International Journal of Clinical Trials
Gautam KB et al. Int J Clin Trials. 2024 Nov;11(4):275-282
http://www.ijclinicaltrials.com
pISSN 2349-3240 | eISSN 2349-3259
DOI: https://dx.doi.org/10.18203/2349-3259.ijct20242673

Original Research Article

Histopathological spectrum of ovarian tumours

K. B. Gautam[1], Ranu Tiwari Mishra[2]*, Sanjay Totade[2]

[1]District Hospital, Rewa, M. P., India
[2]Department of Pathology, Netaji Subhash Chandra Bose Medical College, Jabalpur, M.P., India

Received: 27 August 2024
Revised: 10 September 2024
Accepted: 11 September 2024

***Correspondence:**
Dr. Ranu Tiwari Mishra,
E-mail: ranu.m7317@gmail.com

ABSTRACT

Background: Ovarian neoplasms include wide spectrum of various tumors, which differ in their histo-morphological features, also in their biological behaviour, tumorigenesis, clinical course, and prognosis. Aim of the study was to study the histopathological features of various ovarian tumors and correlate preoperative serum CA125 tumor marker level with histopathological types. Also to study fallopian tubes for fallopian tube precursor lesions such as serous tubal intraepithelial carcinoma (STIC) in serous carcinoma ovary.
Methods: This cross-sectional observational study of the 80 specimens of ovarian tumors was conducted in department of pathology, Netaji Subhash Chandra Bose medical college Jabalpur (M.P.). The relevant data of the patients was collected and analysed as per designed proforma.
Results: Out of 80 cases; majority cases 22 (27.5%) were seen in a 31-40 years age group, followed by 41-50 years age group 18 cases (22.5%). Youngest patient was 7 years old and oldest patient was 88 years old, forming the range of 7 years to 88 years. Epithelial tumors were the most common category, followed by germ cell tumors, sex cord stromal tumors and metastatic tumors. Benign tumors were much higher than borderline and malignant tumors mature cystic teratoma was the most common tumor encountered, followed by mucinous cystadenoma. The commonest malignant tumor was serous carcinoma followed by mucinous carcinoma.
Conclusions: Ovarian neoplasms constitute a wide spectrum of tumors therefore exact categorization is important to adopt appropriate treatment protocols for the proper management of the patient. Histopathological examination is the gold standard for diagnosis and categorization of ovarian neoplasm.

Keywords: Ovarian, Neoplasms, Histopathology

INTRODUCTION

Ovarian neoplasms include a wide spectrum of various tumors, which differ not only in histomorphological features, but also in their biological behaviour, tumorigenesis, molecular biology, clinical course, and prognosis.[1]

Ovaries not only harbor primary tumors of ovarian tissue but are a site for metastatic tumors. Primary tumors may be derived from one of the three ovarian components-the surface or fallopian tube epithelium and endometriosis, pluripotent germ cells, sex cord-stromal cells of ovarian stroma.[2] According to Globocan's 2022, incidence of ovarian malignancy ranked 18 with 324603 cases and ASR (World) 6.7.[3] Ovarian cancer ranked third in women, trailing behind carcinoma breast and carcinoma cervix uteri in India.[3] There is a steady increase in the occurrence of ovarian cancer, and they contribute to about 6.2% of total cancer cases in India.[4]

Primary tumors are more common than secondary tumors and commonest is epithelial tumors which are classified on the basis of their differentiation and extent of

proliferation of neoplastic epithelium. The three major types are-serous, mucinous and endometrioid tumors, which are further classified into benign, borderline and malignant, depending upon the epithelial proliferation and atypia. Serous carcinoma is the most common epithelial malignancy. Serous ovarian carcinomas are of two types: low grade carcinoma and high grade carcinoma. They not only differ in their histological features, but also have distinct mutational profile. Low grade serous carcinomas may develop in association with serous borderline tumors whereas high grade serous carcinomas may arise either from inclusion cyst within the ovary or from in situ lesion in fallopian tube called as STIC.[2] Epithelial malignancies are diagnosed late, by this time, the tumors have generally spread beyond the ovary, and become difficult to treat but women with non-epithelial tumors may show distinctive clinical features, as they are hormonally active.[1,5]

Most commonly used biomarker is serum CA-125. CA-125 is a high molecular weight transmembrane glycoprotein. It is expressed by coelomic and Mullerian derived epithelium including fallopian tube, endometrium and endocervix. Normal value of serum CA-125 levels is less than 35 U/ml. It is raised in approximately 50% of stage l ovarian cancers and 90% of advanced stage patients. Serous tumors are more strongly associated with raised levels of CA-125 and they constituted the most lethal subtype of ovarian cancers.[6]

