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Histopathological Pattern and Age Distribution, of Malignant Ovarian Tumor among Sudanese Ladies

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Abstract

INTRODUCTION: Ovarian cancer is the cause of a high case-fatality ratio, and most of the cases are diagnosed in late stages.

OBJECTIVES: To determine the histopathological types, age distribution, and ovarian tumour stages among diagnosed with ovarian cancer at Al - Amal Tower a multi-referral polyclinic of Radiology & Isotope Center Khartoum (RICK), Sudan.

METHODS: All histopathology reports patients' case from January to June 2015 were reviewed. The cancers classified according to federation international of Obstetrics and Gynecology (FIGO).

RESULTS: There were 127 cases of ovarian cancers. Surface epithelial cancers were the most common 77.7% (n = 98), followed by sex cord-stromal cancers 11.23% (n = 14), Germ cell tumor 1.6% (n = 2). Metastatic cancers were seen from colon and breast in 6.3% and 3.9 % of cases respectively. Few cases (14%) of ovarian cancers were reported before 40 years of age, after the age of 50 is a sharp increase in the incidence of a tumour. The mean age at presentation was 52.36 ± 14.210 years, there is mean age of menarche 13.59 ± 2.706 years. Very few patients used HRT (1.6%) or had been on ovulation induction treatment (8.7%). Most of patients 39 (30.7%) presented in stage IIIC, and stage 1V 32 (25.2%) indicating a poor prognosis.

CONCLUSION: The incidence of different types of ovarian cancers in the present study is similar to worldwide incidence. The surface epithelial tumour is the commonest ovarian cancer, of which serous adenocarcinoma is the commonest and most of our patients present in late stages.

Introduction

Ovarian cancer is the second most common cause of death from gynecologic tumours in the United States [1][2]. Initially, symptoms may be vague or not apparent, but they become more noticeable as the disease progresses. Early symptoms of ovarian cancer include bloating, pelvic nausea pain, and abdominal swelling [3]. Peritoneal cavity, lymph nodes, lungs, and liver are the most common site for metastasis [4]. The risk of ovarian cancer increases with ovulation induction treatment, nulliparity, women on hormonal replacement therapy, and those begin ovulation at a younger age or reach menopause at an older age at a higher risk of ovarian cancer [5][6]. Factors that decrease the risk of ovarian cancer include the use of OCP, tubal ligation, and breastfeeding [6]. Genetic inheritances are responsible for 10% of cases; the estimated risk for women with BRCA1 or BRCA2 is 50% [6]. The most common type of ovarian malignancies is epithelial cell carcinoma which accounts for 95% of cases. There are five main subtypes of epithelial cell carcinoma, of which high - grade serous is most common. These tumours originate from inclusion cysts in the cells overlying the ovaries [5] though some may form at the Fallopian tubes [7] [8]. Infrequent types of ovarian

cancer include germ cell tumours and sex cord stromal tumours [5]. The diagnosis of ovarian cancer is confirmed by histopathology examination.

This study aimed to determine the histopathological pattern of ovarian cancer stages, and the age distribution in the patients diagnosed at Al - Amal Gynecologic Oncology Hospital, Khartoum, Sudan.

Material and Methods

This is a prospective cross-sectional hospital-based study conducted at Al-Amal Tower a multi-referral polyclinic of Radiology & Isotope Center Khartoum (RICK) which is the most leading Oncology Center in Sudan since founded in 1967. Where over 6,800 new cancer cases were diagnosed & managed in 2014. The study was carried out from January to June 2015. The study population composed of patients diagnosed as having ovarian cancer. Eligibility is limited to the Alamal Oncology Tower.

Data collection tools

Demographic data were gathered including age; residence, menarche and family history of breast, ovarian and colonic cancer. History of ovulation induction treatment and uses hormonal replacement therapy were recorded. Histopathology report of the examined specimen was obtained from the histology laboratory.

Ethical consideration

The study was approved by the Ethics Review Committee of the Sudan Medical Specialization Board, Council of Obstetrics and Gynaecology and AL-amal Ethics Committee. Formal consent was taken from each participant written consent was taken from each participant.

Data collection

To ensure completion of questionnaire data was collected by a senior registrar in Obstetrics and Gynecology.

