

Clinicopathological and Immunohistochemical Profile of Malignant Surface Epithelial Ovarian Tumors

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Abstract

Background and Objectives: Ovary is a complex organ in terms of its embryology, Histology, Steroidogenesis and malignancy. Histomorphological study of ovarian tumors is undertaken with the aim of studying age distribution and histomorphological characterization of ovarian tumors. **Material and methods:** The study is undertaken for the period of 2 yrs. From August 2015 to July 2017 prospectively at the Upgraded Department of Pathology, Osmania Medical college. All the specimens of ovarian tumours received were subjected to routine processing and histopathological examination. **Results:** Total numbers of 80 cases were studied. Among these 38 cases (51.6%) were Benign tumours, 9 cases (17.5%) were Borderline tumors and 25 (31.5%) cases were malignant. Benign neoplasms peaked in 4th decade and Malignant in 5th decade. The serous surface epithelial tumors were the Commonest among other surface epithelial tumors. **Conclusion:** Benign surface epithelial tumours are the most prevalent type in this study. Categorization will help in the accurate diagnosis and subsequent treatment.

Keywords: Ovarian tumors, surface epithelial tumors, Immunohistochemistry, ER, PR, P53, Ki67

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Introduction

The ovaries are important organs for reproduction. The ovaries are paired pelvic organs located on the sides of the uterus close to the lateral pelvic wall, behind the broad ligament and anterior to the rectum[1].

The ovary is complex in its embryology, histology, steroidogenesis and has potential to develop malignancy. The ovarian tumors are not a single entity but a complex wide spectrum of neoplasms involving variety of histologic tissues ranging from epithelial tissues, connective tissues, specialised hormone secreting cells to germinal or embryonal cells[2]. The main function of the ovaries is to produce ova to implant after fertilization in the endometrium, the preparation of which is co-ordinated afresh each time by ovarian hormones. It also functions as an endocrine gland in the development of secondary sexual characters as well as their maintenance. Thus the ovary is always in a dynamic state[1].

Ovarian neoplasms have become increasingly important not only because of large variety of neoplastic entities but more because they have gradually increased mortality rate in female genital cancers[3].

Ovarian cancers account for 25% of the all gynaecological malignancies and 3rd commonest cause of death due to malignancies of female genital tract in the western world[4]. In India, ovarian tumors account for 80% of all the gynaecological malignancies[5].

The ovary, after the uterus and cervix, is the second common site for the development of gynaecological malignancy & prognosis remains poor[1]. About 75% of the tumors are benign and 25% malignant. Generally ovarian tumors occur in perimenopausal and post menopausal women, infrequently in children also. The risk of developing an ovarian malignancy peaks in fifth decade of life.

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Ovarian tumors in adolescents and children are not frequently encountered in the clinical practice. The rarity of the condition, asymptomatic nature in earlier stage, variation in clinical presentation and unawareness among girls and parents sometimes makes diagnosis delayed and difficult.

Risk factors for ovarian cancer are much less clear than for other genital tumors, but nulliparity, family history and heritable mutation play a role in the tumor development[6,7]. Women between 40 to 59 years of age who have taken oral contraceptives or undergone tubal ligation have a reduced risk of developing a cancer[8,9].

The most intriguing risk factors are genetic. In past, different terminologies and nomenclatures were used but at present WHO histological typing has been used which has reduced the dilemma of categorizing ovarian tumors.

Serum HCG, Serum CA125, Serum alpha fetoprotein, placental alkaline phosphatase and lactate dehydrogenase are useful tumor markers, but their accessibility to the practising pathologist for rural based poor population remains very limited even today[10].

Screening for ovarian epithelial cancer can be improved by measurement of additional tumor markers such as ovarian cancer antigen OVx, and Ca 15-3, and numerous other antigens and by combination of tumor marker measurement and Doppler colour flow ultrasonography & transvaginal ultrasonography[11].

Aims and Objectives

The study is carried out in department of Pathology, Osmania Medical College, and Hyderabad.

