

Borderline Ovarian Tumors – Definition and Classification

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1. Summary

Borderline ovarian tumors (BOT) usually occur in women in reproductive age. In comparison with invasive ovarian cancers, the borderline tumors occur in younger patients, approximately around 50 years of age, but invasive ovarian carcinoma around age 60. Positive prognosis depend on the facts, that in 80% of cases these tumors are early diagnosed. In this area although the clear classification, borderline ovarian tumors are one of the most controversies in field of onco-gynecology, and that is mostly disputable is the therapeutic approach in general. In these years radical surgical treatment is the gold standard, but in some cases, especially in young patients, there can be ovarian tissue sparing surgery performed.

2. Introduction

Group of borderline ovarian tumors represents its own nosologically unit, with mentioning in 19th century. In 1989 Hermann Johanness Pfannenstiel diagnosed papillary ovarian cystadenoma, to which he contributed the attributed of the tumor with borderline malignant potential. Three years later, in 1901, Carl Abel singled out the group of proliferative papillary cystadenomas, which growth he defined on the edge of benign and malignant lesions.

Terms as “borderline tumor”, or “semi-malignant tumor” had been first presented in the year 1929 by Howard Taylor, who named these kinds of serious cyst-adenomarcinoma tumors [1]. Until the year of 1995 there hadn't been described as tumors with malignant potential, but only as serous ovarian tumors. It had been

done by Cleveland oncology clinics. Definitive classification of malignant potential ovarian tumors was done by FIGO (International Federation of Gynaecology and Obstetrics) in 1971. WHO classification of ovarian tumors in 1973 brought for borderline tumors labelling as “carcinomas with very low malignant potential [2]. Development of the classification until today is lasting more than 125 years.

3. Epidemiology and Risk Factors

The inexact percentage from 10 – 15% of cases of borderline ovarian tumors is done by unclear histopathologic criteria especially in early stages. Thanks to modern techniques of imaging and screening the incidence in diagnostic increased over 500% by last 50 years. This increase is done mostly by exacting the histopathological examination, not by growth of incidence of the disease (Table 1).

Table 1: Survival rate in different stages (3)

Stage	% survival rate
Stage I	99,6%
Stage II	95,8%
Stage III	89%

3.1. Risk Factors

Risk factors classify borderline ovarian tumors into under group of ovarian malignancies with good prognosis. Riman et al.in his population study proved that higher risk factors are

- Obesity
- Smoking
- Hormonal contraception
- Age by giving first delivery

On the other hand lactation and multiparity had decreased risk of occurring borderline ovarian tumor.

4. TNM/FIGO Classifications

TNM/FIGO classification of the tumors with malignant potential is done the same. Way as for malignant tumors from superficial epithelia and stromal tumor. It includes primary tumor, lymph nodes and metastasis [4].

a) Classification of the Primary Tumor

(Table 2)

b) Classification of Regional Lymph Nodes

We classified regional LN, which are pelvic (paracervical, parametrial, hypogastric, iliacal, parasacral, sacral), paraaortal and inguinal. It is needed to prepare and histologically examine at least 10 lymphatic nodes (Table 3).

c) Classification of Metastates

(Table 4)

5. WHO Classification

World human organization classifies borderline ovarian tumors since 1971 from histological point of view according to the cells which create the tumor (Table 5).

Histopathological classification of borderline tumors [6,7].

6. Summary TNM and FIGO Classification

(Table 6)

Table 2: Staging of the disease

<i>Staging of the tumor</i>	<i>description</i>
TX	It is to able to be evaluated
T0	Without primary tumor
T1	Tumor is limited on ovarian tissue (T1a, T1b, T1c)
T2	Tumor affects one or both ovaries and spreads to the pelvic (T2a, T2b T2c)
T3	Tumor affects one or both ovaries and has microscopically detected peritoneal metastates outside the pelvis (T3a, T3b, T3c)
T4	Tumor effects one or both ovaries and has already further metastates

