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Pseudocarcinomatous Hyperplasia of the Fallopian Tube: A Lesion not to be Ignored: A Case Report

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Abstract **Case Report**

Pseudocarcinomatous hyperplasia of the fallopian tube is an entity rarely described in the literature. It is generally associated with endogenous or exogenous ovarian stimulation or secondary to underlying inflammation (pelvic inflammatory disease). It can cause a problem of differential diagnosis with adenocarcinoma. We report the case of a 34-year-old woman with secondary infertility who underwent surgery for bilateral hydrosalpinx and polymyomatous uterus. Anatomopathological study revealed granulomatous salpingitis without caseous necrosis, with bilateral tubal pseudocarcinomatous hyperplasia. In this case, we discuss the elements of the positive and differential diagnosis of this tubal lesion.

Keywords: pseudocarcinomatous hyperplasia, Gynecopathology, Fallopian tube, case report.

INTRODUCTION

Pseudocarcinomatous hyperplasia of the fallopian tube, also known as florid tubal epithelial hyperplasia, is a proliferation of the mucosa of the fallopian tube that was included in the World Health Organization (WHO) classification of tumors of the female genital organs in 2003, under the heading of tumor-like epithelial lesions. It is usually seen in young patients between the ages of 17 and 40 [1, 2].

We report the case of a 34-year-old woman who underwent surgery for bilateral hydrosalpinx, the anatomopathological study of which revealed bilateral tubal pseudocarcinomatous hyperplasia.

The aim of this work is to highlight the various elements required to differentiate this rare lesion from a tubal adenocarcinoma.

CASE REPORT

The patient was 34 years old, with a history of two spontaneous abortions at one month of pregnancy, and presented with secondary infertility. Clinical examination was unremarkable.

Pelvic ultrasound revealed a polymyomatous uterus and a left ovary showing a cystic image suggestive of hydrosalpinx.

Hysterosalpingography showed a dilated left fallopian tube, tortuous in its ampullary and fringed portions but permeable.

Bilateral salpingectomy with myomectomy was performed and the operative specimen was sent to our facility for anatomopathological study.

Macroscopic examination revealed two dilated, cystic fallopian tubes with a whitish cross-sectional appearance and endocystic vegetations and mucoid remodelling in one of them (Figure 1).

Microscopic examination showed a tubal lumen containing fibrino-leukocytic debris; a mucosa made up of distorted, hyperplastic and fused bangs, occupying the lumen and not extending beyond the mucosa, giving a pseudo-glandular and cribriform appearance lined by an epithelium with cubic and ciliated cells. These cells showed mild cytonuclear atypia with nuclear stratification in places; the presence of a few mitosis figures; an inflammatory infiltrate in the axis of the bangs and covering the entire wall with several epithelio-giganto-cellular granulomas without necrosis and mesothelial hyperplasia forming papillae in places (Fig 2 and 3).

Figure 1: Macroscopic photo showing a dilated and cystic aspect of the tube

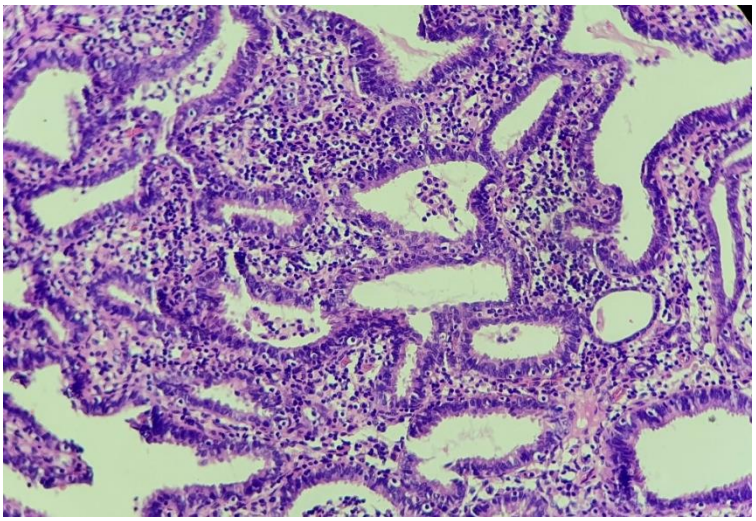

Figure 2: Photomicrograph showing glandular architecture associated with marked inflammation, H&E; 200 x