Observation in patients with BRCA-1 and BRCA-2 germline mutation showed that almost all ovarian carcinomas arising in these patients are high grade serous carcinoma and, are associated with TP53 mutation, secondly they arise from precursor lesions, in the distal end of fallopian tube, (fimbriae) known as STIC. Therefore, in order to carry out efficient risk reductions surgery, bilateral salpingo-oophorectomy should be done, in place of oophorectomy, as a prophylactic measure.[2]

METHODS

This cross-sectional observational study was conducted in department of pathology, Netaji Subhash Chandra Bose medical college Jabalpur (M.P.). This study comprised of the 80 surgical specimens of ovarian tumors submitted for histopathological examination in our department during the study period of 18 months. The relevant data of the patients was collected and analysed as per designed proforma. Ovaries with non-specific findings, non-neoplastic lesions like simple ovarian cyst, stromal hyperplasia etc. and autolyzed specimens were excluded from the study. Specimens were fixed in 10% formalin for 24 hrs. The gross and microscopic descriptions of the ovarian tumors were done as per CAP protocol.[7] Haematoxylin and eosin-stained sections were prepared and examined. Ovarian tumors were categorized according to WHO classification 2014.[8] Fallopian tubes were examined for the specific microscopic features of STIC as per criteria. Blocks of fallopian tubes having morphological features which suggest STIC like lesions were submitted outside laboratory for immune-histochemistry study for p53 expression.

Permissions from institutional ethical committee was taken.

Grossing of fallopian tube

Fallopian tube grossing is done with SEE-FIM (Sectioning and extensively Examining the Fimbriated End) as per CAP guidelines on protocol for examination of specimens from primary tumors of ovary, fallopian tube or peritoneum.[7] According to this, amputation and longitudinal sectioning of the infundibulum and fimbrial segment (distal 2 cm) was done to allow maximal exposure of the tubal plicae. The isthmus and ampulla were cut transversely at 2 to 3 mm intervals. CA125 values were obtained from patient clinical data.

Statistical analysis used

Collected data was analysed by using IBM-SPSS version 23.0. The statistical methods were used to make cross-tabulation, frequencies, percentage, bar diagram and Fisher's exact test was used for comparison of clinical diagnosis and histopathological diagnosis. $P<0.05$ was taken to indicate a statistically significant difference.

RESULTS

Out of 80 cases; majority of tumors 22 cases;(27.5%) were seen in the 31-40 years age group, followed by 41-50 years age group 18 cases, (22.5%). Youngest patient was 7 years old and oldest patient was 88 years old, forming the range of 7 years to 88 years. Maximum cases 46 (60%) were premenopausal women and 31 cases (38.75%) had attained their menopause. There was single prepubertal girl in present study.

Maximum numbers of cases 78; (97.5%) were of primary tumors and 2 cases (2.5%) were metastatic, secondary tumors. Out of 78 primary tumors, 52 cases of epithelial tumors 52 (66.6%), 22 cases of germ cell tumors, (28.2%) and 4 cases of sex-cord stromal tumors n=4; (5.1%) were seen and 53 cases (66.2%) were benign in nature. The borderline tumors were n=4(5%).

Malignant tumors contributed n=23(28.75%) cases. Among 52 cases of epithelial ovarian tumors, 32 cases were diagnosed as benign (32/52=60.37%), 4 cases of borderline and 16 cases of malignant epithelial tumors were seen (30.76%). Serous tumors comprised of 30 (57.69%) cases, mucinous tumors comprised of 19 cases (36.53%) and one case each of benign endometrioid, benign Brenner, malignant mixed epithelial and mesenchymal (carcinosarcoma) tumor was seen.

Table 1: Age wise distribution of the cases.

Age group (in years)	Epithelial tumors	Germ cell tumors	Sex cord stromal tumors	Metastatic tumors	Total no. of tumors
0-10	-	1	-	-	1
11-20	4	3	1	-	8
21-30	5	9	1	-	15
31-40	15	4	1	2	22
41-50	14	4	0	-	18
51-60	9	0	2	-	11
61-70	3	1	0	-	4
71-80	1	0	0	-	1
81-90	1	0	-	-	1
Total	52	22	4	2	80

Distribution of ovarian tumors

Among 32 cases of benign epithelial tumors, serous and mucinous tumors contributed 15 cases (46.9%) each. All borderline tumors were of serous type. Out of 16 malignant epithelial tumors 11 cases; (68.8%) were of serous carcinoma, 4 cases (25%) were of mucinous carcinoma and one case of mixed epithelial and mesenchymal tumor was also seen.

