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STUDY OF HISTOPATHOLOGICAL SPECTRUM OF OVARIAN LESIONS AND NEOPLASMS

Pathology

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ABSTRACT

BACKGROUND: The most common lesions encountered in the ovary are functional or benign cysts and tumours. Neoplastic disorders can be grouped according to their origin from each of the three main ovarian cell types (1)Mullerian epithelium (2)Germ cells (3)sex cord -stromal cells.[1] About 80% of ovarian tumours are benign and these occur mostly in young women between the ages of 20 and 45 years. Borderline tumours occur at slightly older age group. Malignant tumours are more common in older women between the ages of 45 and 65 years[1].

Ovarian cancer accounts for 3% of all cancers in females and is the fifth most common cause of death due to cancer in women [1]

The classification of ovarian neoplasms as per WHO classification is broadly grouped into following categories[1]

- Surface epithelial-stromal tumours
- Sex cord -stromal tumours
- Germ cell tumours
- Metastatic cancer from non-ovarian primary

The most common symptoms of ovarian tumours are abdominal pain and distension, urinary and gastrointestinal tract symptoms due to compression by tumour or cancer invasion and vaginal bleeding. Benign forms may be entirely asymptomatic.

METHODS: All clinical details and data from case sheet and patient history are collected and analysed for all the patients who underwent surgery for ovarian pathology / neoplasms in the Department of Obstetrics and Gynaecology, Govt. Omandurar Medical College and Institute of social obstetrics and Govt. Kasturba Gandhi Hospital Chennai, from Jan 2017 to Dec 2017.

The Study period of this study was from Jan 2017 to Dec 2017. Multiple sections were taken from the neoplasms and tissues were processed in Automated tissue processor and Paraffin blocks made. Statistical analysis of the data was analysed.

RESULTS: Total number of specimens received were 50.

The Histopathology types seen in the study group were

S.No.	Type	Number
1	Serous cystadenoma	19
2	Serous cystadenofibroma	8
3	Mucinous cystadenoma	7
4	Mucinous cystadenofibroma	1
5	Endometriosis	1
6	Borderline serous cystadenofibroma	1
7	Borderline mucinous neoplasm	1
8	Serous cystadenocarcinoma	1
9	Mucinous cystadenocarcinoma	1
10	Benign cystic teratoma	8
11	Benign fibrothecoma	1
12	Krukenberg tumour	1

Among the neoplastic lesions of the ovary, Benign neoplasms formed the majority of neoplasms.

Among the Benign neoplasms, Benign Serous cystadenoma formed the majority with 19 cases, followed by serous cystadenofibroma and Benign cystic teratoma with 8 cases each and Mucinous cystadenoma with 7 cases.

The predominant age group affected was 41 to 50 years with 17 cases followed by 31 to 40 years with 10 cases, with the predominant neoplasm being Benign surface epithelial tumour, with the majority of cases being Benign serous cystadenoma.

CONCLUSION: Ovarian tumours are the common forms of neoplasia in women. Ovarian neoplasms account for a disproportionate number of fatal cancers, being responsible for almost half of the deaths from cancer of female genital tract because many of these ovarian neoplasms cannot be detected early in their development. Hence there is a need for histopathological diagnosis for these tumours, which cannot be diagnosed and categorised merely by clinical and radiological background.

Therefore, specific histopathological diagnosis is necessary as it may aid in the further management of cases, through which cancer deaths can be prevented by utilizing better treatment modalities.

KEYWORDS

Cystadenoma, Cystadenofibroma, Cystadenocarcinoma, Benign Cystic Teratoma, Benign Fibrothecoma, Krukenberg Tumour, Borderline Serous, Borderline Mucinous Neoplasm.

INTRODUCTION

The most common lesions encountered in the ovary are functional or The most common lesions encountered in the ovary are functional or benign cysts and tumors.Neoplastic disorders can be grouped according to their origin from each of the three main ovarian cell types (1)Mullerian epithelium (2)Germ cells (3)sex cord -stromal cells.[1]

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Table-2

Tumor	Age <20	21-30	31-40	41-50	51-60	>60
Benign surface epithelial	1	5	10	17	3	0
Borderline surface epithelial		1	1			
Malignant surface epithelial					2	
Germ cell-benign	1	3	4			
Sex cord stromal benign		1				
Metastatic-malignant			1			

The predominant age group affected was 41 to 50 years with 17 cases followed by 31-40 years with 10 cases,with the predominant neoplasm being Benign surface epithelial tumor

Table-3

S. No.	Type	Age <20	21-30	31-40	41-50	51-60	>60	Total
1	Serous cystadenoma	1	3	4	8	3	0	19
2	Serous cystadenofibroma			3	4	1		8
3	Mucinous cystadenoma		3	1	3			7
4	Mucinous cystadenofibroma				1			1
5	Borderline serous			1				1
6	Borderline mucinous		1					1
7	Serous cystadenocarcinoma					1		1
8	Mucinous cystadenocarcinoma					1		1
9	Benign cystic teratoma	1	3	4				8
10	Fibrothecoma			1				1
11	Krukenberg			1				1
12	Endometriosis			1				1

The predominant age group affected was 41 to 50 years,with the majority of cases being Benign serous cystadenoma.