Statistical analysis

The statistical package for the social sciences (SPSS version 20 for Windows) was used for data analyses. The descriptive statistical analyses used included the mean, standard deviation, and frequency distribution.

Results

The mean age of the study group was 52.36 ± 14.210 years (ranged from14 to 95 years). Their mean age at menarche was 13.59 ± 2.706 years and a mean parity of 4.41 ± 3.396 deliveries. The mean menopausal age was 33.85 ± 21.991 years, and the parity was 4.41 ± 3.99 deliveries. The majority of patients were from the central states of Sudan. Few of them had been using combined oral contraceptives, ovulation induction treatment and hormonal replacement therapy, 7.1%, 8.7%, and 1.6% respectively.

Table 1: Basic characteristics of study population

Mean maternal age	52.36 ± 14.210 years
Age of menarche	13.59 ± 2.706 years
mean parity	2.2756 ±.76301 years
Oral contraceptive pills users	9 (7.1%)
Ovulation induction medications	11 (8.7)
HRT use	2 (1.6)
Nulliparous	22 (17.3)
Multiparous	47 (37)
Grand multiparous	58(45.7)

Data present as number (%).

The family history of ovarian, breast or colonic cancer was positive in 11.8% (n = 15) of cases. The mean age for endometrioid tumours was 64 ± 4 years, while that for mucinous, serous transitional and adenosarcoma was similar. The mean age of occurrence of Sertoli cell tumours and Clear cell tumours was at late reproductive life as shown in Table 1 and 2.

Table 2: The mean age of occurrence of different types of ovarian cancer

Cancer	Age mean ± SD years
Serous adenocarcinoma	54.2 ± 14
Mucinous adenocarcinoma	50 ± 16
Endometrioid tumours	64 ± 4
Clear cell tumours	39 ± 3
Transitional cell tumours	52.4 ± 14.2
Adenosarcoma	52.3 ± 3
Granulosa tumours	53.2 ± 6
Sertoli cell tumours	45.7 ± 3

Bilateral involvement of both ovaries was reported in more than half of cases (52%, n = 66), followed by the right ovary 27.6 (n = 35) and the left ovary 20.5% (n = 2). of all surface epithelial tumours 77.1% (n = 98), 13.3% (n = 13) were borderline tumour. Of all ovarian tumours, serous adenocarcinoma was the most common type (44.1%), followed by mucinous adenocarcinoma 12.6%, detailed of other types are shown in Table 3. The second reported an ovarian tumour was sex cord-stromal tumours which comprised 11.23% of all cases detailed are shown in Table 3. Germ cell tumour was reported in only 1.6% (n = 2) of cases, while metastatic cancer is most commonly seen from the colon (6.3%) and only 3.9 % from breasts (Table 3).

Most of the patients 39 (30.7%) presented in stage IIIC, and 32 (25.2%) presented at stage IV, while 14 (11.0%) of patients presented in stage IC,

and 9 (7.1%) patients presented in stage IIA, and only 8 (6.3%) patients in stage IB, 7 (5.5%) patients at stage IA (Table 4).

Table 3: Rate of occurrence of different ovarian malignancies among the study population

Frequency of surface epithelial-stromal 77.2% (98)			
Pathology pattern		Frequency	%
Serous tumours	Borderline serous	4	3.1
	serous adenocarcinoma	56	44.1
Musinous tumours	Borderline mucinous tumor	1	0.8
	mucinous adenocarcinoma	16	12.6
Endometrioid	endometrioid borderline tumour	4	3.1
tumours	endometrioid adenocarcinoma	3	2.4
Clear cell tumours	Borderline tumours	3	2.4
	clear cell adenocarcinoma	3	2.4
Transitional cell tumours	Borderline Transitional cell	1	0.8
Epithelial stromal	Adenosarcoma	4	3.1
	Carcinosarcoma (mixed muellerian tumor)	3	2.4
Frequency of sex cord-stromal tumours 14 (11.2%)			
Granulosa tumours		12	9.4
Sertoli cell tumours		2	1.6
Frequency of germ cell tumours 2 (1.6%)			
Immature Teratoma		1	0.8
Mixed germ cell tumours		1	0.8
Frequency of metastatic cancer 13 (10.4%)			
Colonic appendiceal		8	6.3
Breast		5	3.9

Discussion

In the present study, we reported an incidence of ovarian epithelial carcinoma of 77.2%. It is approximating the 85% incidence rate quoted from European countries [8] while the even higher rate of incidence (90%) has been reported from United States [9]. The lower incidence of ovarian cancer in our study can be explained by the fact that black women are less likely to develop ovarian cancer compared to white women.

