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Young and Overlooked: Malignant Ovarian Germ Cell Tumours in a West African Cohort

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Abstract

Introduction

Malignant ovarian germ cell tumours (mOGCTs) are rare but highly curable ovarian neoplasms when diagnosed and treated promptly. However, evidence comparing yolk sac tumour (YST)-associated and non-YST mOGCTs in sub-Saharan Africa remains limited. We compared the clinicopathological characteristics, treatment patterns, and survival outcomes of women with YST-associated and non-YST mOGCTs managed at a major referral centre in Ghana.

Methods

We conducted a retrospective cohort study of women with histologically confirmed mOGCTs presented at a multidisciplinary gynaecologic oncology tumour board between 2013 and 2024. Demographic, clinicopathological, treatment, and survival data were extracted from a prospectively maintained database. Patients were classified according to the presence or absence of a YST component. Overall survival was estimated using the Kaplan–Meier method and compared using the log-rank test.

Results

Fifty-eight women were included (median age, 22 years; interquartile range [IQR], 15–45), of whom 58.6% were nulliparous. YST-associated tumours accounted for 41.4% (n = 24) of cases and occurred in significantly younger women than non-YST tumours (median age, 18.5 vs 35.5 years; p = 0.001). Women with YST-associated tumours more frequently presented with International Federation of Gynaecology and Obstetrics (FIGO) stage III–IV disease (69.6% vs 38.5%; p = 0.004) and were more likely to receive no cancer-specific treatment (25.0% vs 2.9%; p = 0.010). Only 13.8% underwent computed tomography (CT) scan for pre-treatment staging. Alpha-fetoprotein (AFP) levels were significantly higher in YST-associated tumours, whereas serum lactate dehydrogenase (LDH) levels were higher in non-YST tumours. The median surgery-to-chemotherapy interval was 105.5 days, and 61.1% (n = 11) of chemotherapy recipients had residual, recurrent, or metastatic disease at treatment initiation. Median overall survival was 14.4 months (95% CI, 8.7–27.3), with a 3-year survival of 29.5%. Three-year survival was significantly lower in women with YST-associated tumours than in those with non-YST tumours (12.5% vs 41.9%; log-rank p = 0.003).

Conclusion

mOGCTs in this cohort were characterised by a high prevalence of YST-associated tumours, advanced-stage presentation, delayed initiation of chemotherapy, and poor survival. Strengthening early referral, improving access to staging investigations and immunohistochemistry (IHC), and reducing surgery-to-chemotherapy delays may improve outcomes in resource-limited settings.

1. Introduction

mOGCTs rare but account for a disproportionate share of ovarian malignancies among prepubertal girls, adolescents, and young women.[1] Among Indigenous Black women in West Africa and women of African ancestry in the diaspora, mOGCTs constitute approximately 5.2%–11.5% of all primary ovarian malignancies.[2–4] These tumours include dysgerminomas, yolk sac tumours, embryonal carcinomas, malignant teratomas—including immature teratoma (IT) and mature cystic teratoma with malignant transformation (MCT_MT)—non-gestational choriocarcinomas, and mixed germ cell tumours.[5] mOGCTs arise from primitive germ cells and exhibit varying degrees of differentiation along somatic and extraembryonic lineages.[6] Dysgerminoma represents the least differentiated subtype, with minimal somatic or extraembryonic differentiation. Embryonal carcinoma resembles embryonic stem cells and is capable of divergent differentiation along both somatic and extraembryonic pathways. Malignant teratomas demonstrate differentiation towards embryonic or somatic tissues, whereas YST and choriocarcinomas represent extraembryonic derivatives of primitive germ cells. Tumours containing two or more mOGCT subtypes are classified as mixed germ cell tumours (MxGCT).[7]

The clinical manifestations of mOGCTs are generally nonspecific.[1] Most patients present with abdominal pain, abdominal distension, or a palpable abdominopelvic mass, reflecting the rapid growth of these neoplasms.[8] Pelvic ultrasonography is usually the initial imaging modality for evaluating an adnexal mass; however, its diagnostic specificity for mOGCTs remains limited, even when combined with established risk-stratification algorithms.[9] CT scan of the chest, abdomen, and pelvis is central to pretreatment staging because it delineates locoregional disease, identifies peritoneal and distant metastases, and assists in assessing resectability.[10] Serum tumour markers also play a pivotal role in the diagnostic evaluation of mOGCTs.[1] Elevations in AFP, LDH, and human chorionic gonadotrophin (hCG) may occur depending on the underlying histological subtype. A markedly elevated AFP, with or without an elevated hCG level, in a young woman presenting with an adnexal mass is highly suggestive of an mOGCT. MxGCT may demonstrate variable biomarker profiles, reflecting the marker-producing characteristics of their constituent histological components.

mOGCTs are clinically important because of their aggressive biological behaviour, rapid growth, and propensity for early dissemination.[8] Despite this aggressiveness, they remain among the most chemosensitive solid malignancies.[11] Fertility-sparing surgery followed by platinum-based chemotherapy constitutes the cornerstone of treatment, particularly because most affected patients are adolescents or young women who may not yet have completed their reproductive plans.[12] The introduction of platinum-based regimens, especially bleomycin, etoposide, and cisplatin (BEP), has substantially improved outcomes, with cure rates exceeding 80% even among patients presenting with advanced-stage disease.[11]

mOGCTs comprise a biologically and histologically heterogeneous group of neoplasms characterised by rapid growth, early dissemination, and prognosis influenced by age, disease stage, histological subtype, and treatment.[13] Although Indigenous Black women in West Africa and the African diaspora bear a

relatively high burden of these tumours,[2–4] contemporary data comparing histology-specific outcomes remain limited. In particular, whether tumours containing a YST component differ from non-YST tumours in clinical presentation, treatment pathways, and survival has not been adequately examined in sub-Saharan Africa. Given the recognised aggressive biological behaviour of YST, we hypothesised that the presence of a YST component identifies a subgroup of mOGCTs characterised by more advanced-stage presentation and poorer survival than non-YST tumours. We therefore compared the clinicopathological characteristics, treatment patterns, and survival outcomes of women with YST-associated and non-YST malignant ovarian germ cell tumours managed at a major referral centre in Ghana.

2. Methods

2.1 Study design

We conducted a retrospective observational cohort study of patients with histology-confirmed malignant ovarian germ cell tumours who were presented to the multidisciplinary gynaecologic cancer tumour board at Komfo Anokye Teaching Hospital (KATH) in Ghana from 2013 to 2024.

2.2 Study Setting and Patient Management

This study was conducted at KATH, a major referral hospital in Kumasi, Ghana. The Obstetric and Gynaecology Department of KATH provides specialised gynaecologic oncology services through a multidisciplinary tumour board comprising local specialists and international collaborators. In 2023, the institution managed 291 histologically confirmed gynaecological malignancies, including cervical, ovarian, uterine, vulvar, and gestational trophoblastic cancers.

All patients were staged according to the FIGO classification. Treatment recommendations were formulated by the multidisciplinary tumour board guided by contemporary evidence-based practice, including National Comprehensive Cancer Network (NCCN) recommendations. Patients presented at initial consultation as either treatment-naïve or following surgery performed elsewhere with a histopathological diagnosis already established. Pelvic and abdominal ultrasonography was routinely performed in all women with suspected ovarian cancer. Cross-sectional imaging with CT and/or MRI was performed when clinically indicated and financially feasible, particularly in patients who had undergone prior surgery. Serum AFP, hCG, and LDH levels were routinely requested in all women aged 40 years and below presenting with clinically suspected ovarian cancer. Serum cancer antigen 125 (CA-125) was selectively requested in patients with clinical, radiological, or intraoperative features suggestive of extensive intra-abdominal disease or lymph node involvement. In previously operated patients, disease status was defined as either operated on with residual disease or operated on without residual disease. For this study, residual disease was defined as the presence of gross macroscopic tumour at the time of initial consultation, irrespective of the surgery-consultation interval.

In patients with extensive abdominopelvic disease, poor performance status, or significant medical comorbidities, image-guided biopsy or mini-laparotomy tissue sampling was performed to establish a histological diagnosis before neoadjuvant chemotherapy. Standard surgical management consisted of tumour resection and omentectomy, with or without lymph node assessment. Histological diagnosis was largely based on haematoxylin and eosin (H&E)-stained sections. IHC was selectively employed in morphologically challenging tumours and cases with mixed or ambiguous histological features where distinction from important differential diagnoses was required.

The first and second review visits were scheduled at 2 and 4 weeks, after primary surgery, respectively. Chemotherapy was generally initiated within 3–5 weeks of the primary definitive surgery when clinically feasible. The FIGO stage, histological subtype, extent of surgical debulking, and treatment intent dictated the indications for chemotherapy. Patients with completely resected tumours generally received three cycles of bleomycin–etoposide–cisplatin (BEP), whereas those with residual, recurrent, or metastatic disease typically received four cycles. Patients undergoing neoadjuvant chemotherapy or those with persistent macroscopic disease after surgery also received platinum-based chemotherapy according to tumour burden and clinical response. Younger patients or those considered unsuitable for cisplatin-based treatment received carboplatin, etoposide, and bleomycin for 4–6 cycles, in accordance with institutional practice and clinician judgement. Treatment response was evaluated using either the World Health Organisation (WHO) response criteria or the Response Evaluation Criteria in Solid Tumours (RECIST), depending on the treatment period and available clinical data.