- To Study and characterize the ovarian tumors based on gross and histopathological features.
- To study prevalence and age distribution and of various ovarian tumors.
- To compare the frequency of benign and malignant neoplasms of the ovary with other studies.

➤ Toanalyse the profile of estrogen receptors (ER), progesterone receptors (PR), ki 67 and p53 in various ovarian epithelial tumors and attempt correlation with clinical and histological findings.

Materials and Methods

This study is conducted for a period of 2 years (August 2015 to July 2017) prospectively. The cases were obtained from the Department of Pathology at Modern Government maternity hospital & MNJ Institute of Oncology and Regional Cancer Centre - Osmania Medical College, Hyderabad.

The gross specimen received were fixed in 10 % formalin for 24 hours and from every specimen multiple sections were taken from representative site for histological examination. The number of blocks varied from four to eight in number. Sections were processed in paraffin, which were cut at five microns thickness. Sections were stained with conventional hematoxylin and eosin stain

The lesions were classified and studied as per the WHO classification of ovarian tumors (2014).

Fixation for light microscopy

All the specimens obtained were fixed in buffered 10% formalin. Fixation time was 12-24 hrs, and then processed.

Technique of processing

- Dehydration- 3 changes of graded alcohol and 2 changes of acetone

- Clearing- Chloroform
- Paraffin impregnation- 2 changes at 60°C.
- Embedded in paraffin wax, labelled and blocks were made after trimming excess paraffin.
- Sections were cut at a microtome setting of 4 microns.
- The sections were floated on a water bath at 60°C.
- Sections were mounted on a slide using a very thin layer of glycerol egg albumin as an adhesive.

Study by light microscopy

For light microscopy one slide from each block was routinely stained with H&E for general study to arrive at a diagnosis.

Immunohistochemistry

Immunohistochemical staining of ER and PR was done using peroxidase-antiperoxidase method according to the protocol described by DAKO.

Observations and Results

The present study included 2 years prospective histopathological evaluation of 80 cases of tumors of ovary in the department of Pathology, Modern government maternity hospital / MNJ Institute of Oncology and regional cancer centre - Osmania Medical College, Hyderabad.

A total of 80 SEOT ovarian tumors were studied. Of these ovarian tumors, 42 were benign, 11 were borderline and 27 were malignant.

Table 1: Distribution of Benign, Borderline and Malignant Tumours

	No. of Cases	Percentage
Benign	42	51.55%
Borderline	11	17.95 %
Malignant	27	30.45 %

Age Distribution

The age range of the tumors diagnosed varied from 22 to 71 years, with a peak incidence in 4th and 5th decade of life. Maximum benign cases were seen between 30-40 years. Maximum number of

malignant tumors were seen between 40-60 years. Youngest patient was 22 years old, oldest was 71 years old.

The maximum number of tumors were seen in the 30-40 years.

Table 2: Age Wise Distribution of Ovarian Neoplasms in The Present Study

Age	Benign	Percent	Borderline	Percent	Malignant	Percent
Up to 20	00	00%	00	00%	00	00%
21-30	06	14.2%	02	18.18%	00	00%
31-40	32	76.1%	01	9.0%	02	07%
41-50	02	4.7%	06	54.5%	07	25.7%
51-60	01	2.6%	01	9.0%	16	59.2%
61-70	01	2.6%	01	9.0%	02	07%
71-80	00	00%	00	00%	00	00%
TOTAL	42	51.55%	11	17.95%	27	30.45%

Table 3: Distribution of Tumors in Parous Women

Type of tumors	Unmarried	Married	
		Nulliparous	Parous
Benign	02	08	32
Borderline	-	-	11
Malignant	01	01	25

Table 4: Clinical Presentation of Patients

Clinical Presentation	Benign	Boderline	Malignant
Mass per abdomen	34	3	27
Associated pain abdomen/ back pain	47	3	20
DUB	5	-	-
Amenorrhoea	4	-	2
Postmenopausal bleeding	10	1	9
Urinary symptoms	7	-	-
Loss of weight	4	-	2
Ascites	1	-	8

The most common clinical presentation was pain in abdomen, seen in 47 cases (26.5%) followed by mass per abdomen in 34 cases (17%).