Table-4

Behaviour	Number
benignBenign	45
borderlineBorderline	2
malignantMalignant	3

The predominant neoplasm in the affected age group was Benign i.e 45 out of 50 cases.

Table-5

Histopathological pattern of ovarian tumors	Number
Surface epithelial tumors	40
Germ cell tumors	8
Sex-cord stromal tumor	1
Metastatic	1

The predominant neoplasm in the affected age group being surface epithelial tumor followed by germ cell tumor .

Table-6

Consistencyconsistency	Number
Cysticcystic	38
Solid/cystic	7
Solidsolid	5

The majority of tumors were cystic in nature.

Table-7

Clinical presentation	Number
Abdominal mass	30
Pain abdomen with abdominal mass	11
Pain abdomen	6
Menstrual disturbance	5
Pressure symptom	1

The major clinical presentation in the affected cases was found to be abdominal mass followed by abdominal mass with pain and abnormal uterine bleeding.

Table-8

Type of specimen	Number
TAH+BSO	25
TAH+Unilateral Salphingo oophorectomy	10
Cystectomycystectomy	15
Totaltotal	50

The majority of specimens received were Total abdominal Hysterectomy and bilateral Salphingo-oophorectomy followed by cystectomy specimens.

DISCUSSION

Comparison of age distribution with other studies

Table-9

Studies	<20	21-30	31-40	41-50	51-60	>60
Saxena,et al.(1980)[2]	38	111	98	72	24	13
Patil,et al.(2005)[3]	14	46	71	33	13	3
Jha and karki,et al.(2008)[4]	11	33	43	34	23	17
Inamdar,et al.(2015)[5]	26	56	48	34	11	5
The present study(20192016)	2	10	16	17	5	0

The present study,when compared with other studies shown above shows increased distribution in the fourth and fifth decades similar to that seen in Jha and Karki et al.

Comparison of clinical presentation with other studies

Table-10

Clinical presentation	Inamdar et al.(2015)	The present study
Mass per abdomen	76	30
Mass with pain abdomen	41	11
Pain abdomen	34	6

Menstrual disturbances	3	5
Pressure symptoms	17	1
Infertilityinfertility	1	
Othersothers	8	

The present study correlated with Inamdar et al study.

Comparison of histopathological spectrum of ovarian tumors with other studies

Table-11

studiesStudies	Surface epithelial tumors	Germ cell tumors	Sex-cord stromal tumors	Metastatic	Miscellaneous
Dr.Vaddatti-tejeswani, et.al(2013)[6]	237 (85.26%)	27(9.71%)	11(3.95%)	3(1.08%)	0
DR. Ivy sharma1,et al.(2014)[7]	62 (60.78%)	31(30.39%)	6(5.89%)	2(1.96%)	1(0.98%)
Nishal,et al(2015)[8]	33(60%)	7(13%)	3 (5%)		12(22%)
The present study	40(80%)	8(16%)	1(2%)	1(2%)	0

In comparison,the present study coincides with other similar studies regarding the higher percentage of surface epithelial tumors ollowed by germ cell,sex-cord stromal tumors and metastatic tumors Comparison of behaviour of tumor with other studies

Table -12

studiesStudies	Benign	Borderline	Malignantmalignant
Gupta,et al.(2007)[9]	72.9%	4.1%	22.9%
Mondal,et al.(2011)[10]	63.1%	7.3%	29.6%
Dr.Vaddatti-tejeswani, et al(2013)[6]	78.05%		21.95%
Dr.Ivy sharma, et al(2014)[7]	78.4%	0.98%	20.6%
The present study	45(90%)	2 (4%)	3 (6%)

The present study correlates with all other studies which showed similar behaviour with the highest incidence of benign followed by malignant and borderline.

Regarding the bilateral tumors in the present study,one case showed simple serous cystadenoma in one ovary whereas the other ovary showed serous cystadenofibroma

The second bilateral case was malignant with krukenberg tumor and the third bilateral case showed mature cystic teratoma in both ovaries.

Surface Epithelial-Stromal Tumors Surface epithelial-stromal tumors are believed to originate from the surface epithelium of the ovary. They are classified as benign if they lack exuberant cellular proliferation and invasive behavior; as borderline (also known as atypically proliferating or of low malignant potential) if there is exuberant cellular proliferation but no invasive behavior; and as malignant if there is invasive behaviour.Most borderline tumors behave clinically as benign tumors and have good prognosis, but some may recur after surgical removal and some may seed extensive implants within the abdominal cavity. Surface epithelial- stromal tumors occur primarily in women who are middle-aged or older and are rare in young adults, particularly before puberty. Most malignant serous tumors are at least partially cystic. They may contain multiple cyst chambers, or loculations, and also solid areas. Most display an abundance of delicate papillae that project into the cyst cavities and, in some cases, outward from the external surface of the tumor. Malignant serous tumors make up one-third of all ovarian serous tumors and approximately half of all malignant ovarian neoplasms. Two-thirds of malignant serous tumors are bilateral.

Mucinous tumors are epithelial ovarian tumors formed by cells that resemble either those of the endocervical epithelium (endocervical or müllerian type) or, more frequently, those of the intestinal epithelium (intestinal type).

Benign mucinous tumors are multiloculated cysts that are filled with opaque, thick, mucoid material.. Surgical removal is curative.