2.3 Data source

The monthly Gynaecologic cancer tumour board maintains a prospectively updated database containing demographic, clinical, pathological, treatment, and follow-up information on women evaluated for suspected ovarian malignancies. Records of eligible patients managed between January 2013 and December 2024 were reviewed (Fig. 1). During this period, 58 patients with histologically confirmed mOGCTs were identified and included in the study. Data extraction was performed independently by two gynaecologic oncology fellows using a standardised data collection form. Missing or incomplete data were reconciled by reviewing of additional hospital records, medical charts, operative notes, pathology reports, chemotherapy records, and follow-up documentation. Most of the missing data were for the survival status (alive or deceased) of patients who were lost to follow-up. In these cases, patients or their relatives were contacted to confirm patient status after obtaining verbal consent.

2.4 Statistical analysis

Demographic, clinical, and treatment-related variables were summarised using frequencies and proportions for categorical variables and means with standard deviations or medians with interquartile ranges (IQRs) for continuous variables, as appropriate. The primary outcome was overall survival (OS), defined as the interval from histological diagnosis to death from any cause or the date of the last documented follow-up. Patients were categorised into YST-associated and non-YST groups. Survival probabilities were estimated using the Kaplan–Meier method and compared using the log-rank test, with a two-sided p-value < 0.05 considered statistically significant. Multivariable survival analysis was not performed because the relatively small sample size and limited number of survival events would have resulted in model overfitting and unstable hazard ratio estimates. [14] Accordingly, all subgroup analyses were

considered exploratory and should be interpreted cautiously because of limited statistical power. Statistical analyses were performed using Stata® version 17.0 (StataCorp, College Station, TX, USA).

2.5 Ethical approval

The study protocol has been published previously.[15] Ethical approval was obtained from the Institutional Review Board and Research and Development Unit of KATH (KATH/IRB/CA/167/25) and the Committee on Human Research, Publications and Ethics of Kwame Nkrumah University of Science and Technology (CHRPE/AP/1030/25). As part of routine clinical care, contact information is collected for all women with ovarian cancer to facilitate patient navigation and minimise treatment default and loss to follow-up. For patients with incomplete clinical data, patients or their next of kin were contacted by telephone after obtaining informed verbal consent, in accordance with ethics committee-approved procedures. This approach was approved by both institutional review boards and is consistent with previously published methods.[16,17]

3. Results

3.1 Histopathological and disease characteristics

The study included 58 women with mOGCTs (Table 1). Pure YST was the most common histological subtype (27.6%), followed by MCT-MT (20.7%), dysgerminoma (19.0%), IT (17.2%), and MxGCT (13.8%). MxGCT, IT, and YST were diagnosed at median ages of 14.5, 17, and 19 years, respectively. Among the 49 treatment-naïve women, 25 (51.0%) presented with advanced disease (FIGO stage III–IV), representing the majority of cases. IHC was performed only in five women (8.6%). One daughter (2.2%) of a woman with a personal history of breast cancer and dysgerminoma tested positive for BRCA2 (p.R2341C) and MSH3 (p.P96L) variants of unknown significance. The IHC cases comprised MxGCTs and an MCT-MT (Cases 372, 242, 252, 83, and 409; Table 2).

3.2 Demographic characteristics

The median age of the cohort was 22 years (IQR, 15–45 years) (Table 3). Women with YST-associated tumours were significantly younger than those with non-YST tumours (median age, 18.5 vs 35.5 years; p = 0.0003). They also had a lower median body mass index (BMI) (20.1 vs 24.1 kg/m²; p = 0.0102). Women with YST-associated tumours were more frequently premenopausal (80.0% vs 48.5%; p = 0.024) and nulliparous (80.0% vs 42.4%; p = 0.007). They were also more likely to be often single (84.0% vs 48.5%), whereas non-YST tumours were more often married (39.4% vs 16.0%; p = 0.009). Occupation did not differ significantly between the groups. Overall, traders comprised the largest occupation group (31.0%, n=18), followed by senior high school students (25.9%, n=15), elementary school pupils (15.5%, n=9) and junior high school students (10.3%, n=6). Farmers (5.2%, n=3), unemployed women (5.2%, n=3), apprentices (1.7%, n=1), and women in other occupations (5.2%, n=3) accounted for the remainder.

3.3 Clinical presentation, imaging, and tumour marker profile

There were no significant differences between the YST and non-YST groups with respect to symptom duration, haemoglobin concentration, use of CT/MRI imaging, tumour size, presence of peritoneal carcinomatosis, distant metastasis, or median CA-125 levels (Table 4). The median symptom duration was 4 months (IQR, 2–5 months) in both groups. Abdominal distension was the most common presenting symptom, occurring in 50.0% (n=29) of women, followed by abdominopelvic mass in 36.2% (n=21) and abdominal pain in 12.1% (n=7). Vaginal bleeding occurred in one patient, who was subsequently diagnosed with a MxGCT comprising YST, dysgerminoma, IT, and choriocarcinoma components. Median AFP levels were significantly higher among women with YST-associated tumours than among those without a YST component (1210.0 vs 3.9 ng/mL; p = 0.0278), with 15 patients (62.5%) in the YST group having AFP levels exceeding 1,000 ng/mL. The median serum LDH levels were significantly higher in the non-YST group (median, 332.6 vs 198.5 U/L; p = 0.0413).

3.4 Treatment patterns and outcomes

Women with non-YST tumours were significantly more likely to undergo surgery alone than those with YST-associated tumours (25 vs 8; P=0.004) (Table 5). Conversely, women with YST-associated tumours more frequently presented with advanced disease (FIGO stage III/IV, 69.6% vs 38.5%; P = 0.045) and were more likely to receive no cancer-specific treatment (6 vs 1; P = 0.004). Among the 18 women who received chemotherapy, 11 (61.1%) had residual, recurrent, or metastatic disease at treatment initiation. The median surgery-to-chemotherapy interval was 105.5 days (IQR, 40–235.5). No significant between-group differences were observed in chemotherapy receipt (P = 0.256), number of chemotherapy cycles administered (P = 0.367), or surgery-to-chemotherapy interval (P = 0.128). Chemotherapy regimens differed significantly between the groups (P = 0.002), with women in the YST group more likely to receive bleomycin–etoposide–cisplatin (BEP). Survival status at last follow-up also differed significantly, with a greater proportion of women with non-YST tumours alive than those with YST-associated tumours (14 vs 3; P = 0.022).

3.5 Overall survival and subgroup analyses

The median overall survival was 14.4 months (95% CI, 9.0–31.3), with estimated survival probabilities of 75.9%, 58.3%, and 29.5% at 6, 12, and 36 months, respectively (Figure 2; Supplementary Table S1). Three-year survival was significantly lower among women with YST-associated than non-YST tumours (12.0% vs 43.6%; log-rank $P = 0.003$) (Figure 3; Supplementary Table S2) and among those with FIGO stage III/IV compared with stage I/II disease (11.5% vs 49.7%; log-rank $P < 0.001$) (Figure 4; Supplementary Table S4). Survival did not differ significantly according to receipt of first-line chemotherapy (23.4% vs 32.9%; log-rank $P = 0.856$) (Figure 5; Supplementary Table S3).

4. Discussion

This study provides one of the few contemporary evaluations of mOGCTs in sub-Saharan Africa and demonstrates that poor survival reflects the combined effects of aggressive tumour biology and deficiencies across the diagnostic and treatment pathway. Women with tumours containing a YST component

represented a distinct high-risk subgroup characterised by advanced-stage presentation and markedly poorer survival.

Dysgerminoma is the predominant mOGCT in Western and Asian populations, followed by IT and YST.[18] In contrast, YST-containing tumours accounted for over 40% of our cohort, consistent with the reported lower incidence of dysgerminoma among Black females.[1] YST is also frequently represented in MxGCTs, commonly in combination with dysgerminoma or IT.[19,20] In our cohort, YST with IT was the most frequent mixed histology, followed by YST with embryonal carcinoma and dysgerminoma. The predominance of YST, either pure or as a component of MxGCTs, may suggest a predilection for extraembryonic lineage differentiation in this population. [6] However, evolving referral patterns following the establishment of in-country gynaecologic oncology training at KATH may also have influenced the observed distribution.

