

# Pathological Changes in the Wall of the Fallopian Tube in Tuboperitoneal Infertility

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**Abstract** Our work studied pathological changes in the wall of the fallopian tube in tuboperitoneal infertility. We examined surgically removed fallopian tubes during medically indicated surgery in women diagnosed with infertility. In tubal-peritoneal infertility, all layers of the wall of the fallopian tube were subjected to an inflammatory and adhesive process. Due to the inflammatory-adhesive process of the interstitium, damage to the intrinsic cell-fibrous structures of the tube wall is noted in the form of dystrophy, destruction, and degeneration, which are the morphological substrate for the non-functioning of the fallopian tube.

**Keywords** Uterus, Tube, Pregnancy, Infertility

## 1. Introduction

Infertility, according to various authors [1,2], occurs in 15-20% and its prevalence tends to gradually increase. In the structure of an infertile marriage, 50-60% is female infertility, among the causes of which the leading role (from 35 to 85%) belongs to the tubo-peritoneal factor and infertility in the absence of fallopian tubes [3,4,5]. Tubal-peritoneal infertility (TPI) is a consequence of the acute or chronic course of inflammatory diseases of the uterine appendages, in which it is possible to perform both radical and organ-preserving operations. Tubal and tubo-peritoneal factors of infertility (TPFI) adversely affect, on the one hand, the incidence of pregnancy using assisted reproductive technologies (ART), in particular IVF and embryo transfer into the uterine cavity (ET), on the other hand, on the number of early embryonic losses. In addition, these infertility factors often contribute to the occurrence of ectopic pregnancy. Many IVF facilities offer prior bilateral tubectomy to improve IVF outcomes [6,7]. However, many researchers [3,4,6] have confirmed the adverse effect of removal of the fallopian tubes on ovarian function, which was accompanied by menstrual irregularities, changes in ovarian function, and a decrease in ovarian reserve. In the available scientific literature there is no data on the morphological study of the wall of the fallopian tube in tuboperitoneal infertility. In this regard, this work sets the goal of studying pathomorphological changes in all layers of the wall of the fallopian tube in tubo-peritoneal infertility.

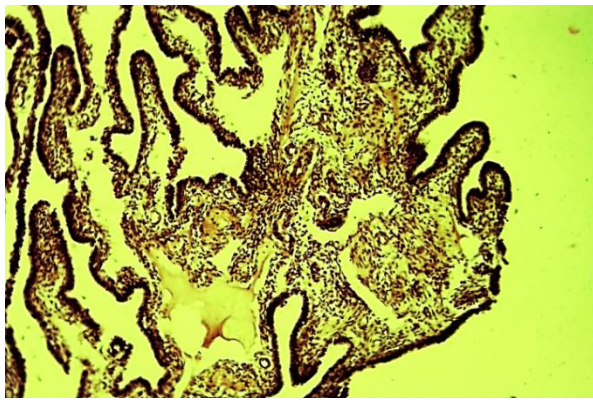
## 2. Material and Methods of Research

The object of the study was biopsy material from the department of biopsy diagnostics of the Russian Medical Center, surgically removed fallopian tubes for salpingitis and infertility. After macroscopic examination, 3 pieces were cut out: the uterine part, the intermediate section, and the ampullary part of the tube. The pieces were fixed in 10% formaldehyde in phosphate buffer for 48 hours, then washed in running water. Dehydration was carried out using alcohols of increasing concentration and chloroform, and embedded in paraffin. Histological sections were stained with hematoxylin and eosin and viewed under a light microscope, and the desired areas were photographed on a computer.

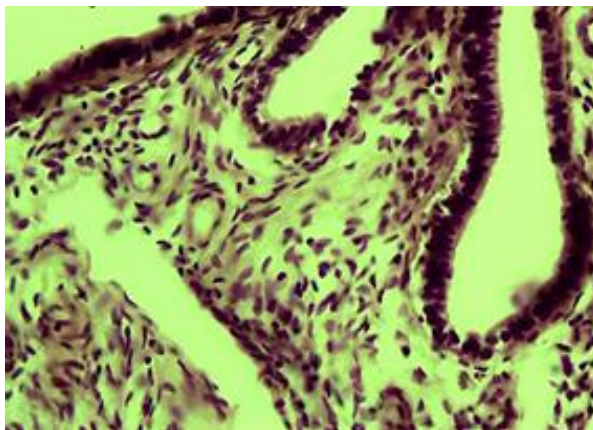
## 3. Results of the Study and Their Discussions

