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Association of Ovarian Tumour with Sociodemographic Background in a Tertiary Level Hospital

*Mst. Shaheen Nawrozy,[1] Marjina Khatun,[2] Sharmin Afrozy,[3]
Abu Hena Mostafa Kamal,[4] Ferdousi Sultana[5]

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Abstract

Intrduction: *Ovarian cancer is a common cause of death in women due to malignancy. Aim of this study was to observe the probable association of some predisposing factors with ovarian tumours.*

Methods: *This study was conducted at the Department of Obstetrics and Gynaecology of Rangpur Medical College Hospital, Rangpur from July 2012 to June 2014. Ovarian tumour cases diagnosed by history, clinical examination and ultrasonography were included in this study by purposive sampling technique; recurrent cases were excluded. Findings were expressed as 'Percentage involved'. In addition, χ^2 test, student's't' test and Odd's ratio were also used for statistical analysis.*

Results: *Total cases were 31–benign 24 and malignant 7. Peak age incidence of benign cases was about 35 years and of malignant cases was above 60 years. Past history of Pelvic Inflammatory Disease (PID) or endometriosis, or family history of ovarian tumour was negative among the cases. Mean parity of benign cases was 2.416 and of malignants was 1.857 ($p>0.10$). Among the oral pill users and non-users, Odd's ratio for benign vs malignant cases was 1.128 ($p>0.10$). Mean CA-125 level and ESR were higher in malignant than the benign (both p value<0.001). Histological types were serous cystadenoma, dermoid cyst, mucinous cystadenoma, poorly differentiated adenocarcinoma, serous cyst adeno-carcinoma and immature teratoma.*

Conclusion: *This small sample size is not at all suitable to draw any inference. Yet, considering peak incidence of benign cases around 35 years and of malignant cases was above 60 years of age; and, Odd's ratio goes in favour of protective role of oral pill against ovarian malignancy. Lack of awareness of our patients might have played some role for our non-conclusive findings regarding past medical or family history.*

Key words: *Ovarian tumour, Age, Parity, Oral contraceptive*

* 1. *Assistant Registrar, Department of Obstetrics and Gynaecology, Mohammad Ali Hospital, Bogra*
2. *Assistant Professor, Department of Community Medicine, Kumudini Women's Medical College, Mirzapur, Tangail*
3. *Lecturer, Department of Physiology, Shaheed Ziaur Rahman Medical College, Bogra*
4. *Assistant Professor, Department of Biochemistry, Kushtia Medical College, Kushtia*
5. *Professor, Department of Obstetrics and Gynaecology, Rangpur Medical College, Rangpur*

Correspondence *Mst. Shaheen Nawrozy, Email: shaheen.nawrozy@gmail.com*

Introduction

Ovarian cancer is a common gynaecological malignancy and one of the most common causes of death in women with malignancy; life time risk of ovarian cancer has been demonstrated to be 1.7% in general population.[1] The incidence of ovarian cancer is 7% out of total female cancers.[2] As ovary is

complex in its embryology and histology and has the potential to develop malignancy, ovarian neoplasm exhibits a wide variation in structure and biological behavior. The ovaries, after the uterus, are the second common site for development of gynaecological malignancy and the prognosis remains poor.[3] Malignant ovarian tumours are leading cause of death from gynaecological cancer.[4] But early stage of this disease is associated with poorly defined or vague symptoms, which often are not severe enough to prompt a woman to seek medical attention.[1] Excluding those which have an endocrine function, ovarian tumours are amazingly quite and rarely give rise to symptoms other than those induced mechanically by the size of the mass. This is why they are really dangerous and the malignant ones are often inoperable by the time they are diagnosed–commonly in stage III and IV.[5]

Benign tumours may occur at any point of life but they are most common during child bearing age with the peak incidence being between 25 and 34 years of age.[6] Borderline malignant ovarian tumours occur most frequently in 30 to 50 years whereas invasive carcinomas are seen more frequently between 50 to 70 years; and, germ cell tumours generally occur prior to puberty or in early adult life. An ovarian tumour in adolescent and postmenopausal women is more often malignant than benign. Most of the germ cell tumours occur in young girls.[5]