Germ cell tumors constitute 22 cases; (27.5%). Mature cystic teratoma 17 cases; (77.27%) was the most common finding. Immature teratoma and dysgerminoma were next in frequency with having 2 cases each and a single case of monodermal teratoma was found in the form of struma ovarii. In present study sex cord stromal tumor were 4 cases (5%) In this, one case was pure sex cord (adult granulosa cell tumor) and three cases were pure stromal tumor (fibroma).

In present study mature cystic teratoma was the commonest benign tumor with 17 cases reported (32.07%) among all benign tumors. Next most common benign tumor was mucinous cystadenoma accounting for 15 cases (28.30%) followed by serous cystadenoma 14 cases (26.41%). Three cases of fibroma and one case each of struma ovarii, serous adenofibroma, Brenner tumor and endometriotic cyst were also reported.

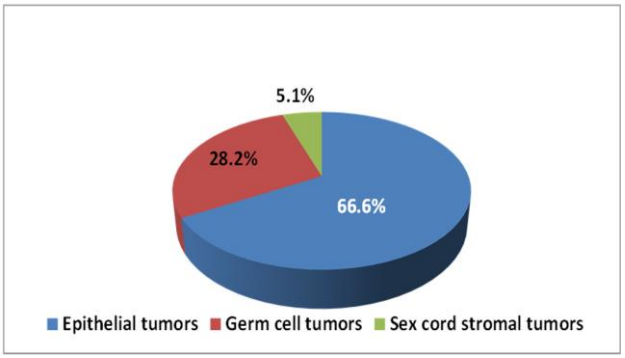

Figure 1: Distribution of primary ovarian tumour according to nature of tumpur.

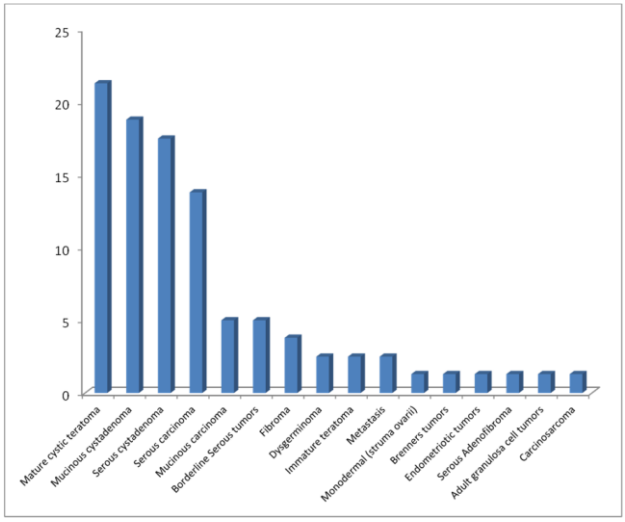

Figure 2: Frequency distribution of total ovarian tumour, (n=80).

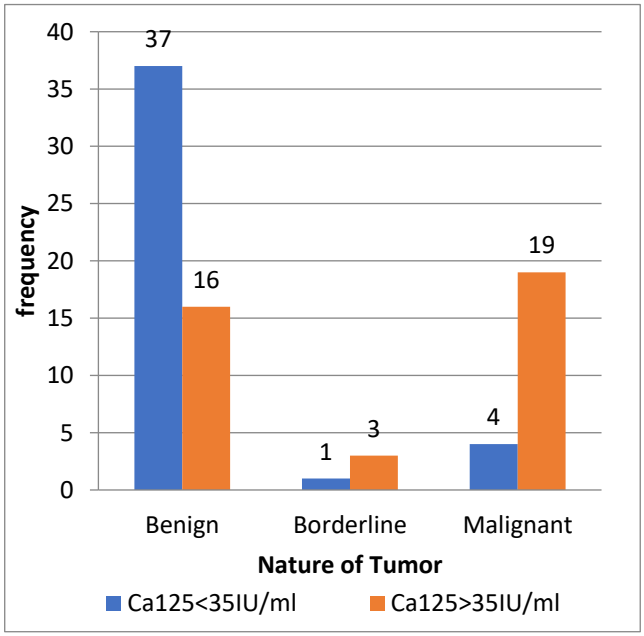

Figure 3: CA 125 association with nature of tumors.