In our cohort, IT and MxGCT predominated in early to mid-adolescence, YST in late adolescence, dysgerminoma in young adulthood, and MCT-MT among older women. The median age of the overall cohort was 22 years, with women with YST-associated tumours being significantly younger (18.5 years). Although age-specific distributions across mOGCT histologies remain incompletely characterised, this finding is consistent with the recognised predilection of YST for adolescence and early adulthood. A multicentre review similarly reported a median age of 13 years among patients with YST-associated germ cell tumours, further highlighting the young age at presentation of this histological subtype.[21]

While haematoxylin and eosin (H&E) sections remain the cornerstone of diagnosis, recognition of an age–histology algorithm may be particularly valuable where IHC and molecular testing are limited. In adolescents, a markedly elevated serum AFP strongly supports YST, potentially reducing the need for extensive IHC panels. However, CD30 may help identify or exclude an embryonal carcinoma component in YST-associated mixed tumours, as demonstrated in Cases 372 and 427. Given the aggressive behaviour and poor survival associated with YST, IHC confirmation is particularly important when characteristic reticular or microcystic architecture is equivocal.[22] Absence of AFP and Glypican-3 expression favours alternative germ cell histotypes.

For poorly differentiated non-epithelial ovarian tumours, SALL4 is useful for establishing germ cell differentiation and distinguishing these tumours from potential mimics, including sex cord-stromal tumours and somatic malignancies arising in MCT-MT.[23] When dysgerminoma is suspected, nuclear OCT4 and membranous CD117 (c-KIT), with PLAP as a supportive marker, provide a useful diagnostic immunophenotype.[24–26] Cytokeratin assessment may additionally assist in evaluating a suspected somatic carcinoma arising within MCT-MT.

Although employment status did not differ significantly between the groups, important patterns emerged that provide insight into the social and economic implications of disease diagnosis.

The higher proportion of school-going girls with YST-associated tumours reflected the younger age profile of this subgroup and is consistent with the recognised predilection of YST for children, adolescents, and young adults.[21] Diagnosis and treatment during this formative period may disrupt education, psychosocial development, and future socioeconomic opportunities, highlighting the importance of age-appropriate supportive care, educational continuity, and fertility-preserving treatment strategies.

Only about one-third of patients presented within three months of symptom onset, with most reporting a longer symptom duration, contrasting with reports that most women with ovarian tumours seek specialist consultation within three months.[27] Although abdominopelvic mass with or without pain is commonly reported in mOGCTs, abdominal distension predominated in our cohort. This may reflect the advanced disease burden, as more than half of treatment-naïve women had FIGO stage III–IV disease. The combination of advanced disease stage at presentation and a relatively short median symptom duration is consistent with the rapid growth and aggressive biology of mOGCTs, suggesting that delays across presentation, referral, diagnostic evaluation, and treatment initiation may permit substantial disease progression..

Our findings are highly suggestive of the limitations in diagnostic capacity. Cross-sectional imaging was performed in only a small proportion of patients, despite its role in detecting peritoneal and distant metastases and assessing resectability.[10] Limited access to CT or MRI may therefore result in underestimation of disease burden and constrain optimal treatment planning. The use of cross-sectional imaging in pre-treatment staging largely depends on the patient's ability to pay. Serum biomarkers may provide valuable diagnostic support in this context. AFP was significantly elevated in YST-associated tumours, with nearly two-thirds exceeding 1,000 ng/mL, a level associated with adverse prognosis.[28] Conversely, higher LDH levels were observed in non-YST tumours, likely as a result of the contribution of dysgerminoma, for which LDH is a recognised surrogate marker.[29] These findings support integrating tumour markers into resource-adapted diagnostic pathways, while recognising that biomarkers cannot substitute for appropriate imaging and histopathological evaluation.

An encouraging finding was the frequent use of fertility-preserving surgery, particularly among women with YST-associated tumours, more than 80% of whom underwent uterus-sparing procedures. This demonstrates adherence to contemporary fertility-preserving principles despite resource constraints.[12] Given the young age of women affected by mOGCTs and the favourable reproductive outcomes reported after conservative surgery, preservation of reproductive potential remains an important component of quality cancer care.

Treatment-related challenges represent an important modifiable determinant of outcome. Despite the established effectiveness of surgery followed by platinum-based chemotherapy, approximately one-tenth of women received no cancer-specific treatment, while the median surgery-to-chemotherapy interval exceeded 100 days.[30] Moreover, many women receiving chemotherapy had residual or metastatic disease at the time of the initiation of chemotherapy. Therefore, the absence of an observed survival benefit from chemotherapy should be interpreted with caution and does not imply a lack of chemotherapeutic efficacy. The intent of treatment was largely palliative rather than an adjuvant due to an advanced or progressive disease state. Aggressive tumour biology, advanced presentation, and delayed systemic treatment may have attenuated the expected benefits of multimodal therapy. These observed treatment gaps likely reflect complex patient- and health-system-level challenges, including, but not limited to, delayed referral, inadequate patient navigation, and limited

supportive care.[31,32] Therefore, reducing delays to definitive treatment represents a potentially actionable strategy for improving outcomes in resource-limited settings.

The median overall survival was only 14.4 months, with an estimated 3-year survival of less than 30%, compared with cure rates exceeding 80% and 5-year survival rates above 95% in contemporary series.[11] Particularly striking was the poor outcomes in women with tumours containing a YST component associated with YST-containing tumours, with a three-year survival of only 12.5%. MxGCT containing a YST component were grouped with pure YST because the presence of YST is considered the principal determinant of tumour behaviour. Consequently, even when admixed with dysgerminoma, IT, or embryonal carcinoma, the YST component is likely to drive the tumour's clinical course, therapeutic requirements, and prognosis. Grouping tumours according to the presence or absence of a YST component therefore provides a biologically and clinically more meaningful classification than one based solely on the predominant histological subtype. Nevertheless, the magnitude of the observed survival disparity is unlikely to be explained by tumour biology alone.[33] Women with FIGO stage I–II disease had markedly better survival than those presenting with stage III–IV disease, highlighting the adverse impact of advanced presentation. Survival in this population depends not only on tumour characteristics but also on the modifiable diagnostic infrastructure, access to chemotherapy, and effective patient navigation.

Strengths and limitations

This study provides one of the few contemporary evaluations of malignant ovarian germ cell tumours in sub-Saharan Africa. It offers a novel comparison of YST-associated and non-YST tumours. It used prospectively maintained, multidisciplinary tumour board data to depict real-world oncology practice in a resource-limited setting. This generates relevant evidence to inform diagnosis, treatment pathways, and policy aimed at improving outcomes of a unique ovarian cancer histotype prevalent in younger women.

This study has several limitations. First, its retrospective design was susceptible to incomplete clinical documentation, missing data, and variation in the quality of routinely collected records. Although additional hospital records and telephone follow-up were used to ascertain survival status, incomplete follow-up information may have introduced outcome misclassification. Second, the limitation posed by the rarity of malignant ovarian germ cell tumours is reflected in the sample size, reduced statistical power for subgroup analyses, and the exclusion of reliable Cox regression for survival modelling. This should limit the interpretation of effect estimates of the study. Third, a higher chance of including women with advanced disease is expected, as the study in a tertiary referral-centre cohort is subject to referral bias. A small number of women received cross-sectional imaging, potentially resulting in incomplete pre-treatment staging, while the selective application of immunohistochemical assessments may have contributed to possible misclassification. Most decisions were based on expert radiological and histological evaluations conducted by the gynaecological oncology tumour board. Variation in diagnostic work-up, surgical management, chemotherapy utilisation, and treatment pathways introduced residual confounding that could not be fully accounted for in the analysis. As a tertiary referral-centre cohort, patients presented at the multidisciplinary tumour board may represent a higher-risk subgroup than women with mOGCTs diagnosed nationally, increasing the likelihood of referral bias and overrepresentation of advanced disease. These findings may affect the precision and generalisability of the findings; however, they also reflect the real-world challenges encountered in managing malignant ovarian germ cell tumours in resource-constrained settings and underscore the need for prospective multicentre studies across sub-Saharan Africa.

Conclusion

Malignant ovarian germ cell tumours in this West African cohort predominantly affected adolescents and young women and were frequently characterised by YST histology and advanced-stage presentation. In contrast to resource-endowed settings, the survival outcomes in the cohort were substantially poor, and this is consistent with limited access to cross-sectional imaging, selective use of IHC, and late initiation of adjuvant chemotherapy after definitive surgery. These findings suggest that deficiencies across the diagnostic and treatment pathway, rather than tumour biology alone, contribute to the observed survival gap. Improving survival outcomes for women with malignant ovarian germ cell tumours in Ghana and the subregion would require improved access to cross-sectional imaging and IHC, and timely initiation of platinum-based chemotherapy.