Early age at menarche and late age at menopause increase the risk of ovarian cancer whereas pregnancy and lactation reduce the risk.[7] Prolonged lactation is associated with lower risk of ovarian cancer.[8] Pelvic inflammatory disease (PID), especially those 35 years and younger were more likely to have developed ovarian cancer than control during 3 years of follow up.[9] Endometriosis is also known to be associated with endometroid müllarian adenocarcinoma.[4]

One strong risk factor of ovarian cancer is family history of the disease. Approximately 10-15% of ovarian cancers are attributed to genetic causes. In breast-ovarian cancer syndrome, majority of patients have mutation in BRCA 1 or BRCA 2 gene. Lynch II syndrome (hereditary non-polyposis colorectal cancer) also has 12% risk of developing ovarian cancer along with risk of developing colon, endometrial, breast cancers.[1]

One study with 7,308 cases and 32,717 controls demonstrated that the longer a women had used oral contraceptive pills the greater the reduction in ovarian cancer risk (p<0.0001). This reduction in risk persisted for more than 30 years after oral contraceptive pills use had ceased, with gradual attenuation of risk reduction over those 30 years.[10] Nulliparity and infertility were found to be associated with an increased risk and multiparity with a reduced risk of benign epithelial ovarian neoplasm. Infertility and PID were associated with increased risks of functional and dermoid cysts.[6] A Finnish cohort study including 87,929 cases also demonstrated lower risk of ovarian cancer in grand multipara, no matter, how many children and at which ages they had delivered.[11]

Evaluating all these into consideration, this study was conducted to observe the association of some predisposing factors with ovarian tumours found at Rangpur Medical College Hospital, Rangpur.

Materials and Methods

This cross sectional descriptive study was conducted at the Department of Obstetrics and Gynaecology of Rangpur Medical College Hospital, Rangpur from July 2012 to June 2014. Cases of the study were enrolled from the same Department and Institution.

The total number of cases (n) included in this study was 31. Sample was collected from in-patients' department by purposive sampling technique. Patients with ovarian tumours diagnosed by history, clinical examination and ultrasonography were included in this study. Previously diagnosed and treated ovarian tumours (recurrent case) were excluded.

The findings were expressed as 'percentage involved'. In addition, χ^2 test, student's '*t*' test and Odds ratio were also used and $p<0.05$ was considered as level of significance.

Results

Among all the cases (n=31) benign cases were 24 (77.41%) and malignant cases were 7 (22.59%) in number. Age range was 16 to 65 years, benign cases were 16 to 50 years of age with a pick incidence of 35 years and malignant cases were 24 to 65 years of age with a peak incidence above 60 years. No significant past medical history as PID or endometriosis that might have association with ovarian tumour was found from all the cases. As well as, family history of ovarian tumour was also negative.

Parity of all the cases ranged from 0 to 6, of which, among benign cases, it was 0 to 6 with mean (±SD) being 2.416 (±1.639) and among malignant cases, it was 1 to 6 with mean (±SD) being 1.857 (±1.864); the difference between the two groups was insignificant ($p>0.10$). (Table I).

Table I: Parity among benign and malignant cases of the study population (n-31)

Type of tumour	Parity (mean ± SD)	*p* value
Benign (n_1-24)	2.416 ± 1.639	>0.10
Malignant (n_2-7)	1.857 ± 1.864	

Student's '*t*' test demonstrates insignificant difference between the two groups.

Among all the cases, 9 (29%) gave history of oral contraceptive pills use only, 5 (16%) gave history of oral contraceptive pills and others (depot progesterone injections or IUCD), 6 (19%) gave history of depot progesterone injections or Intrauterine Contraceptive Device (IUCD) and 11 (36%) gave no history of the use of any of the above. (Table II).