In the malignant category, serous carcinoma was the predominant tumor 11 cases (13.8%), followed by mucinous carcinoma 4 cases (5%), thus making epithelial cancers, the most common cancers of the ovary. Adult granulosa cell tumor and carcinosarcoma were having least prevalence of all 1 case; (4.3%) each.

The correlation between preoperative serum CA125 levels and histopathological diagnosis was evaluated. Chi-square analysis was done and $p<0.0011$ was considered to be statistically significant. There was a significant ($p<0.05$) association between CA-125 levels and serous epithelial tumors. The values are much higher in serous ovarian cancers, as compared to mucinous carcinoma, or other tumors.

Distribution and correlation of p53 staining and STIC lesion in high-grade serous carcinoma and non-high grade ovarian tumors

Total 5 fallopian tubes from high grade serous ovarian carcinoma were submitted. STIC lesions were seen in 2 out of 5 cases (40%). While no STIC lesion was found in non-high grade ovarian tumors in a control group (n=0/4, 0% cases).

In these two STIC lesions, abnormal, mutant, diffuse, strong nuclear p53 expression was seen (n=2/2, 100%). Whereas the remaining (n=3/5, 60%) cases of high grade serous ovarian carcinoma showed only intermittent weak patchy wild type

expression which was considered normal. None of the high-grade serous carcinoma cases showed complete negative staining expression in present study. On the other hand, non-high-grade ovarian tumors in control group did not reveal diffuse strong nuclear p53 expression (0/4).

Table 2: Distribution and correlation of p53 staining and STIC lesion in high-grade serous carcinoma and non-high grade ovarian tumors.

STIC lesion and p53 staining pattern	High-grade serous carcinoma, (n=5)	Non-high grade ovarian tumors, (n=4)
STIC lesion in fallopian tube	2/5 (40%)	0/4 (0%)
Diffuse strong nuclear p53 expression	2/2 (100%)	0/4(0%)
Complete negative p53 expression	0	0
Wild type p53 expression (Focal and patchy)	3/5 (60%)	4/4 (100%)
P53 signature	0	0

All 4/4 cases of non-high grade ovarian tumors (one case each of serous cystadenoma, serious borderline tumors, low grade serous carcinoma and mucinous carcinoma) revealed only intermittent weak patchy wild type expression of p53.

This distribution of diffuse strong nuclear p53 staining (mutant expression) in case of high-grade serous carcinoma with STIC was significant when compared with non-high grade ovarian tumor group.

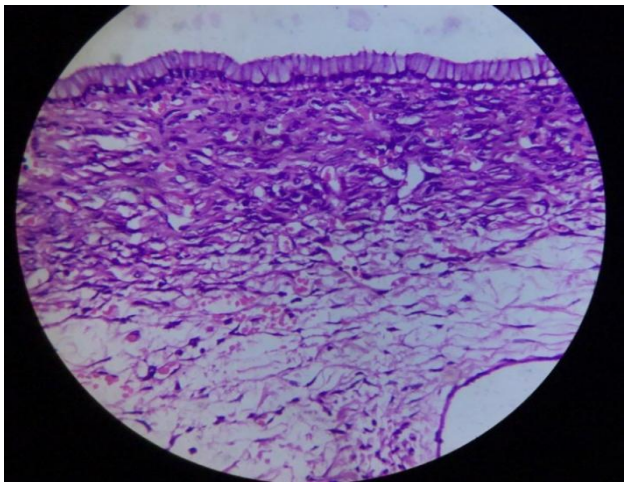

Figure 4: Mucinous cystadenoma: cyst wall lined by tall columnar epithelium with uniform round or oval basal nuclei and abundant clear amphophilic cytoplasm photomicrograph (400×).