Abbreviations

BEP	Bleomycin, Etoposide and Cisplatin
BRCA2	Breast Cancer Susceptibility Gene 2
FIGO	International Federation of Gynecology and Obstetrics
H&E	Haematoxylin and Eosin
IHC	Immunohistochemistry
KATH	Komfo Anokye Teaching Hospital
N/A	Not Available
NCCN	National Comprehensive Cancer Network
RECIST	Response Evaluation Criteria in Solid Tumours
WHO	World Health Organization

YST Yolk Sac Tumour

Declarations

Authors' contributions

KAA: Conceptualisation, methodology, data curation, formal analysis, investigation, writing—original draft, writing—review and editing. RA: Conceptualisation, writing—original draft, writing—review and editing. YN: Conceptualisation, formal analysis, writing—original draft, writing—review and editing. AAK: Writing—review and editing. AS: Writing—review and editing. KA: Writing—review and editing. FD: Writing—review and editing. LDR: Writing—review and editing. RH: Writing—review and editing. GOP: Writing—review and editing. KD: Writing—review and editing. AAS: Writing—review and editing. ECD: Conceptualisation, methodology, formal analysis, supervision, writing—original draft, writing—review and editing. All authors read and approved the final manuscript.

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Data availability

Data generated/analysed during this study are available from the corresponding author on reasonable request.

Declaration

Ethics approval and consent to participant

The study protocol has been published previously.15 Ethical approval was obtained from the Institutional Review Board and Research and Development Unit of KATH (KATH/IRB/CA/167/25) and the Committee on Human Research, Publications and Ethics of Kwame Nkrumah University of Science and Technology (CHRPE/AP/1030/25). As part of routine clinical care, contact information is collected for all women with ovarian cancer to facilitate patient navigation and minimise treatment default and loss to follow-up. For patients with incomplete clinical data, patients or their next of kin were contacted by telephone after obtaining informed verbal consent, in accordance with ethics committee-approved procedures. This approach was approved by both institutional review boards and is consistent with previously published methods

Content for publication

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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References

1. Brown J, Friedlander M, Backes FJ, Harter P, O'Connor DM, De La Motte Rouge T, et al. Gynecologic Cancer Intergroup (GCIG) Consensus Review for Ovarian Germ Cell Tumors. Int J Gynecol Cancer. 2014 Nov;24:S48–54. doi:10.1097/IGC.0000000000000223
2. Akakpo PK, Derkyi-Kwarteng L, Gyasi RK, Quayson SE, Naporo S, Anim JT. A pathological and clinical study of 706 primary tumours of the ovary in the largest tertiary hospital in Ghana. BMC Womens Health. 2017 Apr 17;17(1):34. doi:10.1186/s12905-017-0389-8
3. Etukudo A, Mustapha A, Adamu ZA, Umar SS, Tukur Y, Yahya A, et al. Ovarian Cancers in a Northern Nigerian Hospital: Epidemiology, Clinicopathology and Treatment Options. Niger Med J J Niger Med Assoc. 66(1):99–108. doi:10.71480/nmj.v66i1.604 PubMed PMID: 40309546; PubMed Central PMCID: PMC12038617.
4. George SH, Omotoso A, Pinto A, Mustapha A, Sanchez-Covarrubias AP, Umar UA, et al. An Assessment of Ovarian Cancer Histotypes Across the African Diaspora. Front Oncol. 2021 Nov 26;11. doi:10.3389/fonc.2021.732443
5. Alvarado-Cabrero I. Germ Cell Tumors, Pathology of the Fallopian Tube. In: Stolnicu S, Ali-Fehmi R, editors. Gynecologic Pathology [Internet]. Cham: Springer International Publishing; 2023. p. 113–4. Available from: https://doi.org/10.1007/978-3-030-97378-0_5639 doi:10.1007/978-3-030-97378-0_5639

6. Müller MR, Skowron MA, Albers P, Nettersheim D. Molecular and epigenetic pathogenesis of germ cell tumors. Asian J Urol. 2021 Apr;8(2):144–54. doi:10.1016/j.ajur.2020.05.009 PubMed PMID: 33996469; PubMed Central PMCID: PMC8099689.
7. Goyal LD, Kaur B, Badyal RK. Malignant Mixed Germ Cell Tumors of the Ovary: A Series of Rare Cases. J Reprod Infertil. 2019;20(4):231–6. PubMed PMID: 31897390; PubMed Central PMCID: PMC6928409.
8. Umezu T, Kajiyama H, Terauchi M, Shibata K, Ino K, Nawa A, et al. Long-term outcome and prognostic factors for yolk sac tumor of the ovary. Nagoya J Med Sci. 2008 Mar;70(1–2):29–34. PubMed PMID: 18807293.
9. Moore RG, McMeekin DS, Brown AK, DiSilvestro P, Miller MC, Allard WJ, et al. A novel multiple marker bioassay utilizing HE4 and CA125 for the prediction of ovarian cancer in patients with a pelvic mass. Gynecol Oncol. 2009 Jan;112(1):40–6. doi:10.1016/j.ygyno.2008.08.031 PubMed PMID: 18851871; PubMed Central PMCID: PMC3594094.
10. Sureka B, Meena V, Garg P, Yadav T, Khera PS. Computed tomography imaging of ovarian peritoneal carcinomatosis: a pictorial review. Pol J Radiol. 2018 Dec 4;83:e500–9. doi:10.5114/pjr.2018.80247 PubMed PMID: 30655930; PubMed Central PMCID: PMC6334186.
11. Solheim O, Kærn J, Tropé CG, Rokkones E, Dahl AA, Nesland JM, et al. Malignant ovarian germ cell tumors: Presentation, survival and second cancer in a population based Norwegian cohort (1953–2009). Gynecol Oncol. 2013 Nov 1;131(2):330–5. doi:10.1016/j.ygyno.2013.08.028
12. Ayhan A, Tunç M, Akıllı H, Haberal N, Haberal A. Fertility-sparing approach to malignant ovarian germ cell tumors - Oncologic and obstetric outocome: A retrospective study. Int J Gynaecol Obstet Off Organ Int Fed Gynaecol Obstet. 2024 Mar;164(3):1074–9. doi:10.1002/ijgo.15141 PubMed PMID: 37737565.
13. Hodroj K, Stevovic A, Attignon V, Ferraioli D, Meeus P, Croce S, et al. Molecular Characterization of Ovarian Yolk Sac Tumor (OYST). Cancers. 2021 Jan 9;13(2):220. doi:10.3390/cancers13020220 PubMed PMID: 33435376; PubMed Central PMCID: PMC7826864.
14. Tanner KT, Keogh RH, Coupland CAC, Hippisley-Cox J, Diaz-Ordaz K. Dynamic updating of clinical survival prediction models in a changing environment. Diagn Progn Res. 2023 Dec 12;7(1):24. doi:10.1186/s41512-023-00163-z PubMed PMID: 38082429; PubMed Central PMCID: PMC10714456.
15. Amo-Antwi K, Nartey Y, Agambire R, Davis-Rivera L, Haghighat R, Ocloo BW, et al. Women With Suspected Diagnosis of Ovarian Cancer in Ghana: How Much Do We Know About Them? Front Oncol. 2026 Feb 25;16. doi:10.3389/fonc.2026.1771387
16. Amo-Antwi K, Agambire R, Konney TO, Nguah SB, Dassah ET, Nartey Y, et al. Health-related quality of life among cervical cancer survivors at a tertiary hospital in Ghana. PLOS ONE. 2022 Jun 3;17(6):e0268831. doi:10.1371/journal.pone.0268831
17. Karasik LB, Tamis-LeMonda CS, Ossmy O, Adolph KE. The ties that bind: Cradling in Tajikistan. PLOS ONE. 2018 Oct 31;13(10):e0204428. doi:10.1371/journal.pone.0204428
18. Matz M, Coleman MP, Sant M, Chirlaque MD, Visser O, Gore M, et al. The Histology of Ovarian Cancer: Worldwide Distribution and Implications for International Survival Comparisons (CONCORD-2). Gynecol Oncol. 2017 Feb;144(2):405–13. doi:10.1016/j.ygyno.2016.10.019 PubMed PMID: 27931752; PubMed Central PMCID: PMC6195192.
19. Goyal LD, Kaur B, Badyal RK. Malignant Mixed Germ Cell Tumors of the Ovary: A Series of Rare Cases. J Reprod Infertil. 2019;20(4):231–6. PubMed PMID: 31897390; PubMed Central PMCID: PMC6928409.
20. Tiwary B, Sinha HH, Pandey VK. Malignant mixed germ cell tumor of ovary: a rare case report. Int J Reprod Contracept Obstet Gynecol. 2015;4(2):511–3.
21. Faure Conter C, Xia C, Gershenson D, Hurteau J, Covens A, Pashankar F, et al. Ovarian Yolk Sac Tumors; Does Age Matter? Int J Gynecol Cancer Off J Int Gynecol Cancer Soc. 2018 Jan;28(1):77–84. doi:10.1097/IGC.0000000000001149 PubMed PMID: 29194189.
22. Tumas JB, Chan J. Educational Case: Ovarian neoplasms: A series of educational cases to review malignancies of epithelial, stromal, and germ cell origin. Acad Pathol. 2023 Apr 24;10(2):100080. doi:10.1016/j.acpath.2023.100080 PubMed PMID: 37168283; PubMed Central PMCID: PMC10164830.
23. Miettinen M, Wang Z, Mc. Cue PA, Sarlomo-Rikala M, Rys J, Biernat W, et al. SALL4 expression in germ cell and non germ-cell tumors – a systematic immunohistochemical study of 3215 cases. Am J Surg Pathol. 2014 Mar;38(3):410–20. doi:10.1097/PAS.0000000000000116 PubMed PMID: 24525512; PubMed Central PMCID: PMC4041084.
24. Cheng L, Thomas A, Roth LM, Zheng W, Michael H, Karim FWA. OCT4: a novel biomarker for dysgerminoma of the ovary. Am J Surg Pathol. 2004 Oct;28(10):1341–6. doi:10.1097/01.pas.0000135528.03942.1f PubMed PMID: 15371950.
25. Sever M, Jones TD, Roth LM, Karim FWA, Zheng W, Michael H, et al. Expression of CD117 (c-kit) receptor in dysgerminoma of the ovary: diagnostic and therapeutic implications. Mod Pathol Off J U S Can Acad Pathol Inc. 2005 Nov;18(11):1411–6. doi:10.1038/modpathol.3800463 PubMed PMID: 16056250.
26. Zhao Y, Li Y yuan, Xu Y hao. Clinicopathological significance of plap, NSE and WT1 detection in ovarian dysgerminoma. Chin J Cancer Res. 2003 Jun 1;15(2):136–9. doi:10.1007/BF02974918
27. Goff BA, Mandel LS, Melancon CH, Muntz HG. Frequency of symptoms of ovarian cancer in women presenting to primary care clinics. JAMA. 2004 Jun 9;291(22):2705–12. doi:10.1001/jama.291.22.2705 PubMed PMID: 15187051.
28. GUO YL, ZHANG YL, ZHU JQ. Prognostic value of serum α-fetoprotein in ovarian yolk sac tumors: A systematic review and meta-analysis. Mol Clin Oncol. 2015 Jan;3(1):125–32. doi:10.3892/mco.2014.417 PubMed PMID: 25469282; PubMed Central PMCID: PMC4251123.
29. Kilic C, Cakir C, Yuksel D, Kilic F, Kayikcioglu F, Koc S, et al. Ovarian Dysgerminoma: A Tertiary Center Experience. J Adolesc Young Adult Oncol. 2021 Jun;10(3):303–8. doi:10.1089/jayao.2020.0087 PubMed PMID: 32762565.
30. Feng Z, Wen H, Bi R, Yang W, Wu X. Prognostic impact of the time interval from primary surgery to intravenous chemotherapy in high grade serous ovarian cancer. Gynecol Oncol. 2016 Jun;141(3):466–70. doi:10.1016/j.ygyno.2016.04.016 PubMed PMID: 27098361.
31. Syed ST, Gerber BS, Sharp LK. Traveling Towards Disease: Transportation Barriers to Health Care Access. J Community Health. 2013 Oct;38(5):976–93. doi:10.1007/s10900-013-9681-1 PubMed PMID: 23543372; PubMed Central PMCID: PMC4265215.