Specimen Received

In the present study, TAH with BSO was the most common procedure opted in 52 cases (65%) followed by unilateral

oophorectomy in 20 cases (25%), bilateral oophorectomy in 08 cases (10%).

Laterality of Tumors

Right sided tumors of ovary 48 (60%) were more common than the left sided tumors 25(31%). 07(09%) cases were bilateral.

Table5: Laterality of Tumours

Side of ovary involved	Number of cases	Percentage
Right	48	60%
Left	25	31%
Bilateral	07	09%

Size: There was a wide size range in ovarian neoplasms in the present study. It ranged from 3x2cm to 30x20cm. Majority of them 40(50.1%) were in the size range of 6-10cm, followed by 33(41.2%) in the size range of 11-19cm.

Table 6: Size Range of The Ovarian Neoplasms in The Present Study

Size (cm)	Number of cases	Percentage
≤5	03	3.7%
6-10	40	50.1%
11-19	33	41.2%
≥20	04	05%
Total	80	100%

Consistency of the Tumor: In the present study, the benign tumors were cystic in 40 cases while the malignant tumors were predominantly solid and cystic in 26 cases.

In the present study, majority 53 (66.2%) of ovarian tumors were cystic in consistency of which most of them were benign 38 (49%).

Table 7: Showing Consistency of Benign, Borderline and Malignant Tumors in Present Study

Consistency	Benign	Borderline	Malignant	Total	Percentage
Cystic	38	07	08	53	66.2%
Solid and cystic	04	02	16	22	27.5%
Solid	-	02	03	05	6.2 %
Total	42	11	27	80	100%

Histopathological Distribution of Ovarian Tumors

The tumors were classified according to WHO histologic classification of ovarian tumors and the incidence of different histologic types noted.

Table 8: Number of Cases And Percentage Distribution of Various Types of Seots (n=80)

Histological type	Benign	Borderline	Malignant	Total
Serous	38(53.1%)	07(10.9%)	23(35.9%)	64(75%)
Mucinous	08(50%)	04(25%)	04(25%)	16(25%)
Endometrioid				00
Clear cell				00
Transitional cell				00
SEROMUCINOUS				00

Surface Epithelial Tumors

The commonest epithelial tumors were serous 64 cases (75 %), 16 mucinous (25%), remaining are not presented on my study period.

Table 09: Distribution of Surface Epithelial Tumors

Type of surface epithelial tumor	No of cases	Percentage
Serous Cystadenoma	30	37.5 %
Serous cystadeno fibroma	04	5%
Borderline serous Tumor	07	8%
Serous cystadenocarcinoma low grade	15	18%
Serous cystadenocarcinoma high grade	08	10%
Mucinous Cystadenoma	08	10%
Borderline Mucinous tumor	04	5%
Mucinous cystadenocarcinoma	04	5 %
Brenner Tumor	00	%
Endometrioid Tumor	00	%
un differentiated carcinoma	00	%
Total number of surface epithelial tumors	80	100 %

Serous Tumors

Serous tumors formed the majority of ovarian neoplasms in the study. There were a total of 64 serous tumors, constituting about 75%.

Table 10: Distribution of Serous Tumors

Type	No. ofcases	Percentage
Serous cystadenoma	30	45.5%
Borderline serous tumor	07	10.9%
Serous cystadenocarcinoma	23	35.9%
Serous cystadenofibroma	04	8.5%
Total	64	100%

All serous tumours >1cms were considered as serous cystadenomas. The serous cystadenoma was the most commonest accounting for 34 cases (53.5%) of all ovarian neoplasms.

Gross

The size of the largest benign serous cystadenoma was 20x16 cm and the smallest was 3x3 cm with smooth shiny external surface. Majority of the tumours exuded clear fluid with one case of seromucinous fluid and 3 cases showed features of torsion with

prominent dilated veins over the surface. Bilaterality was seen in 2 cases.

The largest malignant tumor 10x8 cm in size and its cut section was partly solid and partly cystic.