Upon gross pathologic examination, borderline mucinous tumors are similar to benign mucinous tumors but may have solid regions and exhibit papillae projecting into the cyst chambers. About 40% of borderline tumors of endocervical type are bilateral. In contrast, < 10% of borderline tumors of the intestinal type are bilateral. Borderline mucinous tumors of the endocervical type may be associated with mucinous tumorlets or implants in the pelvic and abdominal cavities. Tumors of the intestinal type may be associated with pseudomyxoma peritonei, an accumulation within the pelvis and abdomen of large amounts of mucoid material with few intermixed tumor cells. Most cases of pseudomyxoma peritonei involve the cecal appendix and are thought to originate in mucinous tumors that are primary to the appendix with secondary involvement of the ovaries. Pseudomyxoma peritonei also may be associated with malignant ovarian mucinous neoplasms, and its presence does not indicate dissemination. Treatment of borderline mucinous tumors is surgical. Tumor recurrence and metastatic disease are rare. Pseudomyxoma peritonei follows a relentless and protracted course. Treatment involves the removal of as much tumor as possible followed by abdominal taps to remove fluid and alleviate symptoms.

Compared with borderline tumors, malignant mucinous tumors may contain more papillary projections within the cyst cavities, larger solid areas, and larger areas of necrosis and hemorrhage.Late extraperitoneal recurrences, particularly in the lungs, are characteristic of malignant mucinous tumors.

Sex Cord-Stromal Tumors

Sex cord-stromal tumors are ovarian tumors that are believed to originate in theca cells, other stromal cells, and granulosa cells and their testicular sex cord counterparts, the Sertoli and Leydig cells. These tumors often are associated with endocrine manifestations. They account for approximately 8% of all ovarian tumors and approximately 7% of all malignant ovarian tumors.

Thecomas

Thecomas are rare, solid ovarian tumors formed by stromal cells that resemble the theca cells that normally surround the ovarian follicles. Most thecomas are unilateral and occur in postmenopausal women. They are uncommon before age 30 years. These tumors commonly have estrogenic manifestations, including postmenopausal uterine bleeding, endometrial hyperplasia, and endometrial cancer. Most thecomas are benign, and surgical excision is curative.

Fibromas

Fibromas are rare, solid ovarian tumors arising from the spindled stromal cells that form collagen. On the rare occasions when these tumors are bilateral, they may be associated with nevoid basal cell carcinoma syndrome, also known as Gorlin syndrome. Fibromas are most common during middle age and rare before age 30 years; the mean age at diagnosis is in the late forties. Unlike other sex cord-stromal tumors, fibromas rarely are associated with hormone production. In almost all cases, they are benign and curable by surgical excision. Fibromas with increased cellularity and cell proliferation (mitotic activity) are rare and may follow a malignant course; fibromas of this kind are known as fibrosarcomas.

Germ Cell Tumors

Germ cell tumors are ovarian tumors formed by cells that are believed to be derived form primordial germ cells. These tumors make up approximately one-fourth of all ovarian tumors but only 3–7% of malignant ovarian tumors. More than half of the ovarian neoplasms that develop in children and adolescents are of germ cell origin, with one-third of these being malignant. Conversely, in adults, germ cell tumors are relatively infrequent, and the great majority of them are benign, with most being mature cystic teratomas (dermoid cysts).

The prototypical germ cell tumors are the dysgerminomas. Embryonal carcinomas are germ cell tumors composed of poorly differentiated, multipotential germ cells. Germ cell tumors with differentiation in an embryonal or somatic direction result in teratomas. Those that differentiate in an extraembryonic (placental or trophoblastic) direction result in yolk sac tumors or choriocarcinomas. Mixed subtypes of germ cell tumors also occur frequently.

Teratoma

Teratomas are germ cell tumors that are formed by cells derived from more than one of the three primitive embryonic layers (ectoderm, mesoderm, and endoderm). Teratomas can be mature (benign) or immature (benign or malignant). Teratomas formed predominantly by

endodermal or ectodermal elements are referred to as monodermal or specialized.

Mature teratomas can be solid or cystic. Mature solid teratomas are rare, as most solid teratomas are at least partially immature. Mature teratomas occur mostly in children and young adults. These tumors, most of which are unilateral, grow slowly and usually are large at the time of diagnosis. Surgical excision is curative.

In most mature cystic teratomas, the ectodermal elements predominate; when this is the case, these teratomas are designated as dermoid cysts. Mature cystic teratomas commonly have a single cyst cavity filled with sebaceous material, and they often have a focal internal protuberance that may contain hair, teeth, bone, and/or cartilage. Mature cystic teratomas most commonly occur during the reproductive years. In most cases, surgical excision is curative. Rupture of the tumor may result in peritoneal implants.

CLASSIFICATION OF OVARIAN TUMORS

It is recommended that the World Health Organisation (WHO) classification and nomenclature of ovarian tumors be used because of its wide acceptance.