Table 3: Staging of the lymphatic nodes

<i>Lymph nodes</i>	<i>description</i>
NX	Regional LN are not being able to evaluate
N0	No metastases in regional LN
N1	Metastases in regional LN

Table 5: Histological staging (5)

<u>Type of tumor</u>	<u>subtypes</u>
Serous	- Papillary cystic tumor - Superficial papillary tumor - Adenofibroma - cystadenoma
Mucinous	- interstitial type - endocervical type
Endometroid	- cystic tumor - adenofibroma - cystadenofibroma
Clear cell	- cystic tumor - adenofibroma - cystadenofibroma
Tumors from superficial tissue	- Brenner proliferative tumor
Mixed epithelial tumors	

Table 4: Staging of metastases

<i>Metastates</i>	<i>description</i>
MX	Further metastases are not being able to evaluate
M0	No further metastases
M1	Further metastases

Table 6: TNM/FIGO classification (5)

<i>TNM</i>	<i>Description</i>	<i>FIGO</i>
T1a	Affected one ovary, casing is intact, without any tumor, with negativity of ascites or lavage	IA
T1b	Affected both ovaries, casing is intact, without any tumor, with negativity of ascites or lavage	IB
T1c	One or both ovaries + rupture of casing (tumor on the surface), positivity of ascites or lavage	IC
T2a	Spreading to uterus (Fallopian tube), negativity of ascites or lavage	IIA
T2b	Spreading to pelvic tissue, negativity of ascites or lavage	IIB
T2c	Spreading to uterus (Fallopian tube), positivity of ascites or lavage	IIC
T3a	Microscopic peritoneal metastases outside the pelvis	IIIA
T3b	Macroscopic metastases outside the pelvis up to 2cm	IIIB
T3c	Macroscopic metastases outside the pelvis bigger than 2cm	IIIC
4	Further metastases	IV

7. Microinvasion

Microinvasion occurs in -10% - 15% of all cases of serous borderline ovarian tumors. Characteristics for BOT with microinvasion is by occurring clusters or single cells in the stroma. Localities of stromal invasion contain same cells as the original cells of borderline tumors. Most of the studies recommends considering microinvasion not bigger than 3mm with all around range not more than 10mm². Minimally higher risk of percentual microinvasion (13%) is seen in immunohistochemical examination using epithelial markers. Microinvasion in case of histological exam in mucinous borderline ovarian tumor occurs only in 9%, and invasion into the vascular system in 10%. Criteria for classification of microinvasions are the same as in cases of serous borderline tumors, although separate locations of microinvasions in cases of malignant potential BON are around 1- 2mm [6].

Although the presence of stromal microinvasion means local spreading of the disease, does has significantly prognostic factor in comparison with stage of disease with presence of peritoneal implants and biological behavior and the prognosis is similar with that of serous borderline ovarian tumor without microcalcification [7].

8. Peritoneal Impants

Peritoneal implants in borderline ovarian tumors present extraovarian disease which is present on the serous superficial part of abdominal wall and omentum. Literature is not precise about occurrence of the peritoneal implants and presents wide range of their occurrence from 20 – 46%. Peritoneal implants are in two groups according to their prognostic factor – invasive and non-invasive. Both types can occur concomitantly and its' prognostic value is different. For precise staging there is need of biopsy large enough, from which there can be determined the edge between the implant and all around tissue [8].

8.1. Non-Invasive Peritoneal Implants

The line between impland ad normal healthy tissue (omentum, intestinal wall) is in case f non-invasive implants very clear.

a) Epithelial non-invasive peritoneal implants are papillary proliferation of serous epithelia on the surface of the peritoneum without invation of bottom of peritoneum. They occure in forms of calcification characterized by presence psamomatous cells.

b) Desmoplastis non-invasive peritoneal implants are in histological picture imaged as fibroblastic proliferation similar to the granulation tissue. It occurs in inflammatory infiltration and microcalcification, mitotic activity is missing.

c) Invasive peritoneal implants usually affect peritoneum and omentum, but they can also be found in visceral organs. The line between implant and surrounded tissue is not clear and we can find infiltration of the surrounded tissue by epithelial structures [8].