Table II: Shows the use of different contraceptive methods in this study group (n-31)

Types of Contraceptive Used	Number of cases (n-31)	Subtypes of Contraceptive Used	Number of cases (n-31)
Users of Oral contraceptives	14 (45%)	Oral contraceptive pills only	9 (29%)
		Oral contraceptive pills & others (depot progesterone injections or IUCD)	5 (16%)
Non-users of Oral contraceptives	17 (55%)	Others (depot progesterone injections or IUCD)	6 (19%)
		No contraceptive (None of the above)	11 (36%)

Table III: Distribution of benign and malignant cases among the users and non-users of oral contraceptive pills (n-31)

Category	Malignant (n_2-7)	Benign (n_1-24)	*p* value
Oral pill users (14 cases)	3 (21%)	11 (79%)	
			>0.10
Oral pill non-users (17 cases)	4 (24%)	13 (76%)	

Odds ratio for this distribution is 1.128 which goes in favour of the comment that non-users of oral pill are more prone to develop malignant ovarian tumours than the users.

Among the oral pill users (14 cases), 11 (79%) were benign and 3 (21%) were malignant and among the non-users (17 cases), 13 (76%) were benign and 4 (24 %) were malignant and, the difference was insignificant (p>0.10). (Table III).

Mean (±SD) of CA 125 level in benign group was 25.97 (±15.98) and in malignant group was 7234.82 (±1120.22) (p<0.001); mean (±SD) of ESR in benign group was 24.50 (±13.83) and in malignant group was 46.57 (±14.51) (p<0.001); and, mean (±SD) of Hb% in benign group was 10.79 (±1.01) and in malignant group was 9.75 (±1.72) (p<0.10). (Table IV).

Table IV: Serum CA 125 level, erythrocyte sedimentation rate (ESR) and haemoglobin (Hb%) of benign and malignant cases are as follows

Parameters	Benign cases	Malignant cases	*p* value
Serum CA 125 level (Unit/Litre) Mean ± SD	25.97 ± 15.98	7234.82 ± 1120.22	<0.001
ESR (mm at 1st hour) Mean ± SD	24.50 ± 13.83	46.57 ± 14.51	<0.001
Hb% (gm/dl) Mean ± SD	10.79 ± 1.01	9.75 ± 1.72	> 0.10

Student's '*t*' test was used to demonstrate the level of significance.

The different types of tumours found in this study show that among all the cases (n-31), 12 (38.71%) were serous cystadenoma, 7 (22.58%) dermoid cyst, 5 cases (16.13%) mucinous cystadenoma, 4 (12.90%) poorly differentiated adenocarcinoma, 2 (6.45%) serous cyst adeno-carcinoma and 1 (3.23%) was immature teratoma (Figure 1).

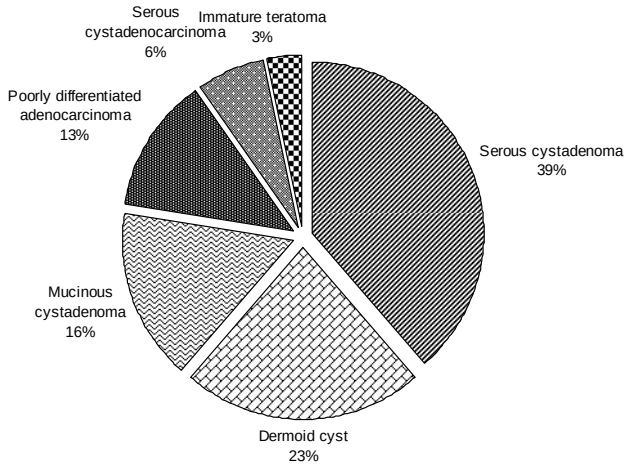

Figure 1: Different histological types of tumours found in this study (n-31)

Discussion

Among the 31 cases included in the present study, 24 (77.41%) were benign and 7 (22.59%) were malignant. In a study including 1,066 ovarian tumour patients, Timmerman et al.[12] found 800 (75%) cases had benign tumours and 266 (25%) had malignant tumours. Another study with 110 cases also found 80 (72%) benign and 30 (28%) malignant cases.[13] Both results are comparable to this study.