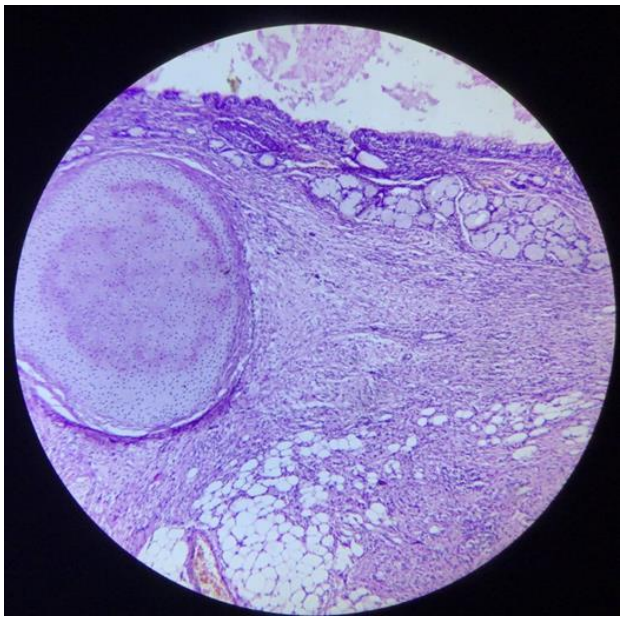

Figure 5: Mature cystic teratoma: photomicrograph (400×)-tumor showed mature tissue like adipose tissue, cartilage, mucous glands, respiratory mucosa, in varying proportion.

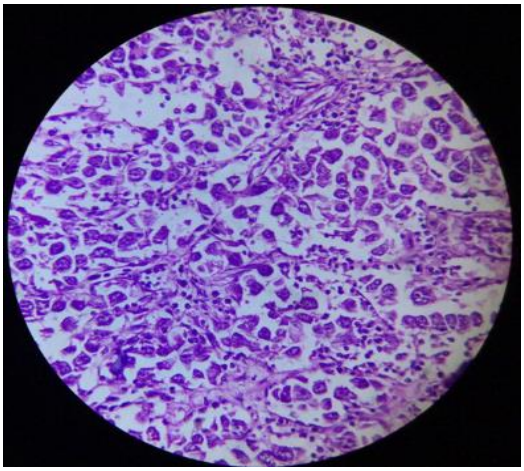

Figure 6: Dysgerminoma: polygonal cells with abundant clear to pale staining cytoplasm separated by fibrous stroma with lymphocytic infiltration (400×).

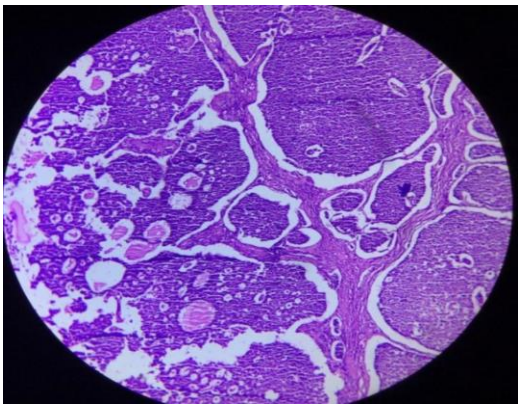

Figure 7: Granulosa cell tumor: microphotograph (100×) nests and sheets of granulosa cells with microfollicular and trabecular pattern. At places, call-Exner bodies were seen.

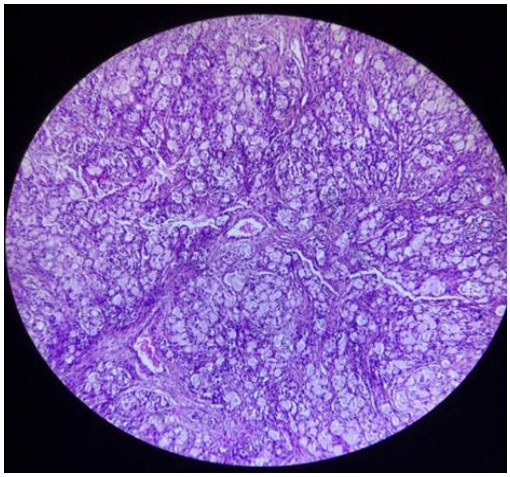

Figure 8: Metastatic tumor: Krukenberg tumor-microphotograph (100×) nests, cords and single file pattern of signet ring cells infiltrating the ovarian stroma.

DISCUSSION

Age distribution of cases

This study of 80 ovarian tumors included age groups from 1st decade to 8th decade patients. In this study we found maximum numbers of cases in age group of 31-40 years (n=22; 27.5%) followed by 41-50 years (n=18; 22.5%). Garg et al also reported occurrence of majority of ovarian tumors in 31-40 years (n= 35; 41.2%), followed by 41-50 years of age group (n =19;22.3%).[9] Patel et al in their study of 162 cases also found maximum number of total cases in 31-40 years of age group (n=55;34%).[10]