These different types of peritoneal implants don't usually occur separately, and we find invasive and non-invasive forms alongside. There is need for precise surgical staging and also experienced histologist who does the perioperational histology. The origin of peritoneal implants stays unclear. Its'close relationship to exophytic form says about the disorientation of superficial cells of exophytic tumor and their followed by their attachment to serosis or omentum. On the other side there is not rare that peritoneal implants are diagnosed also in absence of exophytic ovarian tumor. They are very common in endosalpigosis. These conditions say about possibility of the local development of peritoneal implants solitarily. But the correlation of exophytic tumor with peritoneal implants is more common [8-10].

9. Invasion of Lymphatic System

Tumorous cells of borderline ovarian tumor in lymph nodes are labeled as invasion of lymphatic system. It is described in 20 -25% of patients with serous borderline ovarian tumor, although studies with precise histologisation are very rare, because most of the time the lymph nodes are described only as positive or negative.

Invasion of lymph nodes are in order paraaortal, pelvic, iliacal and omental ILN.

Tumorous invasion of lymphatic nodes is needed to be differentiated from endosalpingiosis, which are bening gland inclusions of Mullerian origin, which occur in pelvic lymphatic system [11].

10. Molecular Pathogenesis

Tumorous process is complex as itself. There had been long believed that tumor is “a genetic disease”. But not all genetic aberrations in tumor are connected by tumorous transformation. Instability of the genoma is responsible for higher risk of accumulation of genetic alterations with risk of occurrence of malignancies and is in right connection with failures of corrective DNA system. There are abnormalities of specific genes responsible for wrong distribution of chromosomes.

Key role in pathogenesis of borderline ovarian tumors is played by mutation of protooncogene K-ras (rous adenosarcoma) and its' mediator B-raf by activation of kinetic cascade MAPK (mitogen-activated proteinkinase). Mutations of K-ras and B-raf are

early genetic moment in cancerogenesis of serous borderline ovarian tumor.

Outcome is permanent signalization and uncontrollable cell proliferation. Punctual mutations, which activate K-ras are in codones 12,13 90%) or in codones 61. These codones are the most sensitive place for occurrence of mutations, which are in 2/3 of all serous borderline ovarian tumors. Progression of SBOT into the serous low-grade carcinoma is analogous in mutations ras and raf [12,13]. Model of the development of invasive carcinomas include line of crossing the serous borderline tumor into the low-grade carcinoma, second line is crossing SBOT into the high-grade carcinoma [14,15].

10.1. Principles of Oncogene and Gene Alterations

(Table 7)

Table 7: Principles of gene alterations [16]

<u>Histologic type of tumor</u>	<u>Gene alteration</u>	<u>% of mutations in the tumor</u>	<u>Precursor and its' invasive carcinoma</u>
Serous	B-raf and K-ras mutation	67%	precursor - Serous cystadenoma or adenofibroma Progression - Into low-grade serous carcinoma
Mucinous	K-ras mutation	> των 60%	Precursor - Mucinous cystadenoma Progression – into intraepithelial carcinoma and then invasive mucinous carcinoma
Endometrioid	- Mutation PTEN	20%	Prekursor - Endometriosis, ,endometrioid adenofibroma Progression – into intraepithelial carcinoma and then invasive endometrioid carcinoma
	- Mutation of β-carotene gene	50%	
	- Microsatellite instability	13 – 50%	
Clear cell	- K-ras mutation	5 – 16%	precursor - Endometriosis, clear cell adenofibroma Progression – into intraepithelial carcinoma and then invasive clear carcinoma
	- Microsatellite instability	13%	
Brenner	Not well described		Precursor - Brenner tumor Progression – into malignant Brenner tumor, carcinoma of the transitory epithelia

11. Ovarian Tumors – Histopathologic Characteristics of Different Types

Approximately 60% out of all ovarian tumors, and 80 – 90% of malignant ovarian tumors are presented from epithelial origin. Apart from mucinous tumors, which mostly origin from teratomas, this ovarian neoplasms most probably originate from the superficial epithelium of ovary, which is derived from coelom epithelia, which are also origin for Mullerian ducts.