Here, the peak incidence of benign tumours was around the age of 35 years and the peak incidence of malignant tumour was above 60 years. Bukhari et al. found that the incidence of benign tumour is more in 20-40 years of age and for malignant tumour it is above 50 years of age.[14] Another study, demonstrated the maximum incidence of benign tumour around 40 years and maximum incidence of malignant tumour above 50 years.[15] Both are, more or less, comparable to this study.

In this study, some nullipara, as well as some grand multipara were found to develop ovarian neoplasm. Mean parity was higher in benign group than in malignant group, but the difference was insignificant ($p>0.10$). As there was no non-neoplastic control group in this study, it is not possible to demonstrate the protective role of parity in the development of ovarian neoplasm. On the other hand, contraceptive prevalence rate in our community has also been increased remarkably and so, scope of comparing grand multipara with the others has been reduced. In one study, multiparity was found to be associated with a significant reduction in risk of ovarian cancer (Odds ratio = 0.6 for 3, and 0.5 for 4 births).[16]

Again, in this study, it was found that 14 (45%) cases used oral contraceptive pills, 6 (19%) cases used anything other than oral pills and 11 (36%) cases used none as contraceptive. Among the non-users and users of oral pills, Odds ratio was 1.128 for malignant and benign ovarian tumours which goes in favour of the comment that "non-users of oral pill are more prone to develop malignant ovarian tumours than the users"; of course the difference was insignificant for 'χ^2' ($p>0.10$). To find out the protective role of oral contraceptive in the development of ovarian neoplasm, a large case control or a cohort study is required.

In the malignant group, CA-125 level ($p<0.001$) and ESR ($p<0.001$) were significantly higher

and Haemoglobin (p>0.10) was insignificantly lower. Terzic et al., involving 112 malignant and 544 benign cases, found mean CA-125 level to be 937.13 Units/Litre in the malignant group and 59.54 U/L in benign group (p<0.000); regarding mean ESR level, their result was 40.16 mm at 1st hour in the malignant group and 19.88 mm at 1st hour in benign group (p<0.000)[17].

The most common histologic type was serous cystadenoma 12 (38.71%). The next was dermoid cyst 7 (22.58%). And, then were mucinous cystadenoma 5 (16.13%), poorly differentiated adenocarcinoma 4 (12.90%), serous cyst adenocarcinoma 2 (6.45%) and immature teratoma 1 (3.23%).

Comparable to this study, done by Mondal et al., involving 957 cases over a period of 10 years, found serous cystadenoma (29.9%) as the most common histological type; followed by, were mature teratoma (15.9%) and mucinous cystadenoma (11.1%). Surface epithelial tumours (60.9%) were the major proportion of malignant ovarian tumours. Serous cystadeno-carcinoma was the predominant malignant tumour (11.3%). Bilateral malignant serous tumours were 49.5%.[18] The incidences found by Danish et al. are as follows: serous cystadenoma (32%), dermoid cyst (22%), mucinous cystadenoma (17%), poorly differentiated tumours (5%), serous cystadenocarcinoma (4%), mucinous cystadenocarcinoma (3%)etc.[19] Which is also more or less comparable to the histologic types of this study.

After all, this small sample size is not at all suitable to draw any inference. Yet, it may be assumed that lack of awareness of our patients probably has contributed to some of our non-conclusive findings regarding past history as PID or endometriosis, or early menarche, or late menopause, or family history of ovarian tumour.

Limitations of this study include

1. Sample size '31' is too small to predict anything definitely.
2. Present study does not represent the whole scenario of our community. Because there are many private hospitals and district hospitals as well, which are also giving services to our population and their scenario are not similar to this hospital.

Contribution of the Authors

First author was the main researcher. Others helped in data collection and statistical analysis.