Table 4: Tumors stages at the time of presentation

Stage	Frequency	%
IA	7	5.5
IB	8	6.3
IC	14	11.0
IIA	9	7.1
IIB	6	4.7
IIC	6	4.7
IIIA	3	2.4
IIIB	3	2.4
IIIC	39	30.7
IV	32	25.2

Studies demonstrated that white women had the highest risk of developing ovarian cancer, followed by Hispanic, Asian, black, and American Indian women [10]. The variation in the incidence of ovarian cancer between nations may be due to other factors such as sample size in each study, biosocial differences of the population, and genetic and other environmental factors.

In the current study, 11.8% of studied cases had a positive family history of ovarian cancer. It was reported that positive family history is considered most important risk, probably mediated through inherited genetic mutation which was found to increase the risk by 5 – 10% compared 1.4% risk in the general population [11]. In the current study, the main age of patients at presentation was 52.36 ± 14.210 years ranged from 14 to 95 years, and almost two-thirds of patients were above 50 years of age, and only 14% of cases were < 40 years of age. Similar results have been obtained by other researchers. The American cancer society reported that ovarian cancer is rare in women less than 40 years of age. Typically the diseases develop after menopause, and almost 50%of all ovarian cancers are found in women 63 years of age or older [12]. Similarly, the US Surveillance, Epidemiology, and End Results (SEER) database reported that ovarian neoplasm is a function of age after 50 years [13]. The mean age reported in the current study for endometrioid adenocarcinoma of the ovary (64 ± 4yrears) is consistent with 60 years of age reported by other authors [14].

The average age of menarche is the study group was 13.59 ± 2.706 years. Epidemiologic studies have inconsistent reports on associations between menarcheal age and ovarian cancer risk. One meta-analysis concluded that there was inversely associated between menarcheal age and the risk of ovarian cancer [15]. It is suggested that later menarcheal age will result in a decreased incidence of ovarian cancer by decreasing a woman's lifetime number of ovulation.

There is consistent literature that infertility and low parity increase the risk of ovarian cancer and multiparity and the use of oral contraceptives decreases the risk [16]. In the present study showed that 16.5% (n = 21) were nulliparous women, 37% (n = 47) were multiparous, while the majority 45.7 % (n = -58) are grand multiparous women. The use ovulation induction medications and hormonal replacement therapy were linked to increased risk of ovarian cancer and the use of HRT for a shorter duration are associated with 20% of ovarian cancer [17]. We reported that few of our patients used ovulation induction treatment (8.7%) and HRT (1.6%).

The incidence of endometrioid adenocarcinoma of the ovary in the current study was 5.5% which is less than 10 - 25% reported in the literature [11] but consistent with a report from Africa countries (4.5%) [18]. The present study, sex - cord stroma cell comprises 11.2% of all ovarian neoplasm, and the majority were granulosa cell tumors which comprise 9.4% of cases; a higher incidence (34.4%) was reported by Akakpo from Ghana opposed to a comparatively similar (7%) incidence rate from USA [19].

We reported an overall 1.6% incidence of germ cell tumours which are mainly immature teratoma and mixed germ cell tumour. Previous studies reported an incidence of 1.1% and 2.6% from Africa [18] and USA [20]. Although the disease is rarely reported in older age, the mean age for immature teratoma in this study was 52.1 years.

In the present study, the incidence of secondary metastatic cancer to ovary was 10.4% this was mainly from colon (6.3%) and ovary (3.9%). The figure is relatively lower than the incidence reported by Stewart et al. [21] who analysed 116 patients diagnosed with metastatic ovarian cancer at the Radboud University Nijmegen Medical Centre; they reported a 15% incidence rate. The latter authors found that 39% were from the gastrointestinal followed by breast in 28% and endometrium in 20% of cases [18].