Mucinous Tumors

These constituted the second most common ovarian neoplasms and included 16 cases (25%) of all ovarian tumors.

Table 11: Distribution of Mucinous Tumors

Tumor type	No. of cases	Percentage
Mucinous cystadenoma	08	50%
Borderline mucinoustumor	04	25%
Mucinous cystadenocarcinoma	04	25%
Total	16	100%

Grossly, the largest tumor measured 30x20 cm, which was also the largest tumor encountered in the present study.

The benign tumors were cystic while borderline and malignant ones were partly solid and partly cystic containing mucinous fluid.

Five cases of borderline mucinous were diagnosed in tumor with papillary projections lined by atypical cells up to one- two cell thickness and no stromal invasion was seen. Malignant tumours showed complex papillary pattern with pleomorphic mucinous cells. Areas of necrosis and stromal invasion were seen.

Gross Pictures



Fig 5: Serous Cystadenoma



Fig 6: Serous Cystadenocarcinoma

Microscopic Pictures

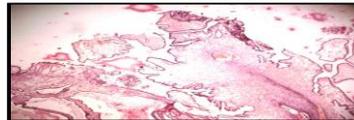


Fig 7: H & E (10x) – Papillary Serous Cystadenoma
Cyst wall lined by cuboidal epithelium with papillary projections

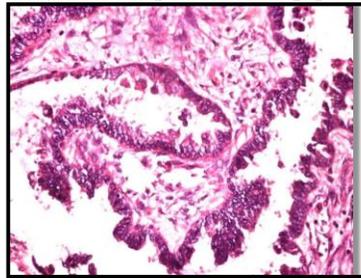


Fig 8: H & E Section- Boderline Serous Tumor
Stratified epithelium with cells showing nuclear atypia and no stromal invasion

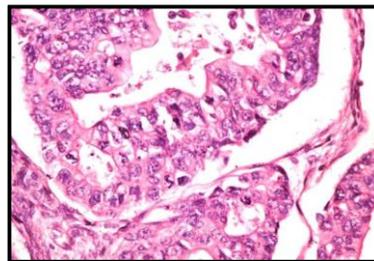


Fig 9: H & E (40x) – Serous Cystadenocarcinoma
Sheets and islands of pleomorphic cells with vesicular nuclei invading the stroma

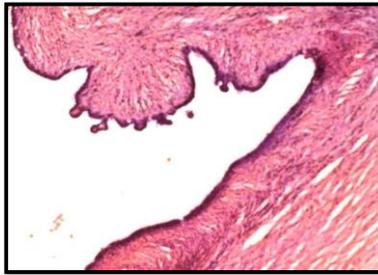


Fig 10:H & E (10x) – Serous Cystadenofibroma
Cyst wall lined by cuboidal to flattened epithelium and a prominent stromal component

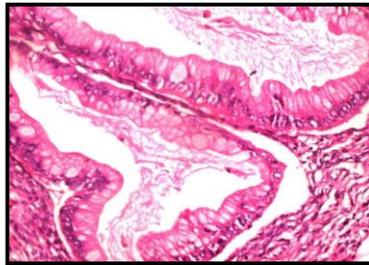


Fig 11:H & E (40x) - Mucinous Cystadenoma

- Cyst wall lined by columnar cells with apical mucin and basal nuclei.
- Immunoreactivity for ER and PR was assessed in each case by Estimating the Percentage of cells showing nuclear staining. When at least 5% of the cells Showed nuclear positivity, the case was Considered positive.
- The reaction for p53 was recorded as either positive or negative.
- The nuclear staining for ki-67 was graded by counting ki-67 labeling index (li) As a percent of positively stained tumor nuclei in 1000 tumorcells in the Hot spotarea of the tumor. The mean ki-67 li is recorded inevery case.
- In the case of serous tumors, ER was expressed in all high andLow-grade tumors.
- The expression of PR was more in low-grade tumors than high-grade ones.
- P53 expression was seen in all high-grade tumors and low-gradetumor.
- The ki-67 li was more in high-grade tumors than low-grade tumors.
- The expression of ER was more in malignant tumors (23/27, 81%) than borderline (07/11, 66%) and benign (21/42, 50%)
- The expression of PR was more in benign (33/42, 68.19%) than borderline(6/11, 54.5%) and malignant tumors (12/27, 44.25%).
- The expression of p53 was less in benign(18/42, 42%) than borderline(7/11, 66.6%) and malignant tumors (22/27, 81%).
- The expression of ki-67 was more in malignant (24/27, 88.8%) than borderline(09/11, 81.33%)and benign tumors (11/42, 26.1%).