WHO Histologic Classification of Ovarian Tumors[11]

SURFACE EPITHELIAL-STROMAL TUMORS

Serous tumors
Benign-cystadenoma
Serous tubal intraepithelial carcinoma (STIC)
Serous borderline tumor/atypical proliferative serous tumor
Serous borderline tumor,micropapillary variant/noninvasive low grade serous carcinoma
Low grade serous carcinoma
High grade serous carcinoma

Mucinous Tumors-endocervical like and intestinal type
Benign -cystadenoma
Mucinous borderline tumor/atypical proliferative mucinous tumor
Mucinous carcinoma

Seromucinous tumors
Seromucinous borderline tumor/Atypical proliferative seromucinous tumor
Seromucinous carcinoma

Endometrioid tumors
Benign-cystadenoma
Endometrioid borderline tumor
Endometrioid carcinoma

Clear cell tumors
Benign
Clear cell borderline tumor
Clear cell carcinoma

Transitional cell tumors
Brenner tumors
Borderline brennerr tumor/atypical proliferative brenner tumor
Malignant Brenner tumor
Transitional cell carcinoma-non-Brenner type
Mixed Epithelial borderline tumor
Mixed Epithelial carcinoma
Carcinoma,subtype cannot be determined
Undifferentiated carcinoma

Epithelial -stromal
Adenosarcoma
Carcinosarcoma(Malignant mixed Mullerian tumor)

Sex-cord- stromal tumors
Granulosa tumor
Fibromas
Fibrothecomas
Thecomas
Malignant Sex cord-stromal tumors
Granulosa tumors
Granulosa cell tumor,adult type
Granulosa cell tumor,juvenile type
Sertoli cell tumor
Leydig cell tumor
Sex cord tumor with annular tubules
Gynandroblastoma
Steroid (lipid) cell tumor
Sertoli-Leydig cell tumor
Other sex cord-stromal tumor

Malignant germ cell tumors
Dysgerminoma
Yolk sac tumor
Embryonal carcinoma
Choriocarcinoma,non-gestational
Immature teratoma
Carcinoma arising in a teratoma
Mixed malignant germ cell tumor

Malignant,not otherwise specified
Metastastic cancer from nonovarian primary:
Colonic,appendiceal
Gastric
Breast

STAGING OF OVARIAN TUMORS

Staging of ovarian surface epithelial-stromal tumors is performed according to the TNM system, the set of guidelines established by the American Joint Committee on Cancer, which is comparable to an alternative staging system approved by the International Federation of Gynecology and Obstetrics (FIGO)

Ovarian Surface Epithelial-Stromal Tumor Staging Protocols: AJCC TNM Systema and FIGO Staging System[12].

AJCC	FIGO	Description
TX		Primary tumor cannot be assessed.
T0		No evidence of primary tumor.
T1	I	Tumor limited to ovaries (one or both).
T1a	IA	Tumor limited to one ovary; capsule intact, no tumor on ovarian surface. No malignant cells in ascites or peritoneal washings.
T1b	IB	Tumor limited to both ovaries; capsules intact, no tumor on ovarian surface. No malignant cells in ascites or peritoneal washings.
T1c	IC	Tumor limited to one or both ovaries, with any of the following: capsule ruptured, tumor on ovarian surface, malignant cells in ascites or peritoneal washings.
T2	II	Tumor involves one or both ovaries with pelvic extension.
T2a	IIA	Extension and/or implants on uterus and/or tube(s). No malignant cells in ascites or peritoneal washings.
T2b	IIB	Extension to other pelvic tissues. No malignant cells in ascites or peritoneal washings.
T2c	IIC	Pelvic extension (2a/IIA or 2b/IIB) with malignant cells in ascites or peritoneal washings.
T3 and/or N1	III	Tumor involves one or both ovaries, with microscopically confirmed peritoneal metastasis outside the pelvis and/or regional lymph node metastasis.
T3a	IIIA	Microscopic peritoneal metastasis beyond pelvis.
T3b	IIIB	Macroscopic peritoneal metastasis (2 cms or less in greatest dimension) beyond pelvis.
T3c and/or N1	IIIC	Peritoneal metastasis (more than 2 cm in greatest dimension) beyond pelvis and/or regional lymph node metastasis.
M1	IV	Distant metastasis (excludes peritoneal metastasis).

Ascites is the accumulation of excessive fluid within the abdominal (peritoneal) cavity. The presence of nonmalignant ascites is not

classified. The presence of ascites does not affect staging unless malignant cells are present.

Lymph nodes located in the pelvis or in the back of the abdomen on either side of the aorta (para-aortic). Liver metastases confined to the capsule are T3/Stage III. Liver parenchymal metastases are M1/Stage IV.

Pleural effusion must have positive cytology for M1/Stage IV malignancy.

HISTOLOGIC GRADING AND PROGNOSTIC FACTORS

Microscopic examination is critical for predicting tumor behavior and deciding the best therapeutic approach. Such examination includes the assessment of specific histologic type and extent of disease and the grading of tumor differentiation (i.e., the extent to which the tumor resembles the normal tissue). Tumors are graded as well-differentiated (G1), moderately differentiated (G2), poorly differentiated (G3), or undifferentiated (G4). As discussed above, surface epithelial-stromal tumors also can be classified as borderline malignancy . Attempts to identify prognostically relevant pathologic features in ovarian cancers are hindered by the diversity of tumors that are encountered. Most reported prognostic information addresses surface epithelial-stromal tumors.