In the present study, bilateral involvement of both ovaries was 52%, while has been reported in only 25% of cases [22][23]. What the frequency of bilaterality of ovarian neoplasm depend primarily on tumour type is involved. Pejovic et al. [24] was the first to raise the question whether bilateral ovarian carcinoma is a due metastasis from another ovary or it occurs as a result of two independent primary tumours. Analysis by karyotyping and genomic hybridisation concluded that bilaterality occurs by a metastatic process [22]. The high occurrence of bilateral (52%) ovarian neoplasm in the present study could be explained by advanced tumour stages at presentation which is an indication poor 5 - year's survival rate.

In conclusion, the incidence of different types of ovarian cancers in the present study is similar to worldwide incidence. The surface epithelial tumour is the commonest ovarian cancer, of which serous adenocarcinoma is the commonest and most of our patients present in late stages. The limitations of this study are the limited number of cases included and being a single centre rather than a multicenter study which is more informative. Further study with a large number of cases is warranted to investigate the predictors of ovarian malignancies among Sudanese women.

References

1. Markman, M. Development of an ovarian cancer symptom index: Possibilities for earlier detection. J Cancer. 2007; 110(1):226-27. https://doi.org/10.1002/cncr.22749 PMid:17486562

2. Ryerson AB, Eheman C, Burton J, McCall N, Blackman D, Subramanian S, et al. Symptoms, diagnoses, and time to key diagnostic procedures among older US women with ovarian cancer. Obstetrics & Gynecology. 2007; 109(5):1053-61. https://doi.org/10.1097/01.AOG.0000260392.70365.5e PMid:17470582

3. Ofor IE, Obeagu K, Ochei, Odo M. International Journal Of Current Research In Chemistry And Pharmaceutical Sciences. Int J Curr Res Chem Pharm Sci. 2016; 3(2):20-28.

4. Ganesan K, Morani AC, Marcal LP, Bhosale PR, and Elsayes KM. Cross-Sectional Imaging of the Uterus, in Cross-Sectional Imaging of the Abdomen and Pelvis. Springer, 2015:875-936. https://doi.org/10.1007/978-1-4939-1884-3_27

5. Yazbek JK, Raju J, Ben-Nagi TK, Holland K, Hillaby, Jurkovic D. Effect of quality of gynaecological ultrasonography on the management of patients with suspected ovarian cancer: a randomised controlled trial. The Lancet Oncology. 2008; 9(2):124-31. https://doi.org/10.1016/S1470-2045(08)70005-6

6. Acharya UR, Molinari F, Sree SV, Swapna G, Saba L, Guerriero S, et al. Ovarian Tissue Characterization in ultrasound a review. Technology in cancer research & treatment. 2015; 14(3):251-61. https://doi.org/10.1177/1533034614547445 PMid:25230716

7. Moyer VA. Screening for ovarian cancer: US Preventive Services Task Force reaffirmation recommendation statement. Annals of internal medicine. 2012; 157(12):900-04. https://doi.org/10.7326/0003-4819-157-11-201212040-00539 PMid:22964825

8. Bapat SA, Mali AM, Koppikar CB, Kurrey NK. Stem and progenitor-like cells contribute to the aggressive behavior of human epithelial ovarian cancer. Cancer Research. 2005; 65(8):3025-29. https://doi.org/10.1158/0008-5472.CAN-04-3931 PMid:15833827

9. Vang R, Shih IM, Kurman RJ. Fallopian tube precursors of ovarian low-and high-grade serous neoplasms. Histopathology. 2013; 62(1):44-58. https://doi.org/10.1111/his.12046 PMid:23240669

10. Beckmeyer-Borowko AB, Peterson CE, Brewer KC, Otoo MA, Davis FG, Hoskins KF, et al. The effect of time on racial differences in epithelial ovarian cancer (OVCA) diagnosis stage, overall and by histologic subtypes: a study of the National Cancer Database. Cancer Causes & Control. 2016; 27(10):1261-71. https://doi.org/10.1007/s10552-016-0806-6 PMid:27590306 PMCid:PMC5418550