32. Likhubi WK, Nyongesa CN, Kisienya FK. Evaluating the Impact of a Patient Navigation Program on Treatment Timeliness, Continuity, and Follow-Up Outcomes for Patients With Digestive Tract Cancer at Kenyatta Hospital. JCO Glob Oncol. 2026 Jun;12(6):e2500751. doi:10.1200/GO-25-00751
33. Mangili G, Sigismondi C, Gadducci A, Cormio G, Scollo P, Tateo S, et al. Outcome and risk factors for recurrence in malignant ovarian germ cell tumors: a MITO-9 retrospective study. Int J Gynecol Cancer Off J Int Gynecol Cancer Soc. 2011 Nov;21(8):1414–21. doi:10.1097/IGC.0b013e3182236582 PubMed PMID: 21795985.

Tables

Table 1: Clinicopathological characteristics of women with malignant ovarian germ cell tumours

Variables	Number[a] (N=58)	Percentage (%)
Disease status at initial consultation		
Treatment-naive	9	84.5
Prior-operated with no residual disease	3	5.2
Prior-operated with residual disease	6	10.3
Histology		
Dysgerminoma	11	19.0
Immature teratoma	10	17.2
MCT_MT	12	20.7
Yolk Sac Tumour	16	27.6
MxGCT	8	13.8
Embryonal Carcinoma	1	1.7
Median age at diagnosis, years (IQR)		
Dysgerminoma	27 (15-37)	
Immature teratoma	17 (14-20)	
[b]MCT_MT	54 (49.5-64.5)	
Yolk Sac Tumour	19 (15-27)	
[c]MxGCT	14.5 (8-20)	
Embryonal Carcinoma	38	
IHC test		
Yes	5	8.6
No	53	91.4
[d]Genetic test		
Yes	1	1.7
No	57	98.3
FIGO Stage, n=49		
Stage 1A/B	17	34.7
Stage 1C	4	8.2
Stage 2	3	6.1
Stage 3	13	26.5
Stage 4	12	24.5

Note: [a] *Values given as numbers unless otherwise stated*

[b]MCT_MT*: Mature cystic teratoma with malignant transformation;* [c]*MxGCT:Mixed germ cell tumours, Case 252: MxGCT with yolk sac tumour and clear cell components (1), Case 264 and 537: MxGCT with yolk sac tumour, dysgerminoma and immature teratoma components, Case 318: MxGCT with yolk sac tumour, dysgerminoma, immature teratoma and choriocarcinoma components, Case 427: MxGCT with yolk sac tumour and embryonal carcinoma components, Case 83*

and 520 extragondal MxGCT with yolk sac tumour and immature teratoma components and Case 372: MxGCT with yolk sac tumour, immature teratoma and embryonal carcinoma components.

[d] *Daughter of a woman diagnosed with dysgerminoma tested positive for BRCA2 (p.R2341C) and MSH3 (p.P96L) variants of unknown significance after genetic counselling and testing.*

Table 2 : Immunohistochemical findings in selected malignant ovarian germ cell tumours

[a]IHC TEST	[b]Case 372	Case 242	Case 252	Case 83	Case 409
GFAP	(+)	N/A	N/A	(+)	N/A
NSE	(+)	N/A	N/A	N/A	N/A
VM	(+)	N/A	N/A	N/A	N/A
OCT 3/4	N/A	(+)	N/A	N/A	N/A
CD 117	N/A	(+)	N/A	N/A	N/A
SALL-4	N/A	(+)	N/A	N/A	N/A
CD 10	N/A	(+)	N/A	N/A	N/A
CD 30	N/A	**(–)**	N/A	N/A	N/A
PLAP	N/A	N/A	**(+)**	N/A	N/A
AE1/AE3	N/A	**(–)**	**(+)**	N/A	N/A
CK 7	N/A	N/A	N/A	N/A	(+)
CK 20	N/A	**(–)**	**(–)**	N/A	(+)
WT 1	N/A	N/A	**(–)**	N/A	N/A
PAX_8	N/A	N/A	**(–)**	N/A	N/A
ER	N/A	N/A	**(–)**	N/A	N/A
NAPSIN A	N/A	N/A	**(–)**	N/A	N/A

Note: [a] IHC: *immunohistochemistry; GFAP, glial fibrillary acidic protein; NSE, neuron-specific enolase; VM, vimentin; OCT3/4, octamer-binding transcription factor 3/4; CD, cluster of differentiation; SALL4: spalt-like transcription factor 4; PLAP: placental alkaline phosphatase; AE1/AE3: pancytokeratin (AE1/AE3); CK, cytokeratin; WT1: Wilms tumour 1 protein; PAX8: paired box gene 8 protein; &Napsin A: Napsin A; N/A = not available*

[b] *Histopathological diagnosis: Case 372: Mixed germ cell tumour composed of yolk sac tumour, immature teratoma, and embryonal carcinoma components; Case 242: Dysgerminoma; Case 252: Mixed germ cell tumour composed of yolk sac tumour and clear cell carcinoma components; Case 83: Extragonadal mixed germ cell tumour composed of yolk sac tumour and immature teratoma components; Case 409: Mature cystic teratoma with associated mucinous carcinoma.*

Table 3. Demographic characteristics of women with malignant ovarian germ cell tumours