New Developments In Ovarian Cancer Pathology

There is a growing effort to define the genetic and molecular makeup of ovarian malignancies. As the complex molecular events associated with ovarian cancer are uncovered, the hope is that in addition to providing insight into the pathogenesis of ovarian neoplasia, these events also will serve as prognostic factors, markers for treatment effectiveness (i.e., predictive factors), and targets for future therapies (e.g., immunotherapy).

Molecular and genetic studies have increased our understanding of the pathogenesis of some ovarian tumors, especially surface epithelial tumors. Loss of heterozygosity on chromosome 17q has been found specifically in serous tumors. Allelic studies have shown that many (if not all) endometrioid and clear cell carcinomas may arise from preexisting endometriosis. Molecular and immunohistochemical markers also can be valuable in determining whether mucinous carcinomas are primary ovarian tumors or secondary metastases from the colon or appendix.Finally, identification of inherited germline mutations in BRCA1 and BRCA2 genes that lead to a high susceptibility to ovarian and breast cancer, has shed light on the pathogenesis of ovarian cancer and at the same time raised complex medical, social, economic, and ethical issues.

Histopathologic grading of malignant ovarian tumors has had only modest prognostic application; however, the use of molecular markers shows some promise. Detection of proliferation markers, such as MIB-1, and mutant suppressor gene products, such as p53, has been shown to be correlated with prognosis, as has detection of the cell cycle inhibitor p27. A number of other cell proteins with Epithelial ovarian tumors are heterogeneous neoplasms which are primarily classified according to cell type into serous, mucinous, endometrioid, clear-cell, transitional, and squamous cell tumors. These tumors are further subdivided into benign, borderline (intermediate), and malignant (carcinoma) depending on the degree of cell proliferation and nuclear atypia, and the presence or absence of stromal invasion .

Despite the lack of ovarian stromal invasion, serous borderline tumors, particularly those with exophytic growth, can implant on peritoneal surfaces and, rarely (~10% of peritoneal implants), progress to low-grade serous carcinoma (LGSC), and invade the underlying tissues. The biologic behavior of invasive peritoneal implants is similar to that of LGSC.

Malignant epithelial tumors (carcinomas) are the most common ovarian cancers accounting for 90% of cases . Although traditionally referred to as a single entity, ovarian cancer is not a homogeneous disease but rather a group of diseases, each with different morphology and biologic behavior.

Currently, based on histopathology, immunohistochemistry, and molecular genetic analysis, at least five main types of ovarian carcinomas are identified: high-grade serous carcinomas (HGSCs; 70%), endometrioid carcinomas (EC; 10%), clear-cell carcinomas (CCC; 10%), mucinous carcinomas (MC; 3%), and LGSC (<5%) .

Hereditary nonpolyposis colorectal carcinoma, various normal and aberrant functions are being investigated as potential prognostic tools.

Role of Immunohistochemistry in ovarian tumors
Serous tumors

The typical keratin profile of serous tumors is CK 7+/CK 20-.They also express CK8,18 and 19,EMA,B72.3,S-100 protein (particularly) borderline tumors.WT1 stains diffusely most ovarian serous carcinomas.

Mucinous tumors express CEA,keratin,EMA,MUC5AC,DPC4 and CK7+/CK20+.

Endometrioid tumors are positive for keratin,EMA,vimentin,PAX-8.

Clear cell tumors are positive for AFP,PLAP,EMA, cytokeratins, hepatocyte nuclear factor 1 beta.

Brenner tumor are positive for keratin,EMA,CEA.
Transitional cell carcinoma are CK20-,uroplakin-,CK7+.
Sertoli-Leydig cell tumors show inhibin,AFP positivity.
Lipid or steroid cell tumors express vimentin,keratin,actin and inhibin.

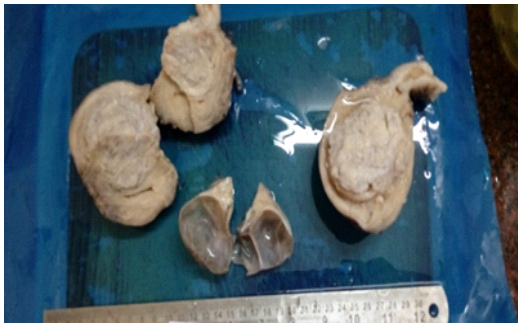

Fig No-1 Benign serous cystadenoma -Gross picture

Fig 1-shows uterus,cervix with ovarian cyst.Cut surface shows uterus with fibroid.Cut surface of ovarian cyst shows thin walled cyst with clear fluid

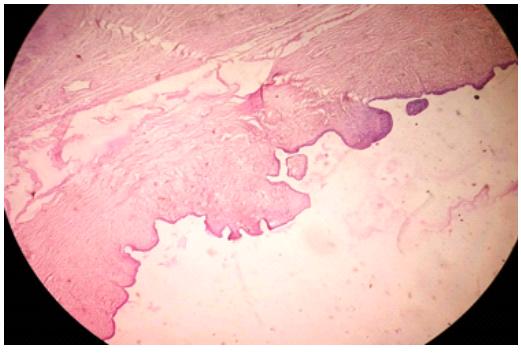

Fig No-2 Benign serous cystadenoma-microscopy

Fig 2 shows cyst wall with flattened to cuboidal epithelium with occassional papillae

Fig No.3 -Benign serous cystadenofibroma-Gross picture

Fig no 3 shows uterus with cervix with one side ovary showing cyst wall with lumen showing gelatinous material with irregularly thickened cyst wall.