Table 12: Expression of ihc markers in benign, borderline, and malignant

SEOT	ER (%)	PR (%)	p53 (%)	Ki-67 (%)
Benign (n=42)	21(50%)	33(68.5%)	18(42%)	11(26.7%)
Borderline (n=11)	07(66.6%)	06(54.5%)	07(66.6%)	09(81.3%)
Malignant (n=27)	23(81%)	12(44%)	22(81%)	24(88.8%)

Er expression in seot

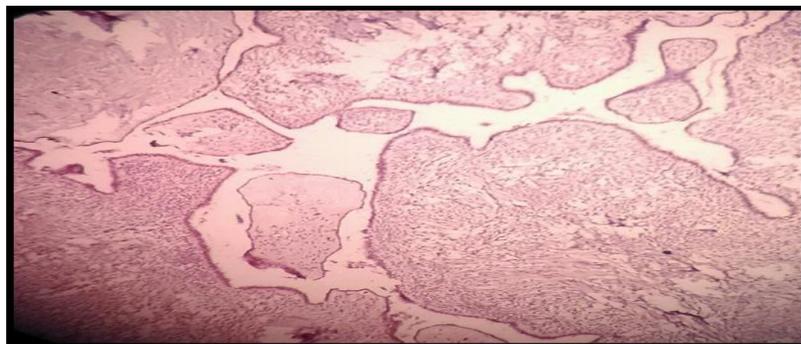


Fig 12:ER Postive(40X) Serous Benign Cystadenomas



Fig 13: ER Positive(40X) Serous BordelineTumor

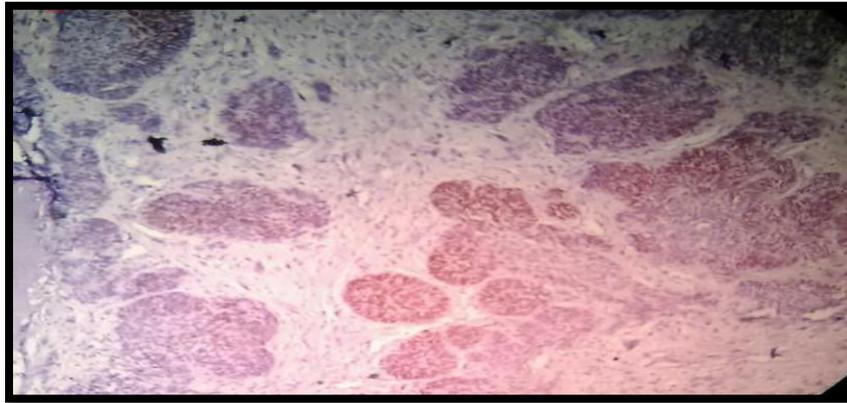


Fig 14:ER Positive (10X) Higly in Serous Cystadeno Carcinoma

Pr expression in seot

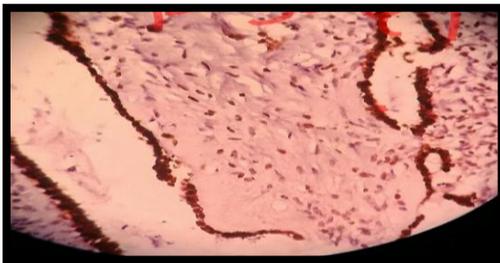


Fig 15: Highly PR Postive(40X) in Benign Cystadenoma

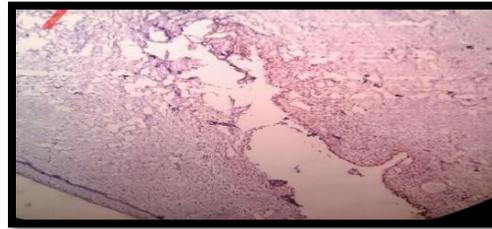


Fig 16: PR Slightly Positive (10X) in Borderline Cystadenoma

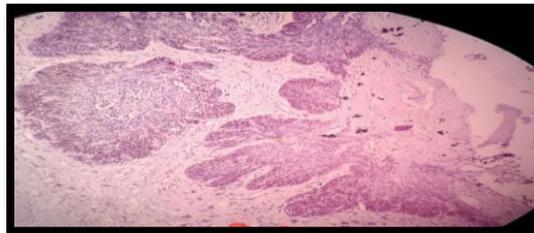


Fig 17:PR Negative in (40X)Serous Cystadeno Carcinoma

P53 expression in seot



Fig 18: P53 Negative (40x) in Benign Cystadenoma



Fig 19: P 53 Focal Positive(40X) in Borderline Cystadenoma