Variables , N=58	Total, n[a] (%)	YST n[a] (%)	NON-YST n[a] (%)	p value
Age, years				0.001
Median (IQR)	22 (15-45)	18.5 (12.5-24.0)	35.5 (18-50)	
≤ 11	6 (10.3)	4 (16.7)	2 (5.9)	
11- 39	36 (62.1)	19 (79.2)	17 (50.0)	
40+	16 (27.6)	1 (4.1)	15 (44.1)	
Body mass index, Kg/m^2				0.005
Median (IQR)	22.3 (17.8-27.8)	20.1 (17.8-25.0)	24.1 (18.7-32.7)	
Underweight	22 (37.9)	12 (50.0)	10 (29.4)	
Normal	14 (24.1)	6 (25.0)	8 (23.5)	
Overweight	10 (17.2)	5 (20.8)	5 (14.7)	
Obese	12 (20.7)	1 (4.2)	11 (32.4)	
Menopausal status				0.042
Pre-pubertal	12 (20.7)	4 (16.6)	8 (24.2)	
Premenopausal	36 (62.1)	19 (79.2)	16 (48.5)	
Postmenopausal	10 (17.2)	1 (4.2)	9 (27.3)	
Parity				
0	34 (58.6)	19 (79.2)	15 (44.1)	0.014
1+	24 (41.4)	5 (20.8)	19 (55.9)	
Education_level				0.822
Primary	18 (31.0)	7 (29.2)	11 (33.3)	
Junior	13 (22.4)	7 (29.2)	6 (18.2)	
Secondary	19 (32.8)	8 (33.3)	10 (30.3)	
Tertiary	1 (1.7)	0 (0.0)	1 (3.0)	
None	7 (12.1)	2 (8.3)	5 (15.2)	
Employment status				
Pupil	9 (15.5)	5 (20.8)	4 (11.7)	0.193
Junior High Student	6 (10.3)	3 (12.5)	3 (8.8)	
Senior High Student	15 (25.9)	9 (37.5)	6 (17.7)	
Apprentice	1 (1.7)	1 (4.2)	0 (0.0)	
Trader	18 (31.0)	4 (16.6)	14 (41.2)	
Farmer	3 (5.2)	0 (0.0)	3 (8.8)	
[b]Others	3 (5.2)	1 (4.2)	2 (5.9)	
Unemployed	3 (5.2)	1 (4.2)	2 (5.9)	
Marital status				0.009
Married	17 (29.3)	4 (16.7)	13 (38.2)	
Single	37 (63.8)	20 (83.3)	17 (50.0)	
Divorced /Widow	4 (6.9)	0 (0.0)	4 (11.8)	

Note: Values are presented as n (%) unless otherwise stated; P values were calculated using Fisher's exact test. IQR, interquartile range; [b]Others included seamstress (n = 2) and beautician (n = 1).

Table 4. Clinical Characteristics and tumour marker profiles of women with YST-associated and non-YST malignant ovarian germ cell tumours

Variables	Total, n[a] (%)	YST n (%)	NON-YST n (%)	p value
Symptoms				
Abdominal distension	29 (50.0)	13 (54.2)	16 (47.1)	0.636
Pelvic mass	21 (36.2)	8 (33.3)	13 (38.2)	
Abdominal pain	7 (12.1)	2 (8.3)	5 (14.7)	
[a]Vaginal bleeding	1 (1.7)	1 (4.2)	0 (0.0)	
Symptom duration, months				
Median (IQR)	**4 (2-5)**	**4 (2-5)**	**4 (2-5)**	0.728
< 3 months	18 (31.0)	8 (33.3)	10 (29.4)	
3-6 months	31 (53.5)	12 (50.0)	19 (55.9)	
6 + months	9 (15.5)	4 (16.7)	4 (14.7)	
Haemoglobin Level				0.558
Mean (SD)	11.2 (9.2-12.4)	10.8 (8.8-12.3)	11.3 (9.8-12.5)	
Normal	22 (37.9)	10 (41.7)	12 (35.3)	
Anaemic	36 (62.1)	14 (58.3)	21 (64.7)	
Use of CT/MRI scans				
Yes	8 (13.8)	5 (20.8)	3 (8.8)	0.255
No	50 (86.2)	19 (79.2)	19 (91.2)	
Tumour size				0.288
Median (IQR)	13.6 (11.7-18.9)	12.6 (11-18.9)	15 (12.6-18.7)	
<10	7 (14.2)	4 (17.4)	3 (11.5)	
10-20	30 (61.2)	14 (60.9)	16 (61.9)	
20+	12 (24.5)	5 (21.7)	7 (26.9)	
Peritoneal carcinomatosis n=49				
Yes	15 (30.6)	9 (40.9)	**6 (22.2)**	**0.217**
No	34 (69.4)	13 (59.1)	21 (77.8)	
Distant metastasis				
Yes	4 (6.1)	3 (13.6)	1 (3.7)	0.314
No	45 (93.9)	19 (86.4)	26 (96.3)	
Ca-125 Median (IQR), n=49	**70 (23-234.0)**	**141.1 (56.1-457.1)**	**45.7 (12.1-117.6)**	**0.141**
> 35	33 (67.4)	17 (81.0)	16 (57.1)	
=/< 35	16 (32.6)	4 (19.0)	12 (42.9)	
AFP (Median (IQR) n=46	**5.7 (3.4-1210)**	**1210 (543.3-2942.9)**	**3.9 (3.3-4.8)**	**0.028**
< 1000	33 (68.8)	9 (37.5)	24.0 (100.0)	
1000+	15 (31.2)	15 (62.5)	0 (0.0)	
LDH Median (IQR) n=45	**214 (175-433)**	**198.5 (175.5-306)**	**332.6 (174-675)**	**0.041**
< 280	29 (60.4)	17 (70.8)	12 (50.0)	
280 +	19 (39.6)	7 (29.2)	12 (50.0)	
HCG Median (IQR)	**2.1 (0.5-4.4)**	**2.1 (0.5-4.4)**	**2.8 (1.3-4.0)**	**0.288**
5 +	9 (19.6)	5 (20.0)	4 (19.0)	
< 5	37 (80.4)	20 (80.0)	17 (81.0)	

Note: Values are presented as n (%) unless otherwise stated; YST, yolk sac tumour and mixed germ cell tumours with yolk sac component; NON-YST, dysgerminoma, immature teratoma and mature cystic teratoma with squamous and mucinous carcinoma; IQR, interval quartile range; SD, standard deviation, Ca-125; AFP; LDH; FIGO: international Federation of Gynaecology and Obstetrics, [a]Vaginal bleeding*: A mixed germ cell tumour with yolk sac, dysgerminoma, immature teratoma and choriocarcinoma presented with vaginal bleeding.*

Table 5. Comparison of treatment patterns and clinical outcomes between YST-associated and non-YST malignant ovarian germ cell tumours.

Characteristics	Total, n[a] (%)	YST n (%)	NON-YST n (%)	p value
Disease status				0.347
Naive	49 (84.5)	22 (91.7)	27 (79.4)	
Prior-operated with no residual disease	3 (5.2)	0 (0.0)	3 (8.8)	
Prior-operated with residual disease	6 (10.3)	2 (8.3)	4 (11.8)	
Treatment Received				
Surgery + Chemotherapy	16 (27.6)	9 (37.5)	7 (20.6)	**0.004**
Surgery Alone	33 (56.9)	8 (33.3)	25 (73.5)	
Chemotherapy Alone	2 (3.5)	1 (4.2)	1 (2.9)	
No cancer-specific treatment	7 (12.1)	6 (25.0)	1 (2.9)	
FIGO Stage				**0.010**
Stage 1/2	24 (49.0)	6 (27.3)	16 (66.7)	
Stage 3/4	25 (51.0)	16 (72.7)	9 (33.3)	
Uterus, after surgery, N=49				**0.007**
Uterus-sparing	**27 (55.1)**	14 (82.4)	13 (40.6)	
Non-uterus-sparing	**22 (44.9)**	**3 (17.6)**	**19 (59.4)**	
Type of surgery				0.206
USO	5 (10.2)	2 (11.8)	3 (9.4)	
USO/OMT/SPB	20 (40.8)	11 (64.8)	9 (28.1)	
USO/PLND_PALND/OMT/SPB	2 (4.1)	1 (5.9)	1 (3.1)	
TAH/BSO	3 (6.1)	0 (0.0)	3 (9.4)	
TAH/BSO/OMT/SPB	12 (24.5)	2 (11.8)	10 (31.2)	
TAH/BSO/PLND_PALND/OMT	7 (14.3)	1 (5.8)	6 (18.8)	
Chemotherapy, receipt				0.256
Yes	**18 (31.0)**	10 (41.7)	8 (23.5)	
No	40 (69.0)	14 (58.3)	26 (76.5)	
Disease status before chemotherapy, n=18				0.174
No gross disease	7 (38.9)	2 (20.0)	5 (62.5)	
Local disease /unresected tumour	8 (44.4)	5 (50.0)	3 (37.5)	
Distant metastases	3 (16.7)	3 (30.0)	0 (0.0)	
SCI, days, median (IQR)	**105.5 (40-235.5)**	51.0 (29-215)	126 (113-528)	0.128
Chemotherapy regimen				**0.002**
BEP	12 (73.3)	10 (100.0)	2 (25.00)	
carboplatin–paclitaxel	3 (16.7)	0 (16.7)	3 (37.5)	
*Others	3 (16.7)	0 (16.7)	3 (37.5)	
Chemotherapy, cycles	4 (3-4)	3.5 (3-4)	5.0 (3.5-6.0)	0.069
< 4 cycles	11 (61.1)	5 (50.0)	2 (25.0)	
4+ cycles	7 (38.9)	5 (50.0)	6 (75.0)	
Follow-up outcome				**0.022**
Alive	17 (29.3)	**3 (12.5)**	14 (41.2)	
Dead	41 (70.7)	21 (87.5)	20 (58.8)	

Note: Values are presented as n (%) unless otherwise stated. YST, yolk sac tumour and mixed germ cell tumours containing a yolk sac tumour component; non-YST, dysgerminoma, immature teratoma, and mature cystic teratoma with malignant transformation; BA, biopsy alone; USO, unilateral salpingo-oophorectomy; OMT, omentectomy or omental biopsy; PLND, pelvic lymph node dissection; PALND, para-aortic lymph node dissection; SPB, selective peritoneal biopsy; TAH, total abdominal hysterectomy; BSO, bilateral salpingo-oophorectomy; SCI, surgery-to-chemotherapy interval; DCI, diagnosis-to-chemotherapy interval; IQR, interquartile range; BEP, bleomycin, etoposide, and cisplatin; Other chemotherapy: carboplatin or cisplatin combined with paclitaxel or etoposide. P-values were calculated using Pearson's chi-square test, Fisher's exact test, or independent-samples t test, as appropriate.