The superficial epithelium is able to differentiate it from serous, mucinous, endometrial or previous epithelium.

Superficial ovarian epithelia tumors, which show controversies between benign cyst-adenocarcinoma or usual ovarian tumors, where discovered more than 80 years ago. In 1929 Taylor described group of patients with “hyperplastic” ovarian tumors without histologic confirmation of stromal invasion or with peritoneal implants. He noticed that these patients have better prognosis than

those with malignant forms, and therefore he suggested the concept of “semi-malignant ovarian tumors” as a definition or primary ovarian neoplasms, which shown characteristics between benign adenomas and malignant adenocarcinomas.

In 1952 Kottmeier included semimalignant epithelial tumors in the classification of ovarian tumors. He defined these neoplasia as papillary tumor and cystadenomas with epithelial changes identical with those, which can occur in invasive carcinomas, but without obvious stromal invasions. In the beginning of 70s years WHO and FIGO included these tumors into classification and recommended, that these neoplasms would be called “borderline malignant” or “tumors with low malignant potential.

To clarify with uniformed classification of ovarian epithelial tumors there had been includes also types of mucinous tumors and rara types of epithelial tumors of the ovary. Therefor there had been established the term “borderline” applied on tumors, which showed epithelial proliferation more often than in benign lesions

but didn't show the destruction by invading stroma. These tumors have better prognosis than ovarian carcinomas. Although there are controversies in nomenclature of these types of ovarian tumors but the latest nomenclature by WHO agrees with "tumors with low malignant potential" as an accepted synonym. This is also more favorable by pathologists (Table 8).

Borderline ovarian tumors are count for 15 – 20% of non-benign epithelial ovarian neoplasms, what which the majority is serous or mucinous type. For exact determinate of border ovarian tumors, it is necessary to determine types of cells from which is the tumor assembled, because in different types of histological borderline tumors, there is different clinical significant display. The group of

borderline ovarian tumors is very heterologous group of lesions. That is the reason why all different controversial aspects are brought up in diagnosis, biological behavior, character of extra-ovarian lesions, molecular and biological relationship to invasive carcinoma and its' clinical management. The most important role is that of pathologist is to differentiate borderline ovarian tumor from the invasive form of the ovary cancer. This difference is one of the most difficult in pathology of ovary cancer in general. Borderline ovarian tumors mostly affect women in reproductive age, therefore it is needed for therapeutic decision also consider tissue sparing surgical treatment, hormonal deprivation and chemotherapeutic treatment.

Table 8: Histopathologic characteristic of different types of borderline ovarian tumors

Type of tumor	Precursor	Progression into cytogenetic	Characteristics	prognosis
a) <u>Serous</u> - Serous - Cystadenoma - adenofibroma	Invasive low-grade serous carcinomas	Mutation in KRAS or BRAF gens in 67%	May have dualistic oncologic pathway: - invasive - noninvasive peritoneal implants may occur, presence of invasive implants is a poor prognostic factor associated with regional lymphadenopathy - typical subtype – (90%) - micropapillary subtype (10%) is closely associated with invasive implans.	70% cases are in stages I, survival is almost 100% - 30% of cases are advanced stages with survival rate is 95,3% if implants are noninvasive and 66% if implants are invasive
b) <u>Mucinous</u>	- Interstitial subtype (90%) - 1 carcinoma in KRAS (>60%) then to – - Invasive - Mucinous - Carcinomas - Mullerian subtype	KRAS > 60%	- Intestinal subtype (81_90%) in cases unilateral, multicystic smooth capsule – is associated with pseudomyxoma peritonei - has larger multilocular cystic lesions Mullerian subtype (10%) is bilateral, in 20 – 30% is exophytic and paucilocular, mimics serous tumor hence termed "serousmucinous": implants may present	82% of cases are in stage I, which accounts for about 5-year survival up to 99 – 100%, shows fluids of different signal intensities on T1- or T2-weighted MR images 18% of cases are advanced stages, mortality may reach up to 50% depending on stage
c) <u>Endometrioid</u>	- Endometriosis - endometroid - adenofibroma - Intraepithelia of carcinoma then to low to -5%, grade invasive endometroid with carcinoma microsatellite	Gene muatations in β -catenin (>50%), loss of heterozygosity or PTEN mutation with (20%) microsatellite	None	Benign course with high survival rates
d) <u>Clear cell tumor</u>	- Endometriosis - Clear cell adenofibroma	Intraepithelial carcinoma then invasive clear cell carcinoma	KRAS mutations (5 – 16%: microsatellite (\cong 13%)	Benign courses with high survival rates
e) <u>Brenner</u> - Benign - Brenner	- malignant - Brenner?	Not yet identified	None	Benign courses with high survival rates