Distribution of cases according to origin of tumors

In the present study of 80 ovarian tumors majority of these cases, 78 (97.5%) were primary ovarian tumors whereas 2 cases (2.5%) were diagnosed as secondary/metastatic. Our results are in accordance with studies conducted by Chandanwale et al, Garg et al and Kaur et al.[9,11,12] Chandanwale et al done a study of 50 ovarian tumors and found majority of tumors (n=47;94%) were primary and remaining were (n=3;6%) were metastatic.[11] Similarly, the study of 85 ovarian tumors conducted by Garg et al also found primary tumors (n=83;97.6%) as a most common tumors of all ovarian tumors and only 2 cases as secondary tumors were detected.[9]

Distribution of ovarian tumors according to histopathological types

Among the primary ovarian tumor, the commonest category encountered in present study was epithelial tumors (n=52; 65%). Germ cell tumors were second largest group and comprised of (n=22; 27.5%) cases followed by sex-cord stromal tumors (n=4; 5%) cases and (n=2;2.5%) cases of metastasis were identified. The findings are in concordance with the studies done by Garg et al found 70.6%, 18.8%, 8.2% and 2.4% cases and Sudha et al also observed 64.13%, 26%, 8.6% and 01% cases of epithelial tumors, germ cell tumors, sex-cord stromal tumors and metastasis respectively.[9,13]

Frequency distribution of various histological types of ovarian tumors of benign biological behaviour

Mature cystic teratoma was the commonest benign tumor with 17 cases reported (32.07%) of all benign tumors. Next most common benign tumor was mucinous cystadenoma accounting for 15 cases (28.30%) followed by serous cystadenoma 14 cases (26.41%). Three cases of fibroma and one case each of struma ovarii, serous adenofibroma, Brenner tumor and endometriotic cyst were also reported. Study done by Ahmed et al and Datta et al also showed benign cystic teratoma as the commonest benign tumor accounting for 35.17% and 70% of all benign tumors respectively.[14,15] Next most common benign tumor was serous cystadenoma followed by mucinous cystadenoma in their study. In present study

among benign epithelial tumor mucinous cystadenoma was slightly more common than serous cystadenoma. Similar findings were also reported by Mankar et al in their retrospective study of 257 cases of ovarian tumors they found that mucinous cystadenoma (32.6 9%) was the most common benign epithelial tumor, however in majority of studies like Sudha et al, Patel et al and Sofi et al serous cystadenoma was the most common benign tumor.[10,13,16]

Frequency distribution of various histological types of ovarian tumors of malignant biological behaviour

Out of 80 cases, 23 cases were diagnosed as malignant tumors. The commonest malignancy was serous carcinoma constituted 11 cases (47.82%) and next commonest tumor was mucinous carcinoma comprising four cases (17.3 9%). This was followed by Dysgerminoma, immature teratoma and metastatic tumors comprising two cases each. One case each of adult granulosa cell tumor and carcinosarcoma were also reported. In serous carcinoma 6 cases were high grade serous carcinoma and 5 cases were reported as low-grade serous carcinoma. Serous carcinoma was the most common malignant tumor in the present study, findings were consistent with previous studies done by Sudha et al, Gupta et al and Chandanwale et al in their study of 50 cases of malignant ovarian tumors also found serous carcinoma as the most common histological type (n=15) followed by mucinous carcinoma(n=7).[11,13,17]

CA 125 level association with ovarian tumor-

CA-125 is an important tumor marker in pre-operative evaluation, and in suggesting the type and malignant nature of ovarian neoplasms. The values are much higher in serous ovarian cancers, as compared to mucinous carcinoma, or other tumors. All 11 (100%) malignant serous tumors showed increased level of CA125. Findings suggest a significant association ($p<0.05$) between raised CA125 levels and serous carcinomas. Similar results were also found in study conducted by Sudha et al.[13] They found much more increased level of CA125 in malignant serous tumors. Mucinous tumors showed moderate increase in CA125 levels. A study done by Nayak et al also concluded that mean serum CA125 concentration in papillary serous adenocarcinoma patients (n=45) was 1456±320 U/ml.[18] They also found highest level of CA125 in serous carcinoma. In mucinous adenocarcinoma (n=24) levels were 756 ±125 U/ml.