Figures

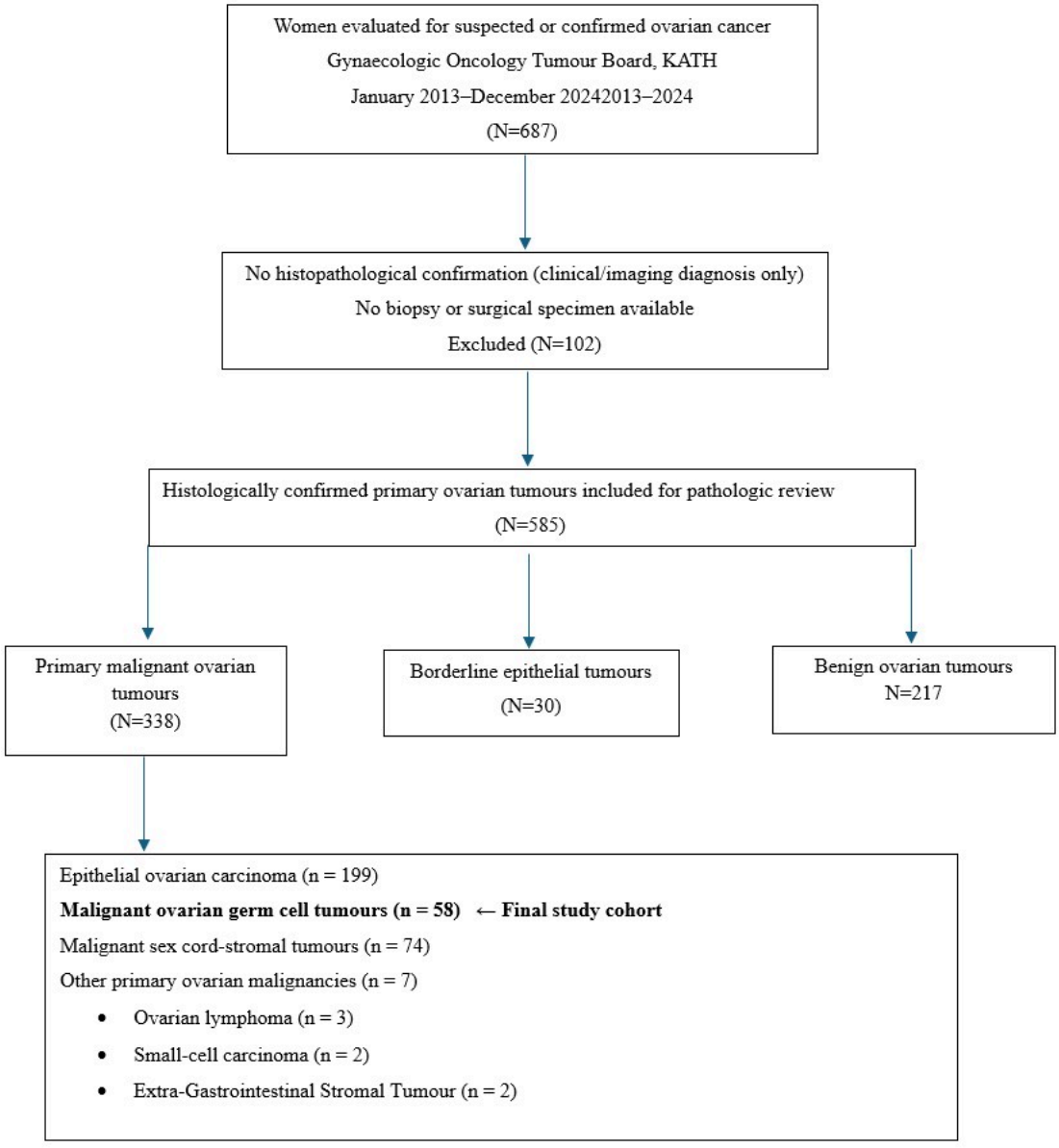


Figure 1

Flow diagram of participant selection for the malignant ovarian germ cell tumour cohort, Komfo Anokye Teaching Hospital, Ghana, 2013–2024.

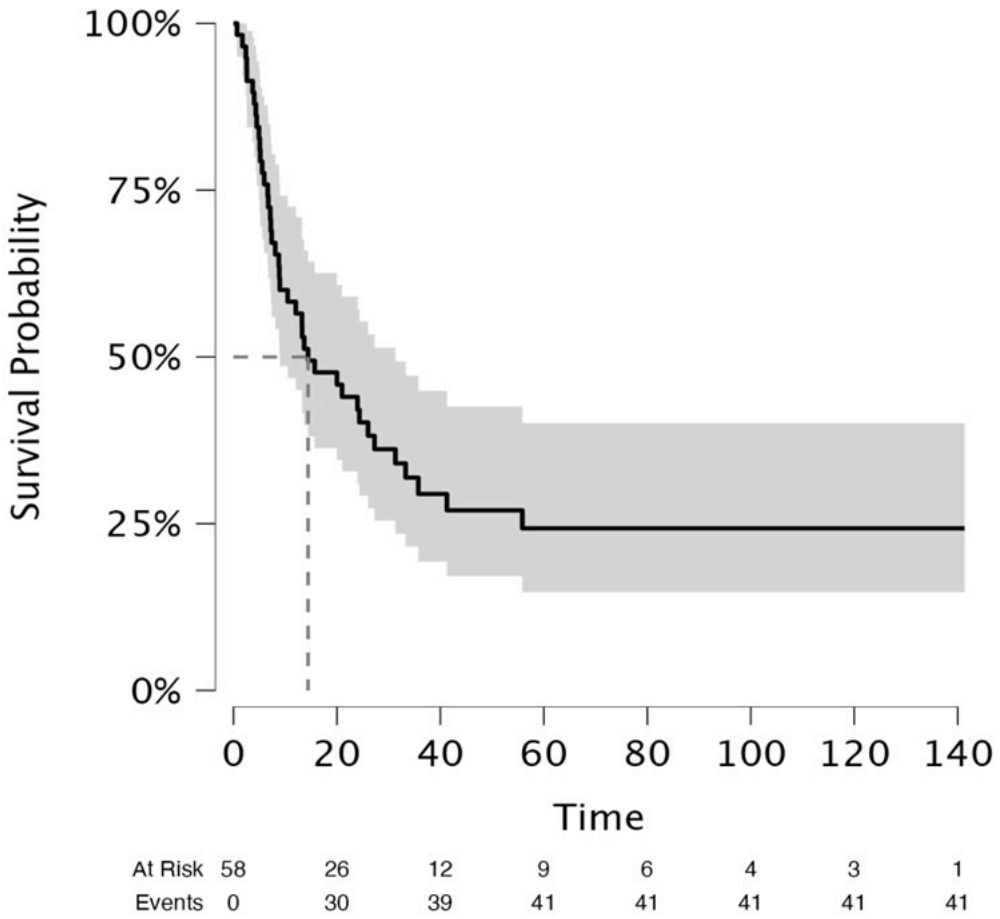


Figure 2

Kaplan–Meier estimates of overall survival among women with malignant ovarian germ cell tumours. Overall survival was calculated from the date of histological diagnosis to death from any cause or the last documented follow-up. The median overall survival was 14.4 months, indicated by the dashed reference line in the Kaplan–Meier curve. The estimated 3-year overall survival was 29.5% (95% CI, 19.3%–44.9%).