11.1. Serous Borderline Ovarian Tumors

Serous borderline ovarian tumors (SBOT) account for 25 -30% of non-benign serous tumors. They are most common in women in the age group between 40 – 50-years of age, which is the perimenopausal period. Mostly in 70% it is localized on one ovary, in case of localisation of both ovaries in early diagnostic it is considered stage I. All other tumors which grow more have different staging

- into the pelvic area (stage II)
- upper abdominal area (stage III)
- late diagnostic is very rare (stage IV)

Macroscopically there are mostly cystic or papillary types of tumors. The size differs, Segal presents on the study of 98 patients diagnosed by serous borderline tumor the size around 2 – 25cm, in average around 10%. Approximately in half of the patients there is exophytic tumor on the surface of the ovary. Origin of this part is from the surface parenchymatous epithelium and there is non cancerous cells found inside of the ovary. Only in 10% of the cases there is only exophytic superficial tumor without any part of intraovarian tumor. Histologically is borderline ovarian tumor considered as non-invasive proliferative disease with cells with small to middle atypical cores. Presence of high number of cell core atypia means important criteria considered for conventional serous papillary carcinoma. Psammomatous solid bodies occur in case of serous borderline ovarian tumors in parts of stromal microinvasion. According to histological signs there is possibility do define its' own variation. Micropapillary variation occurs in 6 -18% of all serous BOT. It belongs to the under group of borderline tumors characterized by specific histological findings of micropapillary signs and worse prognosis. In difference from classical SBOT it occurs bilaterally, has exophytic and faster growth. According to some studies the micropapillary variant has higher incidence, peritoneal implants and shorter disease-free interval in cases if there is a recurrence of the disease comparing to the classical type of serous borderline ovary tumor. That is why all these cases lead to late diagnostic of higher stages, in general survival rate comparing to the micropapillary and classical serous borderline tumor.

Serous borderline tumors are composed from one or more cystic parts which inner lining shows polypomatous nodules and small papillas (endophytic growth) (Figure 1).

Macroscopically the serous borderline tumors are similar of those of serous cystadenomas. It usually comes in form of cystic or papillary tumor, which doesn't contain parts of necrosis of hemorrhage, like carcinomas do, but very often contain macrocalcification psammomatous bodies. Macrocalcifications develop on the

basis of fat tissue necrosis and by picking up the calcium salt in arterial hypertension. According to some studies, macrocalcifications are associated with presence of nanobacterial antigen which was found in ascited.

Inner lining of the cyst shows polypoidous outgrowth and tender papillas (figure – endophytic tumor). These cysts contain serous or thick mucinous liquid. For papilla there is typical hierarchic branching and stratification of epithelial cells, which are different and can create picture “floating” clusters of cells with lumen of the cyst. Polyps and polypoid outgrowth are combines from fibrotic and edematous stroma [17] (Figure 2-9).

