

Quiz

CORRECT ANSWER TO THE QUIZ. CHECK YOUR DIAGNOSIS

CASE REPORT

PROSTATIC PARAGANGLIA-HYPERPLASIA – A HISTOLOGICAL MIMIC OF INTRAPROSTATIC ADIPOSE TISSUE

MARIYA STOILOVA¹, MARIA KOLEVA¹, MARIN BALTOV², ALBENA FAKIROVA³, DORIAN DIKOV⁴

¹Department of General and Clinical Pathology, Medical University of Plovdiv, Plovdiv, Bulgaria

²Department of Forensic Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria

³Department of Pathology, Military Medical Academy, Sofia, Bulgaria

⁴Department of Pathology, Jossigny Hospital, Jossigny, France

Prostatic paraganglia (PP) can simulate adenocarcinoma's fused gland pattern, and a differential diagnostic panel is developed. Their differential diagnosis with benign prostatic lesions has been largely unreported.

Intraprostatic adipose tissue (IPAT) is seen extremely rarely in prostatic specimens, but it must be known, to avoid false positive results for extraprostatic fat carcinomatous invasion in needle biopsy specimens.

We present one case from 100 randomly selected radical prostatectomy specimens in which our diagnosis evolved from IPAT to PP-hyperplasia, after additional immunohistochemical investigations. The presence of PP-hyperplasia, with both parenchymal and neurovascular bundle location, and its differential diagnosis with IPAT has not been previously reported.

Introduction

Intraprostatic paraganglionic tissue is seen in 10% of radical prostatectomy (RP) specimens [1]. As observed in other localisations, PP is characterised by small, solid nests of cells with clear or amphophilic cytoplasm, often with a “zellballen” arrangement and a delicate background network of capillaries [2]. Prostatic paraganglia-tissue can simulate the fused gland pattern of adenocarcinoma (Gleason 4), and a differential diagnostic panel is developed [2, 3]; in contrast, their differential diagnosis with benign prostatic lesions has been largely unreported.

Although extremely rare, IPAT exists, and this deviation from the norm must be known by the pathologist to avoid false positive results for extraprostatic fat carcinomatous invasion (pT3) in needle biopsy specimens [4–7].

Following the discovery of intra-prostatic fat in a patient, (unpublished data), we reviewed 100 randomly selected RP specimens to document the location and frequency of IPAT. Here, we present another case in this series where our diagnosis evolved from IPAT to PP-hyperplasia, after additional considerations and immunohistochemical investigations.

Material and methods

We investigated 100 randomly selected radical prostatectomies specimens, and in one of them our diagnosis evolved from IPAT to PP-hyperplasia, after an additional immunohistochemical examination.

Results

Among all the investigated cases, we present the case of a 63-year-old patient who underwent RP. The pathology result revealed a bilateral acinar prostatic adenocarcinoma Gleason score 7 (4 + 3) (60: 40%), grade group 3 (WHO), without extraprostatic extension; pT2 N0.

The presence, in one of the sections from the peripheral zone of the prostate (right lateral), of a 1 mm periglandular stromal focus with the morphology of IPAT (Fig. 1), without tumour infiltration in it. Histologically, it revealed “a typical brown adipose tissue” morphology, the cells of which show multivacuolar cytoplasm and a rounded, centrally located nucleus (Fig. 2). This focus did not show evidence of stromal alteration or significant inflammatory infiltrate. In another section, in the lateral portion of the peripheral zone of the prostate in close associ-

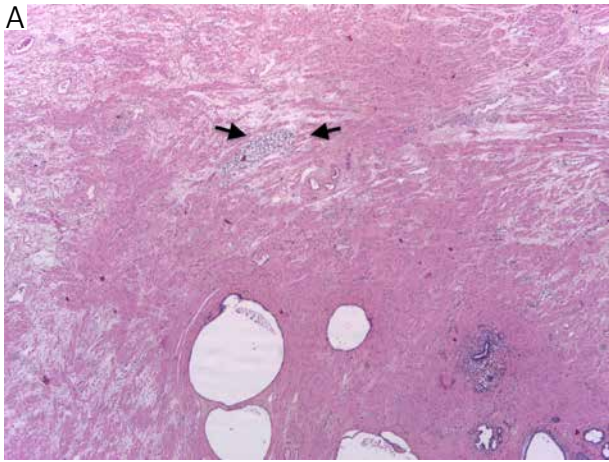


Fig. 1. Periglandular stromal focus showing the morphology of intraprostatic brown adipose tissue (arrows). Haematoxylin-eosin-saffron stain 50×