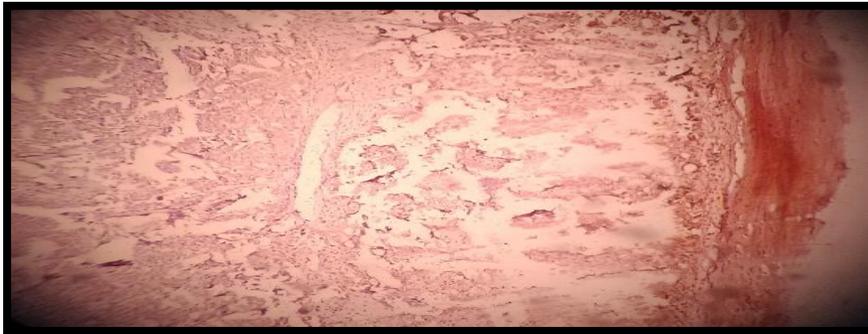


Fig 20: P53 Positive(10X) in Cystadenocarcinoma

Ki 67 expression in seot

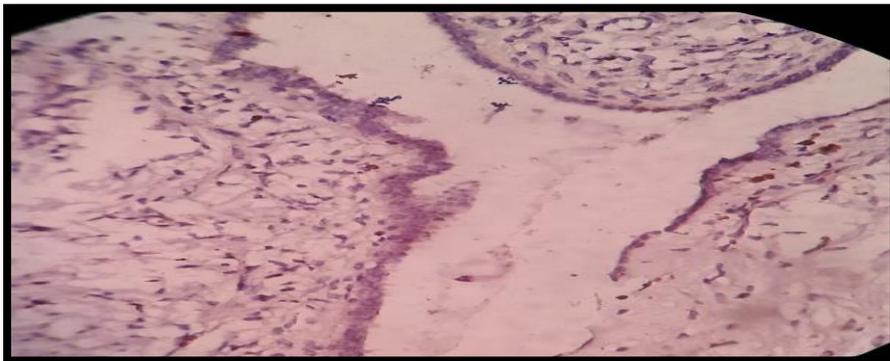


Fig 21: Ki67 Focal Positive (40X) in Benign Serous Cystadenoma

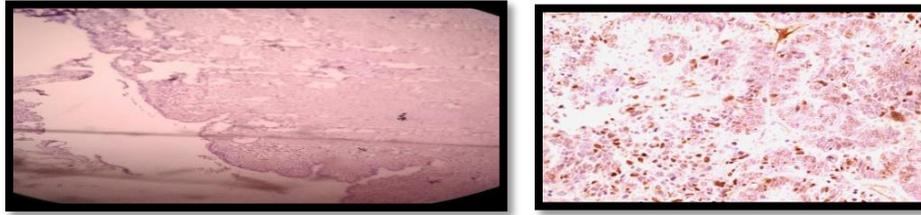


Fig 22:Ki 67 Focal Positive(10X) in Borderline Cystadenomas

Fig 23: Ki 67 Highly Positive(40X) in Borderline Cystadenocarcinoma

Discussion

Ovarian neoplasms are one of the most fascinating tumors in women in terms of their histogenesis, clinical behaviour and malignant potentiality. They account for a disproportionate number of fatal cancers, being responsible for almost half of the deaths from cancers of the female genital tract. This study compared the results with the following studies: Verma & Bhatia et al[12], GG Swamy et al[13], R Jha et al[14]. In the present study, a total of 80 cases of SEOTS were studied in the department of pathology, Osmania Medical college over a period of 2 prospective years.