Distribution and correlation of p53 staining and STIC lesion in high-grade serous carcinoma and non-high grade ovarian tumors

There are cumulative evidences that suggest high grade serous ovarian carcinoma arises from this clonal expansion of secretory cells of the distal end of the fallopian tube rather than from ovary. We also tried to find out the association of high grade serous carcinoma with intraepithelial carcinoma of the fallopian tube. Out of 80 specimens of ovarian tumors, 52 specimens were submitted along with fallopian tube; among which 43 were unilateral salpingo-oophorectomy specimens and 9 cases were bilateral salpingo-oophorectomy specimens. All submitted fallopian tubes were serially sectioned and extensively examined accordingly to SEEFIM protocol. Sections were first examined to look for any tubal pathology, metastasis from adjacent ovarian tumors and STIC on H and E sections. STIC are the non-invasive lesions but showed cytologically malignant tubal epithelial cells characterized by abnormal growth of principally stratified non ciliated cells. Marked nuclear pleomorphism with prominent nucleoli. Increased nuclear cytoplasmic ratio. Loss of cell polarity. Lack of cellular cohesion with shedding of cells into tubal lumen. Lesions were non- invasive in nature. and considered a precursor lesion for high grade ovarian and peritoneal serous carcinoma20. Tubal intraepithelial lesion was first assessed by morphology and blocks of fallopian tubes with suspicious lesion were sent for p53 expression by immunohistochemistry to external laboratory along with paraffin blocks of non- serous tumors as controls. p53 expression study was done by immunohistochemistry using BP 53-12 clone and results were interpreted.

In 10 submitted fallopian tubes 2 types of p53 expression staining patterns were observed: P53 expression was considered wild-type (normal) pattern if there were weakly staining cells/ focal patchy pattern of expression, consistent with normal p53 expression. Normal fallopian tube epithelium also exhibits wild-type pattern of staining. Abnormal p53 mutant pattern of staining was the one which showed diffuse strong nuclear staining.

Diffuse strong nuclear p53 staining (mutant pattern) was reported in 2/5 (40%) high grade serous ovarian carcinoma cases, in serous tubal intraepithelial lesions, and similar staining was also observed in invasive high grade serous carcinoma component in the ipsilateral fallopian tube. None of the fallopian tube from benign, borderline or low-grade serous carcinoma and mucinous carcinoma cases showed this type of abnormal expression, or STIC lesions. Association of diffuse strong nuclear p53 staining in high grade serous carcinomas was significant when compared with benign, borderline or low grade serous tumors and mucinous carcinomas.

Wild type normal staining pattern was seen in 3/ 5 cases of high grade serous carcinoma and all non-high grade serous carcinoma group.

These results of the present study are comparable with the following studies: Choudhary et al observed STIC lesion in 10 out of 27 (37.0 4%) cases of high grade serous ovarian carcinoma while no STIC lesion was identified in non-high grade serous tumor. They also observed a significant association between occurrence of STIC lesion in high grade serous ovarian carcinoma with the $p=0.013$.[20]

Study done by Morrison et al on fallopian tubes removed for non-prophylactic indications (in non-BRCA-1, BRCA-2 carrier patients), identified STIC lesions, in some patients with associated microscopic foci of high grade serous carcinoma in the ipsilateral ovary.[21] The study highlighted the importance of complete examination of fallopian tubes and ovaries to identify STIC and early invasive serous carcinoma. Similarly, Kindelberger et al found that over 70% of sporadic (non-hereditary) ovarian and peritoneal high grade serous carcinomas showed fallopian tube mucosal involvement by STICs, suggesting that STICs may be the precursors for sporadic as well as hereditary high grade serous carcinomas.[22]

Limitations

Observations from the present study suggests that STIC lesions coexist with a significant number of high grade serous carcinoma cases but not associated with rest other ovarian tumors. However further studies need to be done to know the role of STIC in the etiopathogenesis of high grade serous carcinomas, as we did not have any prophylactic bilateral salpingo-oophorectomy specimens and patients' BRCA1 and BRCA2 mutational status was not known.