The results of a morphological study showed that with tubal-peritoneal infertility, all layers of the uterine wall are subject to an inflammatory-sclerotic process. The villi of the mucous membrane of the tube are deformed with a violation of histotopography and are presented in different shapes and sizes. The integumentary epithelium is somewhat thickened, in places metaplastic to squamous single-layer epithelium. Among the integumentary epithelium, atrophy and disappearance of the ciliated and secretory epithelium are noted; instead, intermediate cells are hyperplastic. The nucleus of the latter is hyperchromatic, relatively small and randomly located, some of them penetrate into the connective tissue plate proper. The lamina propria is expanded in area and is represented by cell-proliferative,

sclerotic connective tissue (Fig. 1). The connective tissue consists of mature fibrous areas. Vessels of different sizes and shapes, their walls are thickened due to perivascular sclerosis. Study with a large microscope lens showed that the connective tissue lamina propria is dominated by mature histiocytic cells and fibrous structures. Moreover, these cells and fibrous structures are oriented around the vessels and are located parallel to the integumentary epithelium. Among the cellular composition of the connective tissue, lymphoid cells and macrophages are determined, which indicates the presence of an inflammatory process in the lamina propria (Fig. 2). Polymorphism of the integumentary epithelium or the presence in the composition of the integumentary epithelium of low round-nuclear, multirow and tall cylindrical cells. In the lamina propria of the villi of the fallopian tube mucosa, diffuse and relatively dense lymphoid infiltration is determined (Fig. 3). Lymphoid cells include small, medium and large cells and plasma cells. Due to cellular infiltration, the connective tissue is loosened in the form of disintegration of fibrous structures and swelling of the intercellular substance. The covering epithelium is flattened and desquamated over a large extent; among them there are large cells due to vacuolization of the cytoplasm.



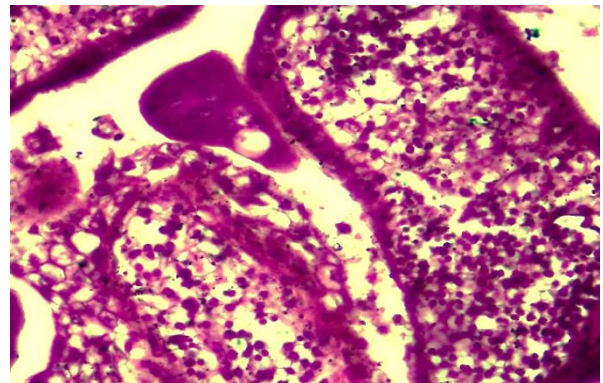
**Figure 1.** Tubal-peritoneal infertility. The villi are of different shapes and sizes, the lamina propria is fibrotic and inflamed. Color: G-E. UV: 10x10



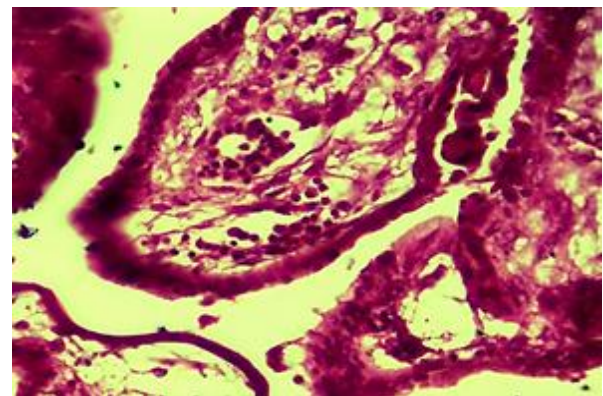
**Figure 2.** Tubal-peritoneal infertility. The lamina propria contains many connective tissue cells and fibers. Color: G-E. UV: 10x40

The lamina propria of individual villi are markedly edematous, small-celled, with areas of myxmatosis and

necrobiosis. Connective tissue cells are single, arranged in bundles instead of with lymphoid cells and oriented around the vessels. The fibrous structures are destructive, homogenized due to severe edema and disorganization of the intercellular substance (Fig. 4). In some villi, destruction and disintegration of both the integumentary epithelium and the lamina propria are noted. The integumentary epithelium is destroyed, thinned with signs of dystrophy and destruction. In some places, the covering epithelium is desquamated and forms cellular accumulations between the villi.