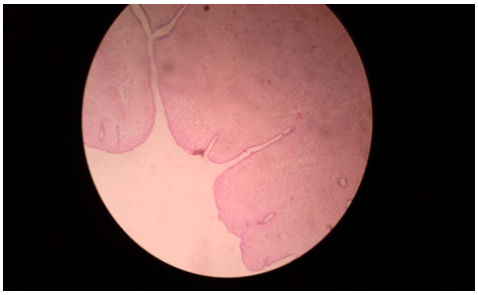

Fig No.4 Benign Serous Cystadenofibroma-microscopy

Fig no.4 shows ovarian cyst wall with papillae lined by flattened epithelium with the underlying area showing dense stroma

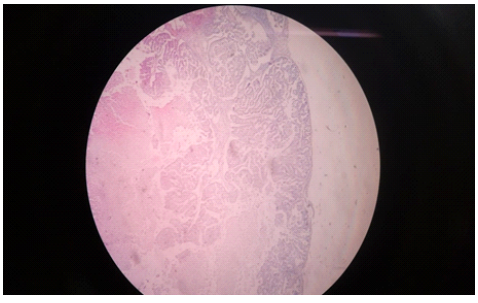

Fig No-5 Borderline serous neoplasm-microscopy

Section shows cyst wall with lining epithelium showing papillae with stratification with no infiltration of underlying stroma

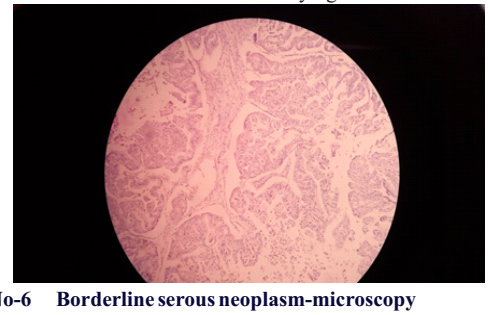

Fig No-6 Borderline serous neoplasm-microscopy

Section shows ovarian cyst with cyst wall showing lining epithelium with stratification with no infiltration of underlying stroma

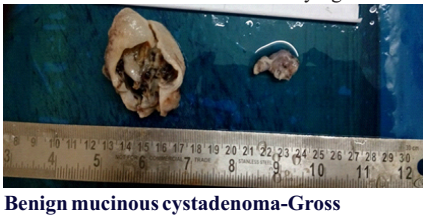

Fig No-7 Benign mucinous cystadenoma-Gross

Fig No.7 shows ovarian cyst with irregularly thickened cyst wall with mucinous material.No solid areas seen.

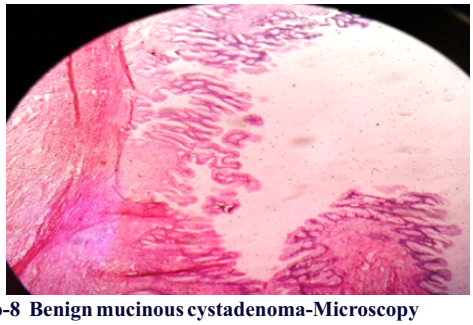

Fig No-8 Benign mucinous cystadenoma-Microscopy

Section shows cyst wall lined by tall columnar epithelium with mucin.

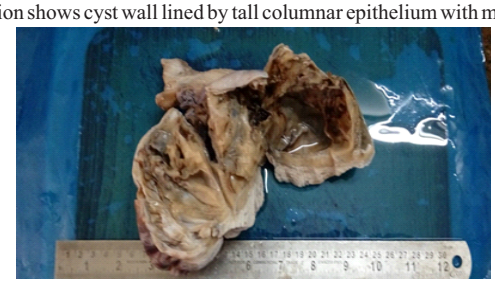

Fig No-9: Granulosa cell tumor-Gross

Fig. shows irregularly thickened cyst wall with focal solid areas.

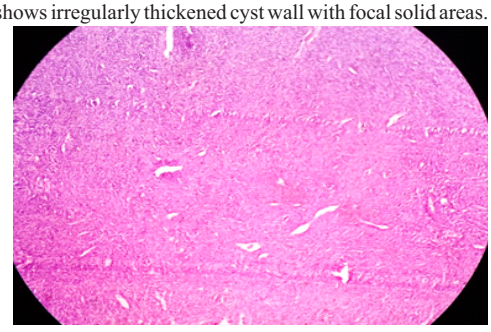

Fig No-10 Granulosa cell tumor -low power-microscopy

Section shows neoplasm arranged in sheets with round to oval cells with nuclei showing grooves

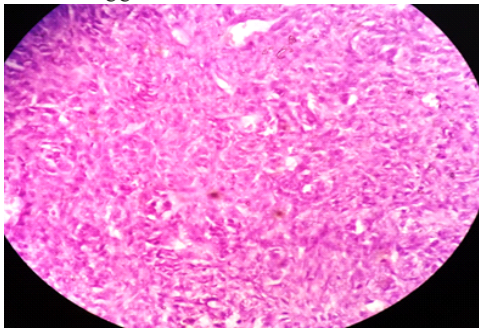

Fig No-11 Adult granulosa cell tumor-high power microscopy

Section shows neoplasm arranged in sheets with round to oval cells with nuclei showing grooves.Focal areas show call-exner bodies.