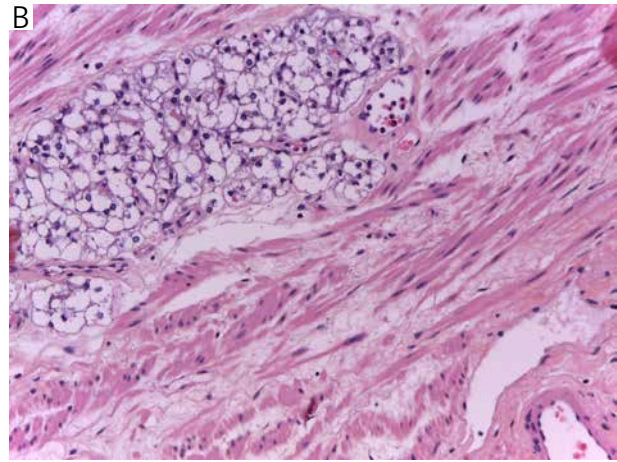


Fig. 2. A lobule composed of cells with multi-vacuolar cytoplasm and rounded, centrally located and enlarged nuclei. Haematoxylin-eosin-saffron stain 200×

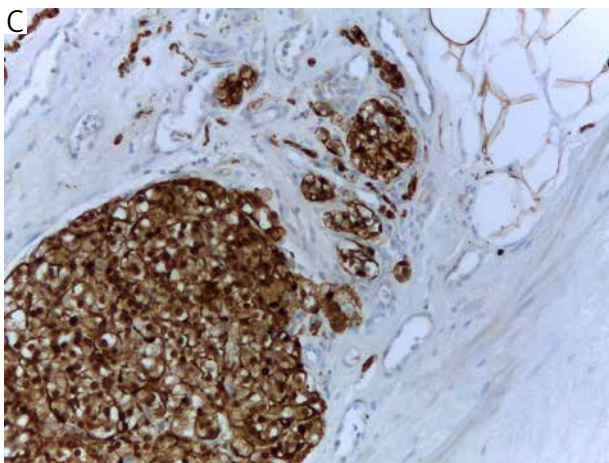


Fig. 3. Positive expression of S100 protein in multivacuolated cells in absence of “sustentacular pattern” and membranous expression in adjacent white adipocytes (right top). Immunohistochemical stain: anti-S100 protein 100×

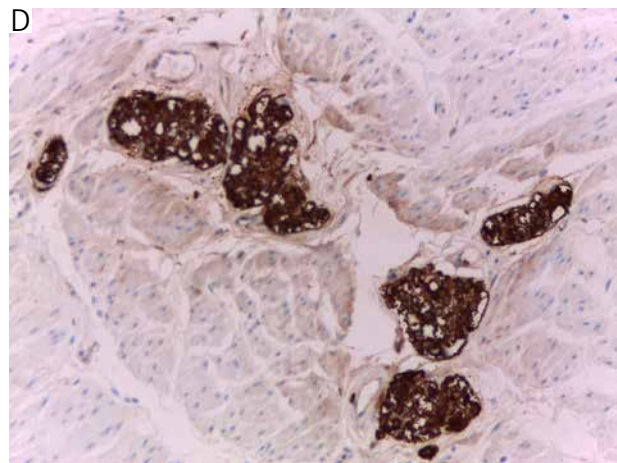


Fig. 4. Strong chromogranin expression in multivacuolated cells in stromal lobules. Immunohistochemical stain: anti-chromogranin 200×