According to WHO there is classification of serous borderline tumors into:

a) typical serious borderline ovarian tumor – which is histologically presented by tumorous cells of low and middle degree of core atypia, there is just no invasion into the stroma. Mytotic activity is very low [19]

b) micro-papillary type – in 6 – 18% of the all cases of SBOT. It is the under group of borderline ovarian tumors characterized by non-hierarchically branching of papillas. There are wide, edematous papillas growing out of the cystic wall and then long tender micropapillas with minimal supporting wall or without it. This type is compared to the head of medusa.

On the other hand the difference from the typical SBOT, it grows usually bilaterally, exophytic tumor and faster growth. It has lower incidence for peritoneal implants [6].

c) Serous borderline ovarian tumor with microinvasion - it is a separate group [6,7].



Figure 1: Serous adenocarcinoma with cystic and solid fluid



Figure 2: Cystic lesion of unilateral ovary

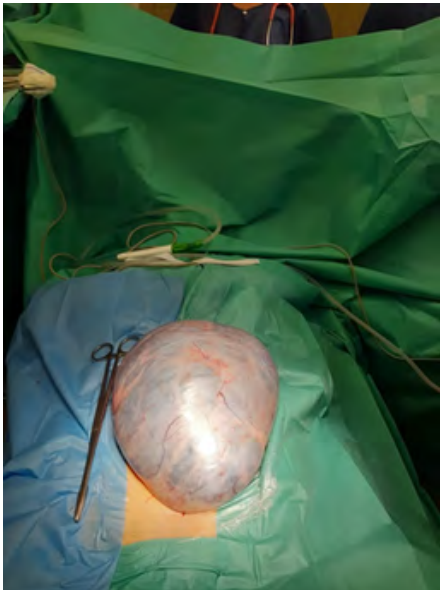


Figure 3: Cystic formation in situ

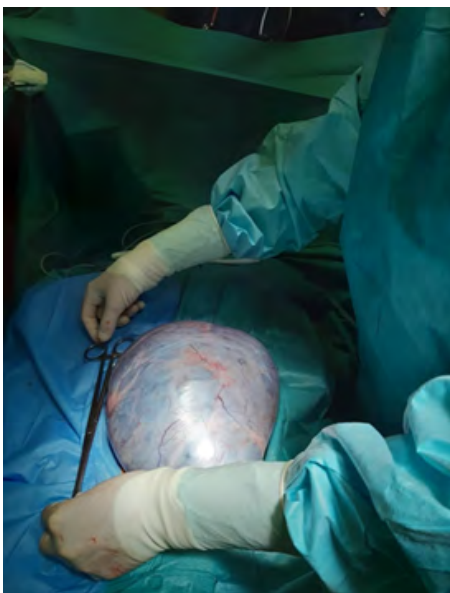


Figure 4: Comparison of the size

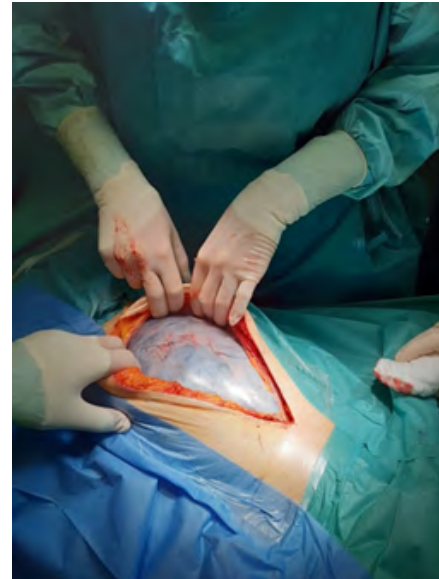


Figure 5: Tumor in situ

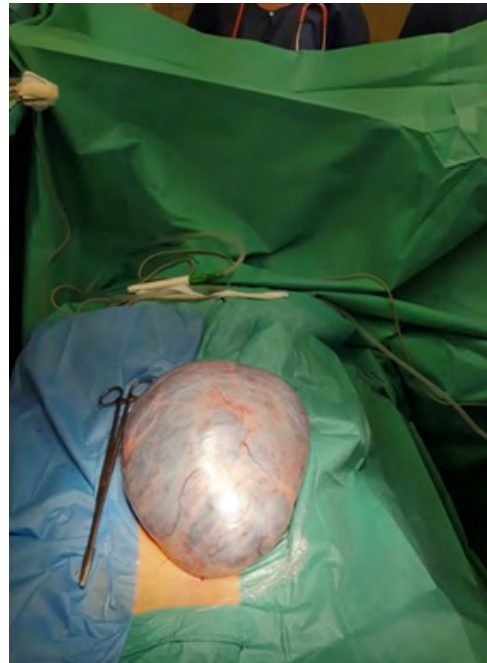


Figure 6: Precise preparation



Figure 7: Tumor size

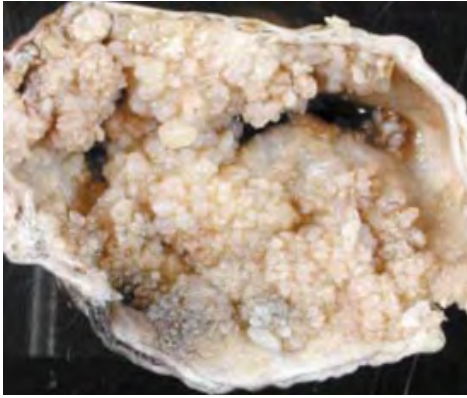


Figure 8: Endophytic growth of borderline ovarian tumor with sparing skin cover (28)

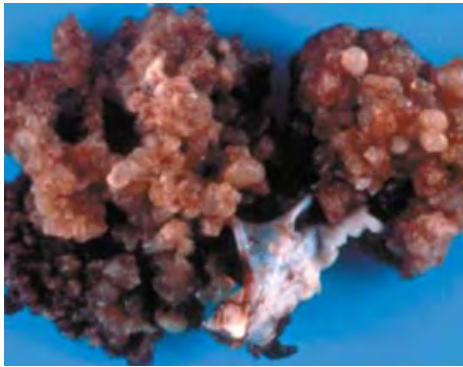


Figure 9: Endophytic growth of borderline ovarian tumor with invading through skin cover (18)

12. Mucinous Borderline Ovarian Tumors (MBOT)

Mucinous borderline ovarian tumors belong to the group of the biggest human tumors. In comparison with serous tumors is its bilaterality quite rare in 20 – 30% of cases [7] (Figure 10 and 11).