**Figure 3.** Tubal-peritoneal infertility. The lamina propria of the villi of the tube mucosa is diffusely infiltrated with lymphoid cells. Color: G-E. UV: 10x40

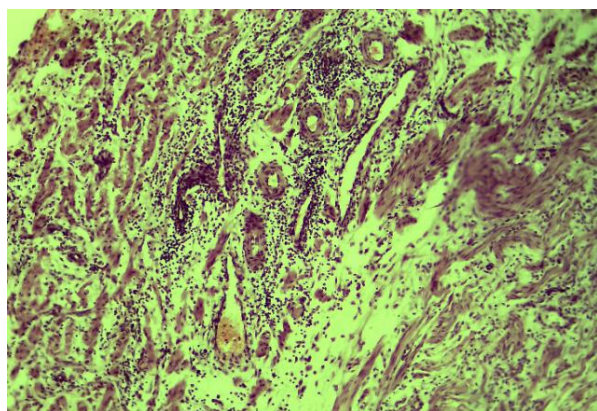


**Figure 4.** Tubal-peritoneal infertility. Swelling, loosening and necrobiosis of the lamina propria of the villi of the mucous membrane of the uterine tube. Color: G-E. UV: 10x40

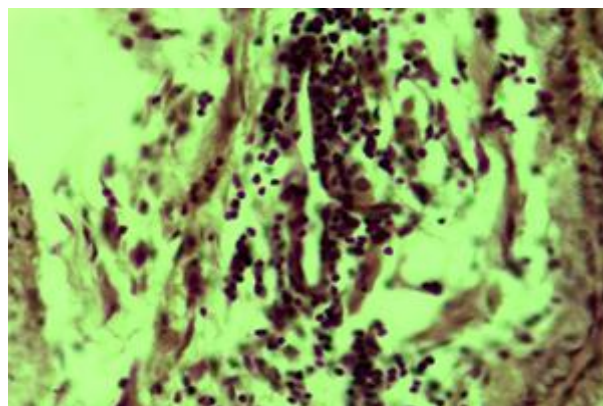
The results of a morphological study of the submucosal and muscular layers of the wall of the fallopian tube showed that the cellular-fibrous and vascular structures are loosened and scattered due to pronounced edema of the intercellular substance, the presence of a diffuse inflammatory infiltrate and disorganization of cellular structural elements. The connective tissue of the submucosal layer is subjected to severe swelling and loosening with disruption of the histotopography of both cellular and fibrous structures. The intercellular substance is diffusely infiltrated with inflammatory cells, which indicates irreversible damage to all tissue structures of this layer of the pipe wall. The muscle layer is also significantly thickened due to severe edema and inflammatory infiltration of the intercellular space (Fig. 5). Moreover, inflammatory infiltration is more concentrated in the circumference of venous vessels with destruction



of the cell-fibrous structures of their walls. There is no inflammatory infiltration around the arterial vessels; the arterial wall is thickened due to the proliferation of cellular elements and swelling of fibrous structures. Lymphoid cells and macrophages predominate among the inflammatory infiltration. The muscle bundles are fragmented and scattered into separate small bundles with disruption of the histostructure of myofibrils and nuclear structures. Thus, the above pronounced inflammatory changes developing in all layers of the wall of the fallopian tube lead to disruption of elasticity, peristalsis, food and oxygen supply of all cellular-functional structures of the fallopian tube.