Age

In present study, peak incidence of SEOTs is seen in the age group of 30 to 40. This is similar to the studies like that done by Verma and Bhatia (1981)[12] and Jha et al (2008)[14] in which the majority of patients were in the 31 to 40 age group.

Benign, Borderline and Malignant

The current study results had shown that 51.5% of patients were benign. This compares well with older studies like the one published by Verma and Bhatia (66.99%) [12]. This selection bias may have inflated the number of malignant cases and reduced the benign ones. 30.5% of tumors in this study are malignant. This was comparable to studies like Verma et al[12] but is much more than Swamy[13] (25%) and Jha et al[14] (16%). This study showed a slightly lower percentage of malignant ovarian tumors (30.5%) but this was similar to the study done by Verma and Bhatia. The study done by Jha et al[14] recorded the least percentage of malignant cases (16.1%).

Comparison of Proportion of benign, borderline and malignant tumors in various studies

The percentage of borderline tumors in this study was 17.5 % which was comparable to the study done by Gupta et al (4.1)[15].

Laterality

Most of the benign tumors in this study were unilateral with only 12% cases showing bilateral tumor involvement. This is comparable to findings of study done by Jha et al[14] (93.34% unilateral). In contrast Swamy et al[13] reported a bilaterality rate of 29% in benign tumors. In malignant tumors that were noted a bilaterality rate of just 7.5% compared to Swamy et al[13] who reported a bilaterality rate of 50% and Jha et al[14] in which 42% of the malignant tumors were bilateral.

Subtypes with Various Studies

In the present series, as in all the other studies, it was found that surface epithelial tumors to be the most common tumors ranging from 56% (Jha et al)[14] to 71% (Pilli et al)[17]. In this series there were 80 ovarian neoplasms. The relative percentage of various histological subtypes of ovarian tumors in this study was comparable to most of the studies that compared the results with. (Jha et al[14], Swamy et al[17], Tyagi et al[18] etc.). Serous cystadenoma comprised 59 cases (29.5%) in this study while Tyagi et al[18] reported 39.5%.

In present study the expression of ER was more in malignant tumors (81%) than borderline (66.6%) and benign (50%). This is parallel to study done Jha et al[14] in malignant(88%),in borderline(60.2%),in benign(40.5%).This may support the role of estrogen in oncogenesis. In our study The expression of PR was more in benign (62.4%) than borderline (50%) and Malignant tumors(54%). This probably indicates the protective effect of progesterone in the Development of ovarian carcinomas. Jha et al[14] demonstrated that the mean ki-67 li in benign tumors was 24.9% in borderline tumors, it was 68.8%; in malignant tumors, it was 85.8%. When compared with the benign tumors, ki-67 li was found to be significantly increased in the malignant tumors. In the present study, similar results were obtained viz., the mean ki-67 li for benign, borderline, and malignant was 26.7%, 81.3%, and 88.8% respectively.

Conclusion

- Ovarian tumors are one of the most researched topics in gynecological Pathology.
- Benign are the most common, of these surface epithelial tumors are the commonest, affects mainly reproductive age group. Clinical features are vague and are late manifestations. Malignant tumors are less common, serous are more prone for going into malignancy.
- ER expression in epithelial ovarian tumors with adverse prognostic factors supports the mitogenic role of estrogen in ovarian tumors.
- Expression of ER was more in malignant tumors than borderline and benign. As compared to ER the expression of PR was more in benign than borderline and malignant tumors.
- p53 was expressed more often in malignant tumors followed by borderline and benign tumors.
- The mean ki-67 labeling index was the highest in malignant followed by borderline and benign tumors.
- Ki-67 index was higher in tumors with adverse prognostic factors. Hence, it would help in prognostication and differentiation of the three morphological type.
- P53 were expressed only in malignant tumors suggesting their carcinogeni role and help in the differentiation of borderline and malignant tumors.
- The findings of this study indicate that IHC marker report of ER, PR status and Ki-67 If included in each pathology report will pave the way for better Understanding of Biological behavior and modify treatment strategies.
- The role of IHC is now employed not only for diagnosis but also for other Parameters including prognosis, staging, and prediction of response to therapy and for the selection of therapeutic agents.