ation with the neurovascular bundle, a second focus of adipose tissue could be seen with a size of 6 mm. About 60% of this microscopic focus showed “brown fat-morphology”. The initial immunohistochemical investigation shows an absence of prostatic-specific antigen expression (data not shown). The S100 protein shows intense, simultaneous nuclear and cytoplasmic expression in the cells with “brown adipocytes morphology” (Fig. 3), and nuclear and membranous in mature white adipocytes; it is not observed the “sustentacular pattern” of S100-expression, characteristic for paraganglia. Additional immunohistochemical examination, however, contrary to the original diagnostic idea, showed that the multivacuolated cells in stromal lobules show strong expression for chromogranin (Fig. 4), synaptophysin and GATA3 (data not shown).

Discussion

Based on these histological and immunohistochemical characteristics, our initial diagnosis was: presence of areas of intraprostatic brown adipose tissue.

A subsequent literature review found a complete lack of such described case, as well as extreme rarity of IPAT, found in 0.66–3.98% of patients on autopsy material, needle biopsy, and RP specimens [4–7]. The intraprostatic adipose tissue is often in small amounts and located peripherally, near the prostate fibromuscular tissue (capsule) [4–7].

This made us question our initial diagnosis and make additional immunohistochemical stains for chromogranin, synaptophysin, and GATA3. All 3 markers turned out to be strongly positive in multivacuolated cells with lobular architecture. Ob-

viously, this necessitated a correction of our initial diagnosis and its replacement with PP-hyperplasia. Paraganglia-hyperplasia [8] is characterised by the presence of an increased number of cellular lobules, which are poorly circumscribed, some confluent, composed of chief cells with hyperchromatic and mildly enlarged nuclei; the “sustentacular pattern” is reduced.

The expression of GATA3 in PP should be known by the pathologist and taken into account in differential diagnosis with prostatic infiltration of urothelial carcinoma [9].

We also investigated CD68, whose expression is negative (data not shown) and could serve in a differential diagnostic plan with xanthogranulomatous prostatitis.

Conclusions

The presence of PP-hyperplasia, with both parenchymal and neurovascular bundle location, and its differential diagnosis with IPAT has not been previously reported. The case described by us expands the diagnostic and differential-diagnostic horizons for morphologists dealing with normal and oncological prostate histology.

Disclosures

1. Institutional review board statement: Not applicable.
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

References

1. Ostrowski ML, Wheeler TM. Paraganglia of the prostate. Location, frequency, and differentiation from prostatic adenocarcinoma. *Am J Surg Pathol* 1994; 18: 412-420.
2. Srigley JR. Benign mimickers of prostatic adenocarcinoma. *Mod Pathol* 2004; 17: 328-348.
3. Kawabata K. Paraganglion of the prostate in a needle biopsy: a potential diagnostic pitfall. *Arch Pathol Lab Med* 1997; 121: 515-516.
4. Cohen RJ, Stables S. Intraprostatic fat. *Hum Pathol* 1998; 29: 424-425.
5. Billis A. Intraprostatic fat : does it exist? *Hum Pathol* 2004; 35: 525.
6. Joshi A, Shah V, Varma M. Intraprostatic fat in a prostatic needle biopsy : a case report and review of the literature. *Histopathology* 2009; 54: 912-913.
7. Nazeer T, Kee KH, Ro JY, et al. Intraprostatic adipose tissue : a study of 427 whole mount radical prostatectomy specimens. *Hum Pathol* 2009; 40: 538-541.
8. Gellert LL. Hyperplasia-paraganglia. Available from: <https://www.pathologyoutlines.com/topic/adrenalparagangliahyperplasia.html> (accessed: 22.10.2022).
9. So JS, Epstein JI. GATA3 expression in paragangliomas: a pitfall potentially leading to misdiagnosis of urothelial carcinoma. *Mod Pathol* 2013; 26: 1365-1370.

Address for correspondence:

Maria Koleva, PhD
 Department of General and Clinical Pathology
 Medical University of Plovdiv
 Plovdiv, Bulgaria
 e-mail: mariya.kolevaivanova@gmail.com