**Figure 5.** Tubal-peritoneal infertility. Diffuse edema and inflammatory infiltration of the interstitium of all layers of the wall of the fallopian tube. Color: G-E. UV: 10x10

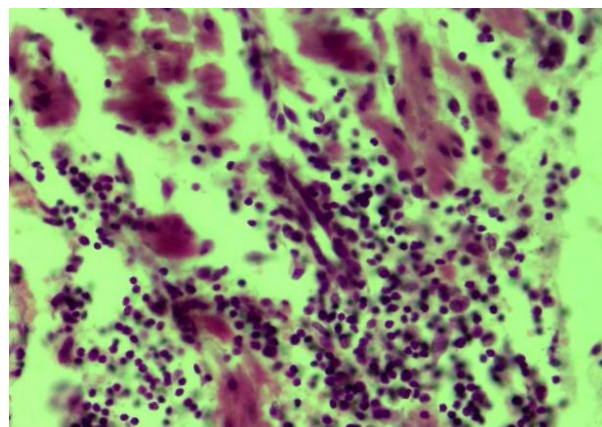


**Figure 6.** Tubal-peritoneal infertility. Inflammatory infiltration of the wall of the vein vessel of the fallopian tube wall. Color: G-E. UV: 10x40

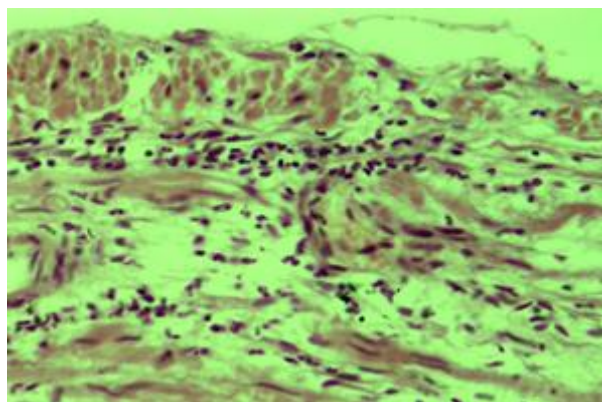
When studying at high magnification with a microscope lens, it was noted that the inflammatory infiltration oriented around the vessels infiltrates the walls of the vessel, destroying cellular and fibrous structures, due to which the lumen of the vessel is narrowed (Fig. 6). Among the inflammatory infiltration, lymphoid cells with hyperchromic nuclei predominate, which indicates autoimmune damage to the tissue structures of both the vascular wall and smooth muscle cells. The presence of autoimmune inflammation in the wall of the fallopian tube also leads to disruption of peristalsis, conductivity and nutrition of food and oxygen to the cellular structures of the tube wall. The development of a

proliferative inflammatory infiltrate in the interstitium of the muscular layer of the wall of the fallopian tube indicates the development of chronic salpingitis. The lympho-proliferative inflammatory infiltrate tightly surrounds the microvessel of the muscle layer and directly infiltrates muscle cells with foci of destruction and disorganization of myofibrils and nuclear structures (Figure 7).

During the morphological study, we took into account the peculiarities of the structure and composition of the outer layer of the wall of the fallopian tube. What is that the serous membrane of the tube is normally represented by a small layer of smooth muscle cells, well-developed interstitial connective tissue and mesothelial integumentary cells. It is noted that this layer of the fallopian tube wall is also diffusely infiltrated with inflammatory cells. The inflammatory infiltrate is mainly represented by lymphoid and histiocytic cells, which more pronouncedly infiltrate the inner layer of interstitial tissue facing the muscle layer (Figure 8). The muscle cells of the serous layer of the tube wall are located longitudinally and they are colored more intensely compared to the muscle cells of the muscular layer. The interstitial connective tissue of the serous membrane of the tube is represented by loosely arranged fibrous structures with single cellular elements, which are also subject to swelling and loosening.



**Figure 7.** Tubal-peritoneal infertility. Lymphoproliferative infiltration of the muscular layer of the fallopian tube wall. Oxarca: G-E. UV: 10x40



**Figure 8.** Tubal-peritoneal infertility. The serous membrane of the tube wall is infiltrated with lymphohistiocytic cells. Color: G-E. UV: 10x10

## 4. Conclusions

1. It has been established that in tubal-peritoneal infertility, all layers of the uterine wall are subject to an inflammatory-sclerotic process.
2. Discomplexation, deformation, inflammation and fibrosis of the lamina propria, atrophy, and metaplasia of the integumentary epithelium were noted.
3. Diffuse lymphoproliferative inflammation of the submucosal and muscular layers with destruction of the tube's own cell-fibrous structures was determined.
4. Thickening of the serous membrane due to a set of edema and inflammatory infiltration, thickening of muscle cells with destruction and desquamation of the mesothelium.

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