CONCLUSION

Ovarian neoplasms constitute a wide spectrum of various tumors that differ histopathologically, as well as clinically. Therefore, a detailed and thorough study, and exact categorization of various types of ovarian tumors is important to adopt appropriate treatment protocols for the proper management of the patient. Histopathological examination is the gold standard for diagnosis and categorization of ovarian neoplasms.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Torre LA, Trabert B, DeSantis CE, Miller KKD, Samimi G, Runowicz CD, et al. Ovarian Cancer Statistics 2018. CA cancer J clin. 2018;68(4):284-96.
2. Kumar V, Abbas AK, Aster JC, Sign M. Robbins and Cotran pathologic basis of disease, vol. II. The Female Genital Tract, Ovaries. Tenth edition. 2020;1016-9.
3. Ferlay J, Ervik M, Lam F, Laversanne M, Colombet M, Mery L, et al. Global Cancer Observatory: Cancer Today. Lyon, France. Int Agency Res Cancer. 2024. Available at: https://gco.iarc.who.int/today. Accessed on 15 June 2024.
4. Takiar R. Status of Ovarian Cancer in India (2012-14). EC Gynaecol; 2019;8.5:358-64.
5. Karst AM, Drapkin R. Ovarian Cancer Pathogenesis: A Model in Evolution. J Oncol. 2010;932371:13.
6. Gupta D, Lis CG. Role of CA125 in predicting ovarian cancer survival-a review of the epidemiological literature. J Ovarian Res. 2009;2:13.
7. College of American Pathologists (CAP). Protocol for examination of specimens from patients with primary tumors of ovary, fallopian tube, or peritonium. 2018.
8. WHO Classification of female reproductive organs. International Agency for research on cancer, 4th Edition. 2014. Available at: https://publications.iarc.fr/Book-And-Report-Series/Who-Classification-Of-Tumours/WHO-Classification-Of-Tumours-Of-Female-Reproductive-Organs-2014. Accessed on 3 June 2024.
9. Garg N, Anand AS, Annigeri C. Study of histopathological spectrum of ovarian tumors, Raichur, Karnataka. Int J Med Heal Res. 2017;3(10):12-20.
10. Patel AS, Patel JM, Kamlesh SJ. Ovarian tumors-Incidence and histopathological spectrum in tertiary care center, Valsad. IAIM. 2018;5(2):84-93.
11. Chandanwale SS, Jadhav R, Rao R, Naragude P, Bhamnikar S, Ansari JN. Clinicopathologic study of malignant ovarian tumors: A study of fifty cases. Med J DY Patil Univ. 2017;10:430-7.
12. Sarabjeet K, Jassal V, Bodal VK. A study of histological pattern of ovarian carcinoma in tertiary care Hospital Punjab. Int J Curr Res Biol. 2018;3(3):63-70.
13. Sudha V, Harikrishan V, Sridevi M, Priya P. Clinicopathological correlation of ovarian tumors in a tertiary care hospital. Indian J Pathol Oncol. 2018;5(2):332-3.
14. Sofi MA, Bashir N, Afshan KA, Ali K. Histopathological Pattern of Ovarian tumors-An Experience. Int J Clin Diagnost Pathol. 2019;2(2):15-21.
15. Dutta A, Reshma I, Saikia P, Borgohain4 M. Histopathological spectrum of ovarian neoplasms in a tertiary care hospital. Int J Contemporary Med Res. 2018;5(8):H1-4.
16. Mankar DV, Jain GK. Histopathological profile of ovarian tumours: A twelve year institutional experience. Muller J Med Sci Res. 2015;6:107-11.
17. Gupta N, Yadav M, Gupta V, Chaudhary D, Patne SCU. Distribution of various histopathological types of ovarian tumors: A study of 212 cases from a tertiary care center of Eastern Uttar Pradesh. J Lab Physicians. 2019;11(1):75-81.
18. Ashwani N, Chaitra S, Padma K, Swarup A, Cherukumudi A, Chandran PR. Correlation between CA 125 and Staging and Histopathological Type of Ovarian Cancer. J Med Sci Clin Res. 2015;3(11):NA.
19. Kurman RJ, Ellenson LH, Ronnett BM. Blaustein's pathology of the female genital tract Disease of fallopian tube and paratubal region, (sixth ed.); New

York: Springer. 2011;560.
20. Chaudhary VK, Solanki R. Association of high-grade serous ovarian carcinoma with intraepithelial carcinoma of fallopian tube. Int J Med Sci Public Health. 2016;5:1956-9.
21. Morrison JC, Blanco LZ Jr, Vang R, Ronnett BM. Incidental serous tubal intraepithelial carcinoma and early invasive serous carcinoma in nonprophylactic setting: analysis of a case series. Am J Surg Pathol. 2015;39(4):442-53.
22. Kindleberger DW, Lee Y, Miron A, Hirsch MS, Feltmate C, Medeiros F, et al. Intraepithelial carcinoma of the fimbria and pelvic serous carcinoma: evidence for a causal relationship. Am J Surg Pathol. 2007;31(2):161-9.

Cite this article as: Gautam KB, Mishra RT, Totade S. Histopathological spectrum of ovarian tumours. Int J Clin Trials 2024;11(4):275-82.