lobular structure, such as the testis (3).

We recently encountered a patient with a testicular germ cell tumor presenting in a kind of “experiment of nature” setting, which enabled us to explore the possible existence of the sick lobe in the testicle.

**Methods and Results**

A 31-year-old patient underwent a left orchiectomy in 2012 for a pT1 mixed germ cell tumor, mainly consisting of seminoma, associated with extensive germ cell neoplasia in situ (GCNIS), tubular atrophy and Leydig cell hyperplasia in the residual surrounding parenchyma.

In view of these findings, the patient underwent sonographical screening of the right testis, which showed in Octobre 2018 a lesion of 11 mm in the inferior and a lesion of 7 mm in the superior part of the testicular parenchyma.

Intra-operative frozen section was performed on both lesions, showing that both consisted of pure seminoma. Consequently, a right orchiectomy was performed.

The resection specimen was extensively sampled, but showed no additional invasive tumor; the small seminoma foci were completely removed via the frozen section.

The samples taken randomly from the parenchyma showed several areas consisting of GCNIS, atrophic tubuli and Leydig cell hyperplasia (approx. 30%).

This peculiar setting enabled us to study the pre-invasive neoplastic status throughout the almost whole testicular parenchyma and this can therefore be considered as an “experiment of nature”.

The areas of GCNIS and atrophy were sharply delineated from the normal parenchyma and were distributed in a lobar-like fashion, strongly suggesting that the “sick lobe” theory is also valid in the testicle.

**Conclusions**

We took advantage of the specific setting this patient presented in (“experiment of nature”) to show that GCNIS and testicular atrophy occurred in a strikingly lobar and sharply delineated pattern of distribution throughout the parenchyma, yielding evidence that the sick breast lobe has a testicular counterpart. Further studies on similar patients presenting in this rare setting are warranted.

**Bibliography**