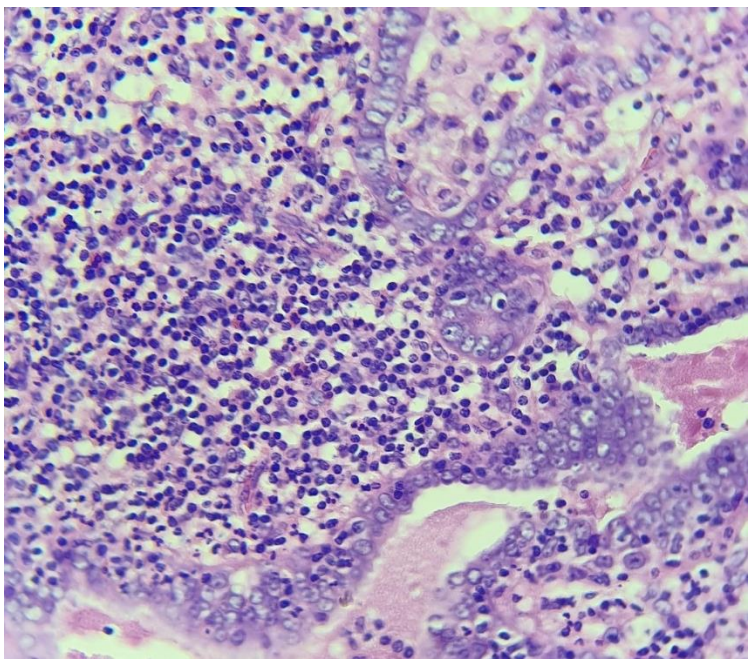

Figure 3: Photomicrograph showing stratified cells; their nuclei are regular with slight atypia and no mitotic figures, H&E; 400x

Immunohistochemistry was performed on both specimens, showing: a low mitotic index with Ki67 in the tubal epithelium and associated labeling of lymphocyte nuclei in the inflammatory infiltrate. Heterogeneous nuclear staining of hyperplastic tubal epithelium with anti -p53 interpreted as non-mutated.

Zhiel staining was performed and found to be negative.

The morphological appearance and immunohistochemical profile were consistent with granulomatous salpingitis without caseous necrosis, featuring bilateral pseudocarcinomatous tubal hyperplasia with reactive atypia. The course was simple with no complications.

DISCUSSION

Tubal epithelial hyperplasia has been reported in association with endogenous or exogenous hyperoestrogenism. It may thus accompany other lesions associated with estrogen stimulation [3], such as endometrial hyperplasia or low-grade endometrioid adenocarcinoma. They can be seen in estrogen-secreting ovarian lesions such as stromal ovarian hyperplasia, estrogen-secreting ovarian tumors (thecomas; granulosa tumor). They are observed in patients treated with tamoxifen, a situation that favours hyperoestrogenism [2]. In a series reported by Stern *et al*., [4], seven of the 39 patients with tubal epithelial hyperplasia lesions had received estrogen.

Some authors suggest an association between tubal epithelial hyperplasia lesions and borderline serous carcinoma of the ovary, given that the epithelium of the tube, uterus and ovary are embryologically linked [5].

This lesion can also occur in the context of chronic tuberculous and non-tuberculous salpingitis. Cheung and colleagues have highlighted the association of various forms of chronic salpingitis, including tuberculous salpingitis, with hyperplastic lesions of the tubal mucosa. However, Moore and Enterline noted the presence of epithelial hyperplasia in 18.5% of surgically resected fallopian tubes, which showed associated PID in 50% of cases [6].

Macroscopic examination shows no specific aspect. Pseudocarcinomatous lesions are usually discovered incidentally. The fallopian tubes may be normal or enlarged. Pyosalpinx or hydrosalpinx may also be observed [2].

Histologically, this lesion is characterized by florid epithelial hyperplasia. Bangs are often fused, resulting in a gland-like appearance or cribriform architecture. Tubal epithelium shows mild to moderate nuclear atypia, loss of polarity, nuclear stratification and some mitotic figures. Atypical nuclei are elongated and slightly enlarged. Their chromatin is finely granular, with one or two small nucleoli. They are associated with signs of severe chronic inflammation [1, 2, 7].

Tubal carcinoma should not be confused with tubal hyperplasia with reactive atypia in PID. Several factors may favour the diagnosis of pseudocarcinomatous hyperplasia: the patient's young age, bilateral involvement, absence of a visible mass on macroscopic examination. Solid tumor proliferation, marked nuclear atypia, mitosis and P53 overexpression, as well as a high Ki67 index (>60%), suggest a diagnosis of carcinoma rather than reactive hyperplastic lesions. Acute or chronic salpingitis is usually present in hyperplasia, but some carcinomas may have an inflammatory component [6, 8]. All these elements of differential diagnosis are summarized in Table 1.

Table 1

	Tubal hyperplasia	**Tubal carcinoma**
Age	27-40 years	Post-menopause
Macroscopy	Non-specific, Enlarged tube with thickened wall, of microscopic discovery	Obvious tumour mass
Architecture	Complex pseudoglandular architecture without solid appearance	Variable proportion of solid architecture
Nuclear atypia	Mild to moderate	Moderate to severe, prominent nucleoli
Mitoses	rare	Frequent
Infiltration of tubal wall	Pseudoinvasion of muscularis propria Mesothelial hyperplasia	Present
Ki67	low	High
P53	Heterogeneous (non-mutated)	Diffuse positive (mutated)
Signs of associated chronic pelvic inflammatory disease	present	Absent or very rare

CONCLUSION

Pseudocarcinomatous tubal hyperplasia is a rare, benign lesion, little known to pathologists, which can mimic a carcinomatous lesion, hence the importance of correct diagnosis with a view to appropriate management.

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