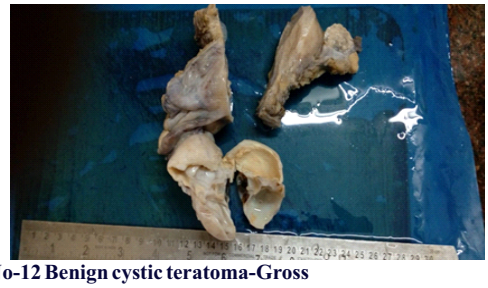

Fig No-12 Benign cystic teratoma-Gross

Fig shows uterus with cervix with one ovary showing cyst with focal areas of cartilage, fatty tissue and the lumen showing pultaceous material with hair

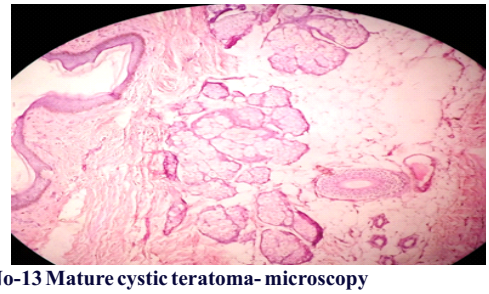

Fig No-13 Mature cystic teratoma- microscopy

Section shows cyst wall lined by stratified squamous epithelium with cyst wall showing sebaceous gland, hair follicle, sweat gland, fatty tissue and focal areas showing glandular epithelium. The lumen shows keratinous debris material and anucleate squames.

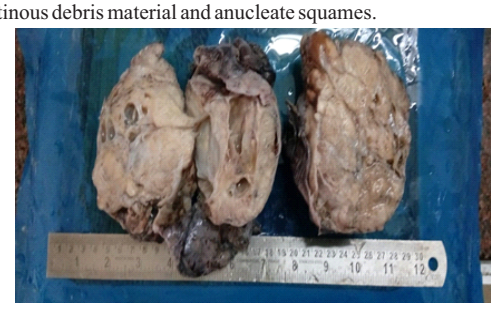

Fig No-14 Papillary serous cystadenocarcinoma-Gross

Fig 14-shows ovarian neoplasm with cut surface showing solid and cystic areas with the solid areas showing granular papillary areas.

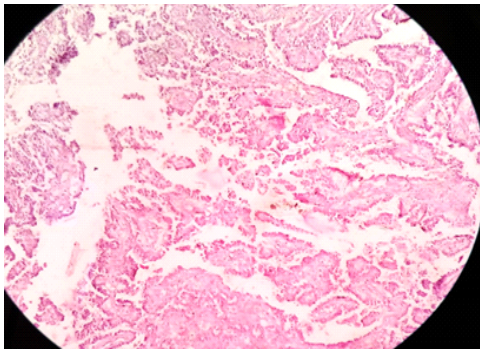

Fig No-15 Papillary serous cystadenocarcinoma-Microscopy-low power

Section shows malignant ovarian neoplasm with papillae lined by stratified flattened to cuboidal epithelial cells with hyperchromatic pleomorphic nuclei. Focal ares show psammomatous calcification.the neoplasm is seen to infiltrate into underlying stroma.

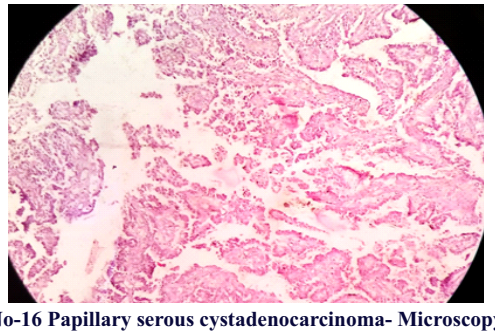

Fig No-16 Papillary serous cystadenocarcinoma- Microscopy low power.

Section shows malignant ovarian neoplasm with papillae lined by stratified flattened to cuboidal epithelial cells with hyperchromatic pleomorphic nuclei. Focal areas show psammomatous calcification. the neoplasm is seen to infiltrate into underlying stroma

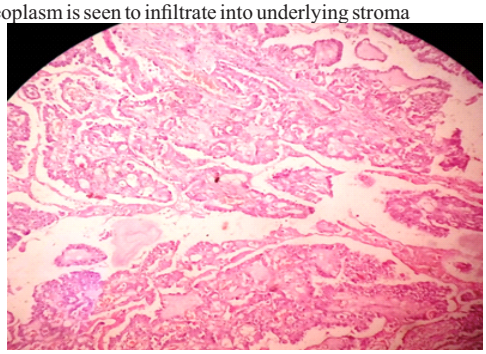

Fig No-17 Papillary serous cystadenocarcinoma-Microscopy-High power

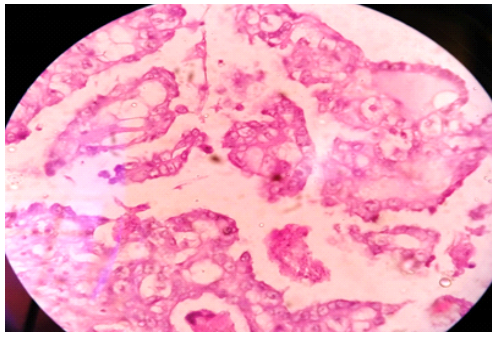

Fig no. 18 Papillary serous cystadenocarcinoma-Microscopy-High power

Fig No.17 and 18---Section shows malignant ovarian neoplasm with papillae lined by stratified flattened to cuboidal epithelial cells with hyperchromatic pleomorphic nuclei.Focal areas show psammomatous calcification.the neoplasm is seen to infiltrate into underlying stroma