11. Wright JD, Chen L, Tergas AI, Patankar S, Burke WM, Hou JY, et al. Trends in relative survival for ovarian cancer from 1975 to 2011. Obstetrics and gynecology. 2015; 125(6):1345. https://doi.org/10.1097/AOG.0000000000000854 PMid:26000505 PMCid:PMC4484269

12. Wentzensen N, Poole EM, Trabert B, White E, Arslan AA, Patel AV, et al. Ovarian Cancer Risk Factors by Histologic Subtype: An Analysis From the Ovarian Cancer Cohort Consortium. J Clin Oncol. 2016; 34(24):2888-98. https://doi.org/10.1200/JCO.2016.66.8178 PMid:27325851 PMCid:PMC5012665

13. Parazzini F, Franceschi S, La Vecchia C, Fasoli M.The epidemiology of ovarian cancer. Gynecologic oncology. 1991; 43(1):9-23. https://doi.org/10.1016/0090-8258(91)90003-N

14. Terada T. Endometrioid adenocarcinoma of the ovary arising in atypical endometriosis. International journal of clinical and experimental pathology. 2012; 5(9):924. PMid:23119109 PMCid:PMC3484489

15. Akakpo PK, Derkyi-Kwarteng L, Gyasi RK, Quayson SE, Anim JT. Ovarian Cancer in Ghana, a 10 Year Histopathological Review of Cases at Korle Bu Teaching Hospital. African Journal of Reproductive Health. 2015; 19(4):102-06. PMid:27337859

16. Gong TT, Wu QJ, Vogtmann E, Lin B, Wang YL.Age at menarche and risk of ovarian cancer: a meta-analysis of epidemiological studies. Int J Cancer. 2013;132(12):2894-900. https://doi.org/10.1002/ijc.27952 PMid:23175139 PMCid:PMC3806278

17. Winn ML, Lee NC, Rhodes PH, Layde PM, Rubin GL. Pregnancy, breast feeding, and oral contraceptives and the risk of epithelial ovarian cancer. J Clin Epidemiol. 1990; 43:559-568. https://doi.org/10.1016/0895-4356(90)90160-Q

18. Collaborative Group On Epidemiological Studies Of Ovarian Cancer, Beral V, Gaitskell K, Hermon C, Moser K, Reeves G, Peto R. Menopausal hormone use and ovarian cancer risk: individual participant meta-analysis of 52 epidemiological studies. Lancet. 2015; 385(9980):1835-42. https://doi.org/10.1016/S0140-6736(14)61687-1

19. Horta M, Cunha TM. Sex cord-stromal tumors of the ovary: a comprehensive review and update for radiologists. Diagnostic and Interventional Radiology. 2015; 21(4):277. https://doi.org/10.5152/dir.2015.34414 PMid:26054417 PMCid:PMC4498422

20. Levitin A, Haller K, Cohen HL, Zinn DL, O'Connor M. Endodermal sinus tumor of the ovary: imaging evaluation. AJR. American journal of roentgenology. 1996; 167(3):791-93. https://doi.org/10.2214/ajr.167.3.8751702 PMid:8751702

21. Stewart CJ, Leung YC, Whitehouse A. Fallopian tube metastases of non-gynaecological origin: a series of 20 cases emphasizing patterns of involvement including intra-epithelial spread. Histopathology. 2012; 60(6B):E106-E14. https://doi.org/10.1111/j.1365-2559.2012.04194.x PMid:22394169

22. Micci F, Haugom L, Ahlquist T, Abeler VM, Trope CG, Lothe RA, et al. Tumor spreading to the contralateral ovary in bilateral ovarian carcinoma is a late event in clonal evolution. Journal of oncology. 2009; 2010.

23. Kumar V, Abbas AK, Fausto N, Aster JC. Robbins and Cotran pathologic basis of disease. Elsevier Health Sciences, 2014.

24. Pejovic T, Heim S, Mandahl N, Elmfors B, Furgyik S, Flodérus UM, et al. Bilateral ovarian carcinoma: cytogenetic evidence of unicentric origin. International journal of cancer. 1991; 47(3):358-61. https://doi.org/10.1002/ijc.2910470308 PMid:1993543