Biology of these types of mucinous ovarian tumors is very different from those of serous type. There are two basic histological subtypes of mucinous borderline ovarian tumors defined as:

a) Interstitial type – epithelia of this type of mucinous tumor reminds of intestinal wall with neuroendocrine cells. Represents 85 – 90% of all mucinous BOT. These tumors are usually very big, multilocular and unilocular with fluid or mucous substance ingredient. Bilaterally there occur approximately in 5%. Stratified epithelia and overall look reminds hyperplastic or adenomatous intestinal polypus. Sometimes there can be found cytologically malignant cells, stratified into more than four layers, creating solid, cribriform or papillary picture and they are called borderline tumors with intraepithelial carcinoma.

Interstitial type of mucinous borderline ovarian tumor with finding pseudomyxoma peritonei is very typical alongside with mucinous kystoma or adenocarcinoma of appendix.

b) Endocervical type has characteristic of epithelium of endocervix, epithelium covers wide papilla, which contain cylindric mucinous cells. This type of MBOT is very rare, approximately 5 – 14% of all mucinous borderline ovarian tumors. They occur bilaterally in

40%. Histopathologic characteristic and also clinical presentation reminds of serous borderline ovarian tumors and it occurs alongside with SBOT, so it's called mixed or seromucinous borderline ovarian tumor. It is clear that in this mixed type and also Mullerian type of tumor the predisposition is endometriosis. Endocervical types of tumor are in comparison with interstitial type smaller and have less cysts. Characteristic sign for them is inflammatory infiltration in papilla and also in extracellular free area. In this type the development of peritoneal implants is probably granted to transformation from the endometriosis locuses on peritoneum [6] (Figure 12).