References

1. Levy G, Purcell K. Premalignant and Malignant Disorders of Ovaries and Oviducts. In: DeCherney AH, Nathan L, Laufer N, Romen AS. Current Diagnosis and Treatment Obstetrics and Gynaecology, 11th ed. USA: McGraw-Hill Companies, Inc. 2013: p.848.
2. Dhillon PK, Yeole BB, Dikshit R, Kurkure AP, Bray F. Trends in breast, ovarian and cervical cancer incidence in Mumbai, India over a 30-year period, 1976–2005: an age–period–cohort analysis. Br J Cancer. 2011, 105: 723–730.
3. Padubidri VG, Daftary SN. Disorders of the Ovary and Benign Tumours. In: Shaw's Text Book of Gynaecology, 14th ed. USA: Elsevier Publishing, Thomson Press. 2008: p.329-333.
4. Gabra H. Epithelial Ovarian Cancer. In: Edmonds DK, ed. Dewhurst's Textbook of Obstetrics and Gynaecology, 7th ed. USA: Black Well Publishing, Oxford University Press. 2007: p.625.
5. Kumar P, Malhotra N. Tumours of Ovary. In: Jeffcoate's Principles of Gynaecology, 7th ed. India: Jaypee Brothers Medical Publishers (P) Ltd. 2008: p.524..

6. Westhoff CL, Beral V. Patterns of ovarian cyst hospital discharge rates in England and Wales, 1962-79. BMJ. 1984; 289: 1348-1349.

7. Pieta B, Chmaj-Wiezchowska K, Opala T. Past obstetric history and risk of ovarian cancer. Ann Agric Environ Med. 2012; 19(3): 385-388.

8. Su D, Pasalich M, Lee AH, Binns WC. Ovarian cancer risk is reduced by prolonged lactation: a case-control study in southern China. Am J Clin Nutr. 2013; 97: 354-357.

9. Lin HW, Tu YY, Lin SY, Su WJ, Lin WL, Lin WJ, et al. Risk of ovarian cancer in women with pelvic inflammatory disease: a population based study. Lancet Oncol. 2011; 12(9): 900-904.

10. Beral V, Doll R, Hermon C, Peto R, Reeves G and Collaborative Group on Epidemiological Studies on Ovarian Cancer. Ovarian cancer and oral contraceptives: collaborative reanalysis of data from 45 epidemiological studies including 23,257 women with ovarian cancer and 87,303 controls. Lancet. 2008; 371 (9609): 303-314.

11. Hinkula M, Pukkala E, Kyyronen P, Kauppila A. Incidence of ovarian cancer of grand multiparous women- a population based study in Finland. Gynecol Oncol. 2006; 103(1): 207-211.

12. Timmerman D, Testa AC, Bourne T, Farrazzi E, Ameye L, Konstantinovic ML, et al. Logistic Regression Model to Distinguish Between the Benign and Malignant Adnexal Mass Before Surgery: A Multicenter Study by the International Ovarian Tumour Analysis Group. J Clin Oncol. 2005; 23 (34): 8794-8801.

13. Wasim T, Majrroh A, Siddiq S. Comparison of clinical presentation of Benign and Malignant Ovarian Tumours. J Pak Med Assoc. 2009; 59: 18-21.

14. Bukhari U, Menon Q, Menon H. Frequency and Pattern of Ovarian Tumours. Pak J Med Sci. 2011; 27 (4): 884-886.

15. Kayastha S. Study of ovarian tumours in Nepal Medical College Teaching Hospital. Nepal Med Coll J. 2009; 11(3): 200-202.

16. Chiaffarino F, Pelucchi C, Parazzini F, Negri E, Franceschi S, Talamini R,et al. Reproductive and hormonal factors and ovarian cancer. Ann Oncol. 2001; 12: 337-341.

17. Terzic MM, Dotlic J, Likic I, Ladjevic N, Brndusic N, Arsenovic N, et al. Current diagnostic approach to patients with adnexal masses: which tools are relevant in routine praxis? Chin J Cancer Res. 2013; 25(1): 55-62.

18. Mondal SK, Banyopadhyay R, Nag DR, Roychowdhury 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 Cancer Res Ther. 2011; 7(4): 433-437.

19. Danish F, Khanzada MS, Mirza T, Aziz S, Naz E, Khan MN. Histomorphological spectrum of ovarian tumours with immunohistochemical analysis of poorly or undifferentiated malignancies. Gomal J Med Sc. 2012; 10(2): 209-215.