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References

1. Rosai Juan. Rosai and Ackerman's Surgical Pathology, Vol 2, 10th Ed. New Delhi: Elsevier Publication, 2012, 1553-1609.
2. Howkins J, Bourne G. Shaw's textbook of Gynaecology. 13th Ed, Elsevier publication. 2006;2:41-47.
3. Crum CP, Lee KP, GenestDR, Mutter G, Granter SR, Nucci MR et al. Diagnostic Gynaecologic and Obstetric Pathology. Philadelphia. Elsevier Saunders, 2006;680-988.
4. Novak ER, Jones GS, Jones HW. Ovarian tumors. In: Gynaecological and obstetrical pathology. 6th edition. Philadelphia; Saunders, 1967, 365-413p.
5. Jagadeshwari N, Reddy Satyabhama R, Rao KS. Incidence of Ovarian tumours. J ObstGynecol India. 1971; 21:747-52.
6. Azizs, Kuperstein G, Rosem B, Cole D, Nedelew R, Mclaughlin J, Narod SA. A genetic epidemiologic study of carcinoma of the fallopian tube, Gynecoloncol. 2001; 80:341.
7. Narod SA, Boyd J. Current understanding of the epidemiology, and clinical implication of BRCA 1 and BRCA 2 for ovarian cancer. Curr. Opin obstetric gynecol. 2002; 14:19.
8. Nogales FF, Ruiz Avila I, Concha A, Del Morel. Immature endodermal teratomas of the ovary. Embryologic correlation and immunohistochemistry. Human Pathol. 1993; 24: 364-370.
9. Ness RB, Grissoja, Vergona R, Klapper J et al. Study of health and Reproduction (SHARE) study group 1: oral contraceptives, other methods of contraception, and risk of ovarian cancer, Epidemiology. 2001; 12:307.
10. R.C. Bast Jr., S. Knauf, A. Epenetos, B. Dhokia, L. Daly, M. Tanner, J. Soper, W. Creasman, S. Gall, R.C. Knapp et al. —Coordinate Elevation of Serum Markers in Ovarian Cancer But Not in Benign Disease, Cancer. 1991; 15(68):1758-1763.
11. Jacobs I, Oram D. Screening for ovarian cancer. Biomed. Pharmacother. 1988; 42:589-596.
12. Verma, Bhatia. Ovarian Neoplasms - A study of 403 tumors. J Obstet. Gynecol. India. 1981; 31:106-111.
13. GG Swamy, N Satyanarayana. Clinicopathological analysis of ovarian tumors – A study on five years Samples. Nepal Med Coll J. 2010; 12(4):221-223.
14. R Jha, S Karki. Histological pattern of ovarian tumors and their age distribution. Nepal Med Coll J. 2008; 10(2):81-85.
15. Gupta SC, Singh PA, Mehrotra TN et al. A Clinicopathological study of ovarian tumours. Ind. J PatholMicrobiol. 1986;29:354-362.
16. Pilli G, Suneeta KP, Dhaded AV, Yenni VV. Ovarian tumors – Study of 282 cases. JIMA. 2002; 07:100-2.
17. GG Swamy, N Satyanarayana. Clinicopathological analysis of ovarian tumors – A study on five years Samples. Nepal Med Coll J. 2010; 12(4):221-223.
18. Tyagi SP, Tyagi GK, Logani K. A Pathological study of 120 ovarian tumors. J. Obst & Gynecol Ind, 1967; 423-433.

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