Figure 10: Abdominal cut preparation for cystic tumor



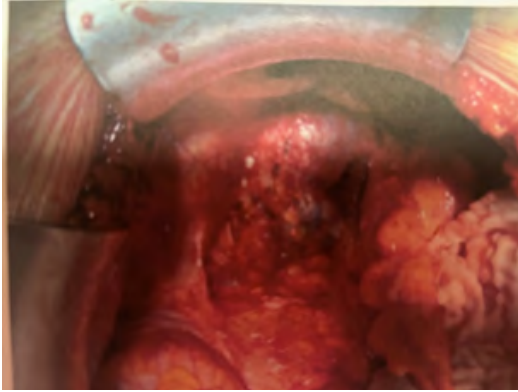
Figure 11: Mucinous borderline ovarian tumor invading the superficial layer with Fallopian tube



Figure 12: Omentectomy

12.1. Pseudomyxoma Peritonei

Pseudomyxoma peritonei is term used in case of presence mucous and gelatinous masses inside peritoneum. It is very rare tumor, which affects peritoneum by form of carcinosis, characterized is by slow progression of diseases with high volume of mucinous epithelial cells. Primary tumor is usually starting in appendix and after its' rupture it causes progressive dissemination of mucin-producing epithelia cells into the peritoneal cavity. Tumor of appendix



Figures 13 and 14: Pseudomyxoma peritonei

13. Endometrioid Borderline Ovarian Tumors

This type occurs very rarely approximately in 0,2% types of all ovarian epithelial tumors. It is defined by atypic proliferation and contains atypical and histologically malignant endometrioid glands surrounded by thick collagenous stroma. It can grow from superficial ovarian epithelia or also from endometriosis. In most cases it's similar to adenofibroma with complex endometrial structure. Biologically taken it is benign [7, 19, 20].

14. Clear-Cell Borderline Ovarian Tumors

Just like endometrioid types they also occur rarely in 0,2% of all cases of borderline ovarian tumors. They are defined by presence of atypical or histologically malignant glands or cysts outlined by clear cells [11]. Clear cells are rich with collagen that doesn't react on coloring with hematoxylin or eosin, so the cytoplasm of these cells is not colored at all or very pale. Clear-cell borderline ovarian tumors are associated with endometriosis. In these cases there is no finding of extraperitoneal implants [22].

15. Brenner Borderline Ovarian Tumors

They occur in 3 – 5% of borderline tumors and they are 16 – 20cm big. Mostly they are being unilateral. The tumors have solid and also cystic part, which in case of cystic part this has papillary or polypous content. Papilla are covered by transient epithelia [12]. In 25 – 30% they are associated with endometriosis [22-24].

Mixed epithelia borderline ovarian tumors

Most rare cases of borderline ovarian tumors, they content one or more epithelia of all above mention types. Mostly it is combination of serous and mucinous type of epithelia [11].

doesn't have to be by the surgeon macroscopically visible, that is the reason why appendectomy is always indicated in cases of diagnosis or mucinous borderline ovarian tumor with perioperative histological finding of pseudomyxoma peritonei (29).

Mucinous ovarian tumors with presence of pseudomyxoma peritonei are considered as metastatic in all cases. There is need for histologisation of the mass (Figure 13 and 14).

16. Discussion

One of the most complicated parts of gynecologic pathology belongs the specter of diseases, which belongs to the category of benign lesions and malignant carcinomas. This is the problematic of borderline ovarian tumors.

Only the development of definitive classification lasted more than 125 years. Diagnostics of borderline ovarian tumors depends on histopathologic examination and belongs to the responsibilities of experienced pathologist.

It is very controversial while it belongs to the diseases of women in early fertile age.

According to the fact that this disease has malignant potential, therefore exact and precise classification of borderline ovarian tumors is needed for further treatment management.

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