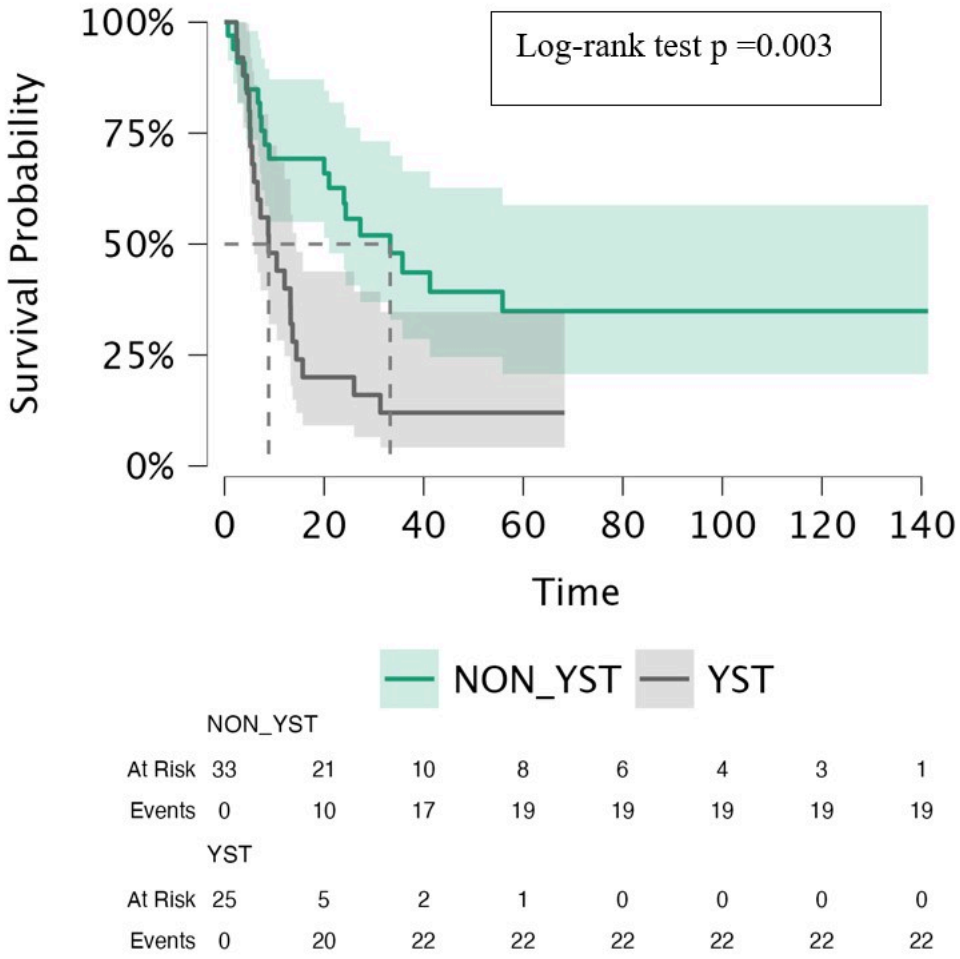


Figure 3

Kaplan–Meier estimates of overall survival according to YST status among women with malignant ovarian germ cell tumours. Patients were stratified into YST-associated (YST) and non-YST (NON_YST) malignant ovarian germ cell tumours. Survival distributions were compared using the log-rank test. Overall survival was calculated from the date of histological diagnosis to death from any cause or the last documented follow-up.

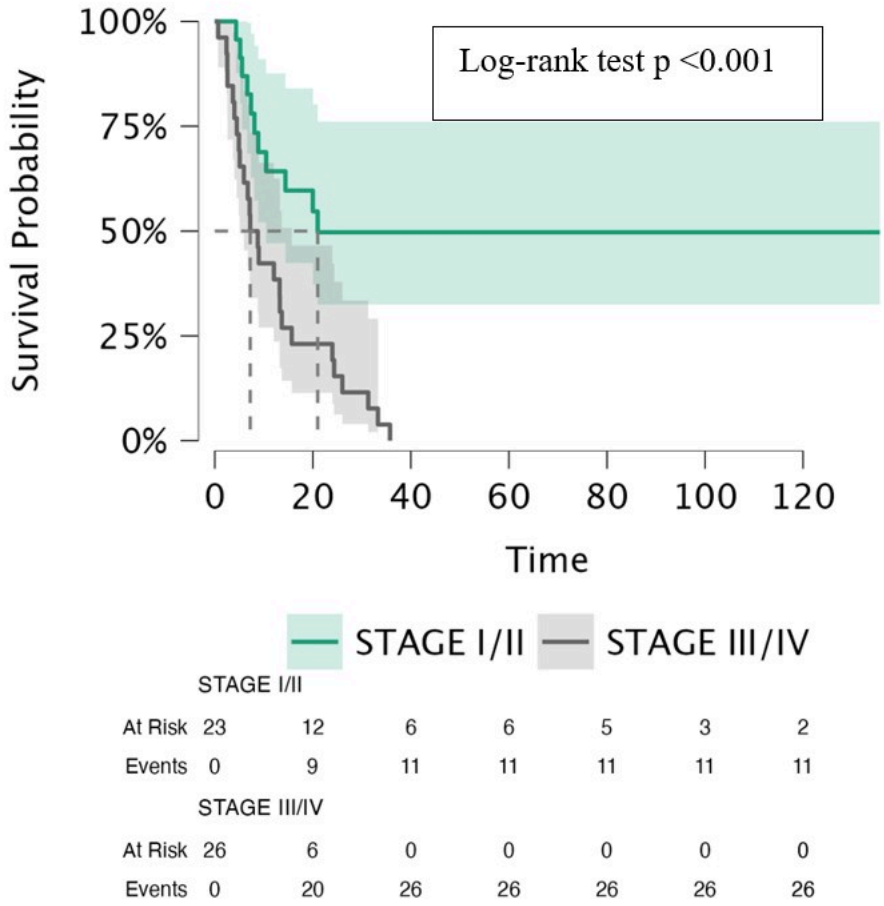


Figure 4

Kaplan–Meier estimates of overall survival according to FIGO stage among women with malignant ovarian germ cell tumours. Patients were stratified according to FIGO stage (Stage I/II vs Stage III/IV). The log-rank test was used to compare survival between groups.Overall survival was calculated from the date of histological diagnosis to death from any cause or the last documented follow-up.

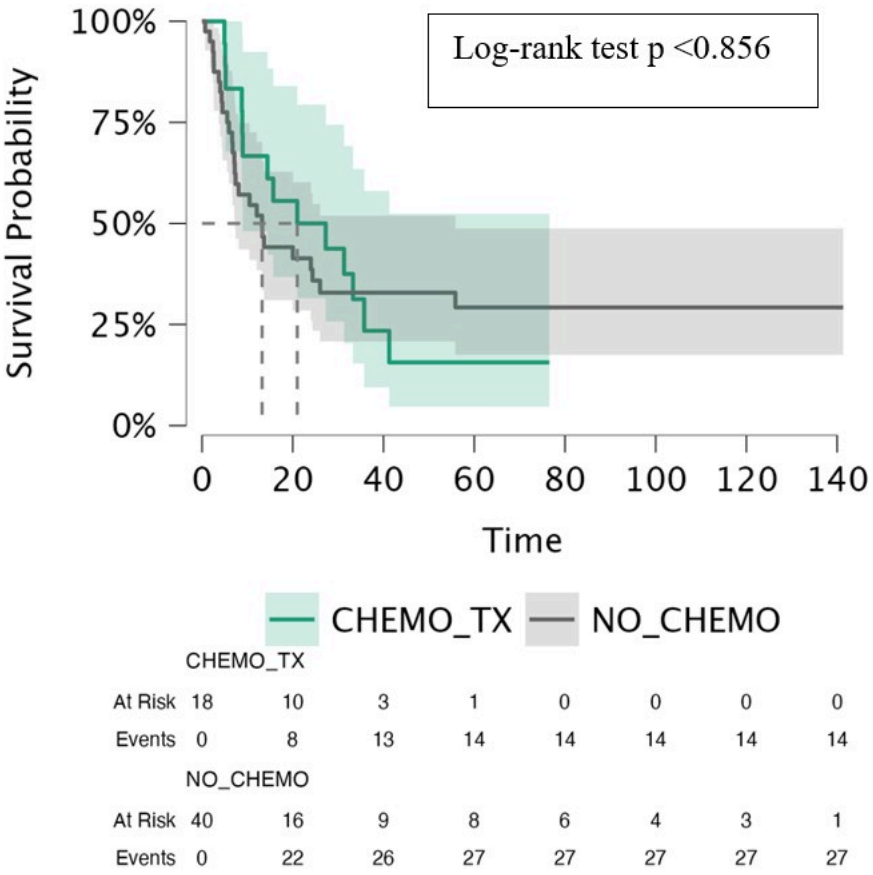


Figure 5

Kaplan–Meier estimates of overall survival according to receipt of first-line chemotherapy among women with malignant ovarian germ cell tumours. Patients were stratified according to receipt of first-line chemotherapy (CHEMO_TX) or no first-line chemotherapy (NO_CHEMO). Survival distributions were compared using the log-rank test. Overall survival was calculated from the date of histological diagnosis to death from any cause or the last documented follow-up.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- 1.mOGCTSupplementaryResults.docx