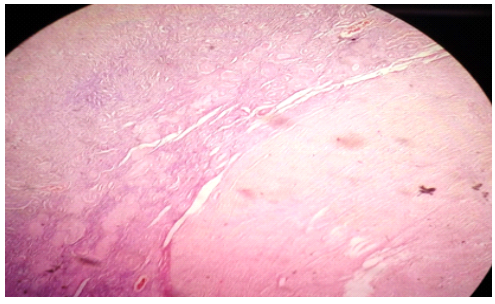

Fig No.19 Fibrothecoma -Microscopy

Section shows benign ovarian neoplasm consisting of spindle shaped stoma arranged in fascicles with focal luteinisation of stroma and spindle cells.

CONCLUSION

Ovarian tumors are the common forms of neoplasia in women.Ovarian neoplasms account for a disproportionate number of fatal cancers,being responsible for almost half of the deaths from cancer of female genital tract because many of these ovarian neoplasms cannot be detected early in their development.Hence there is a need for histopathological diagnosis for these tumors,which cannot be diagnosed and categorised merely by clinical and radiological background.

In the era of personalized cancer medicine, reproducible histopathological diagnosis of tumor cell type is a sine qua non condition for successful treatment. For instance, it has been found that different tumor types respond differently to chemotherapy. The poor response rate of clear cell carcinoma (15%) contrasts notably with that of high grade serous carcinomas (80%), resulting in a lower 5-year survival for clear cell compared with high grade serous carcinomas in patients with advanced stage tumors (20% versus 30%) . The clear cell and mucinous types, in particular, are candidates for clinical trials to identify more active therapy than what is presently used .

Although the mesothelial origin cannot be excluded, there is now compelling evidence that a number of what have been thought to be primary ovarian cancers are actually originated in other pelvic organs and involve the ovary secondarily. In fact, it has been proposed that high grade serous carcinomas arise from precursor epithelial lesions in the distal fimbriated end of the fallopian tube , whereas endometrioid and clear cell carcinomas originate from ovarian endometriosis . This presentation summarizes recent advances in the molecular pathology which have greatly improved our understanding of the biology of ovarian carcinoma and are also relevant to patient management.

Therefore,specific histopathological diagnosis is necessary as it may aid in the further management of cases,through which cancer deaths can be prevented by utilizing better treatment modalities.

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REFERENCES

1. Robins and Cotran pathologic basis of disease,South Asia Edition,Elsevier,2014.
2. Saxena HMK.Devi G.Prakash P.Pankajan P.Ovarian Neoplasms,Aretrospective study of 356 cases.J obstetGynec.India 1980 30:522-527.
3. Patil V.Clinicopathological study of ovarian tumors a three year study.Gulburga:Rajiv Gandhi Institute of Health sciences.2005.
4. R Jha and S Karki.Histological pattern of ovarian tumors and their age distribution.Nepal Medical Coll J 2008:10(2):81-85.
5. Inamdar S.S,Bharati M bhavikatti,Prabhu.M.H.Occurence of ovarian tumors in Karnataka.Journal of drug discovery and theraupeutics 2015:3 (27):17-23.
6. Dr.Vaddatti tejeswini,Dr.E.Sudhakar Reddy,Dr.P.Premalatha,Dr.G.Vahini.Study of morphological patterns of ovarian neoplasms.JDMS,Sep-Oct 2013:10 (6) :11-16.
7. Dr.Ivy sharma,Dr.Usha sarma and Dr.U.C.Dutta.Pathology of ovarian tumor-A hospital based study.International Journal of Medical science and clinical invention 2014(6):284-286.
8. Arpita J.Nishal,Kinnari S.Naik.Jigna modi.Analysis of spectrum of ovarian tumors:A study of 55 cases.Int J Res Med Sci .2015 oct:3(10):2714-2717.
9. Gupta N,Bisht D,Agarwal AK,Sharma VK.Retrospective and prospective study of ovarian tumors and tumor like lesions.Indian J pathol Microbiol.2007:50;525-7.
10. Mondal SK,Bandyopadhyay R,Nag DR,Roy Chowdhury S,Mondal PK,Sinha SK.Histologic pattern,bilaterality and clinical evaluation of 957 ovarian neoplasms,A 10 year study in a tertiary hospital of eastern India.J Can Res Ther 2011:7:433-7.
11. Tavassoli FA,DevileeP,eds.Pathology and Genetics of Tumors of Breast and Female Genital Organs.Lyon,France,IARC press,2003,WHO Classification of tumors.Vol 5.
12. Rosai J.,Female Reproductive System:Surgical Pathology ,10th Edition,Elsevier,2012.