

Case Series

Ovarian adult granulosa cell tumour: A clinicopathological series

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ABSTRACT

Ovarian granulosa cell tumours (GCTs) are rare neoplasms derived from sex-cord stromal cells, constituting 2% to 5% of all ovarian cancers. They are predominantly of the adult form, which accounts for about 95% of cases and primarily affects perimenopausal and postmenopausal women, with a peak incidence at ages 50–55. These tumours are noteworthy for their hormone secretion, mainly estrogen, leading to endometrial alterations such as hyperplasia and cancer. Patients typically present with symptoms like abnormal vaginal bleeding, abdominal pain, or distension. Despite their classification as low-grade malignancies with a tendency to remain localized, they exhibit an indolent course and risk of late recurrence. This case series reviews seven instances of ovarian GCTs diagnosed at Fortis Memorial Research Institute, Gurugram, between 2015 and 2022. It highlights the variable clinical presentation and the necessity for prolonged follow-up. The cases involve women aged 37 to 62 who presented with symptoms ranging from postmenopausal bleeding to large abdominal masses. Diagnostic imaging and elevated inhibin b levels were common. Surgical interventions included hysterectomy, salpingo-oophorectomy, and omentectomy, with final histopathology confirming adult granulosa cell tumours. All patients were managed surgically with or without adjuvant treatment and have shown no disease recurrence. This series underscores the importance of recognizing the diverse presentations of adult GCTs and the critical need for long-term monitoring to manage potential recurrences effectively.

Keywords: Disease recurrence, Hysterectomy, Ovarian GCTs, Postmenopausal bleeding, Surgery

INTRODUCTION

Ovarian granulosa cell tumours (GCTs) are rare neoplasms originating from the sex-cord stromal cells of the ovary, accounting for 2% to 5% of all ovarian malignancies.^[1,2]

These tumours are classified into two histological subtypes: adult (95%) and juvenile (5%). The adult form is predominant, comprising approximately 95% of GCTs, and primarily affects women in the perimenopausal and postmenopausal age groups, with the highest incidence observed between 50 and 55 years of age.^[3] Adult granulosa cell tumours are the most common hormone-secreting ovarian malignancies, typically producing estrogen, which can cause endometrial changes, including hyperplasia and endometrial carcinoma. Common presenting symptoms include abnormal vaginal bleeding, abdominal pain, or distension.^[4,5]

Although considered low-grade malignant tumours, adult GCTs often remain localized and follow a slow-growing clinical course, with the potential for late recurrence.^[6]

This case series examines all cases of ovarian GCT diagnosed at Fortis Memorial Research Institute, Gurugram, from 2015 to 2022. It highlights the diverse clinical presentations and progression of adult-type GCTs, underscoring the importance of implementing lifelong follow-up protocols.

CASE SERIES

Case 1

A 62-year-old postmenopausal woman, para 2, live 2, presented in 2015 with postmenopausal bleeding. An endometrial biopsy revealed simple endometrial hyperplasia without atypia. Her serum inhibin B level was elevated at 639.36 pg/mL, while

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Table 1: Clinicopathological characteristics of patients with adult granulosa cell tumours

Case	Age (years)	Presentation	Tumour size (cm)	FIGO Stage	Histology / mitotic index	Endometrium	Treatment	Follow-up / outcome
1	62	Postmenopausal bleeding	5 × 4.5 × 3.5	IA	3–4/10 HPF, Ki-67 30%	Simple hyperplasia	TAH + BSO + PLND + omentectomy + peritoneal biopsies	Disease-free
2	39	Large abdominal mass	24 × 20 × 11	IA	Scattered mitoses, Ki-67 ≤1%	Benign endometrium	TAH + BSO + Omentectomy + Peritoneal biopsies	Disease-free
3	48	Vaginal bleeding	5 × 4 × 2.5	IA	4/10 HPF	Simple hyperplasia	TAH + BSO + Omentectomy + Pelvic node sampling + Peritoneal biopsies	Disease-free
4	37	Irregular cycles	8 × 7.5 × 5	IC2	8–10/10 HPF	Proliferative	Fertility-sparing surgery → later completion hysterectomy + BSO	Conceived, delivered a baby; disease-free
5	57	Postmenopausal bleeding	8 × 5.5 × 4	IC2	Ki-67 3%	Proliferative	TAH + BSO + PLND + PALND + Omentectomy	Disease-free
6	34	Pain, irregular menses	10 × 7	IA	Ki-67 20%	Hyperplasia without atypia	TAH + BSO + Omentectomy	Disease-free
7	33	Recurrent abdominal pain, mass	8 × 6 (initial); multiple recurrent deposits	Initial IA → Recurrent disease	2–4/10 HPF; ER/PR+	Not reported	Multiple cytoreductive surgeries + chemo + hormonal therapy	Recurrence; lost to follow-up in 2019

FIGO: International Federation of Gynecology and Obstetrics, TAH: Total abdominal hysterectomy; BSO: Bilateral salpingo-oophorectomy, PLND – Pelvic lymph node dissection, ER: Estrogen receptor and PR: Progesterone receptor, IA-FIGO Stage IA (tumor limited to ovary), IC2 – FIGO Stage IC2 (capsule rupture or surface involvement), HPF: High power field, Ki-67 – Ki-67 proliferation index.

other tumour markers, including CA-125, CA19-9, and carcinoembryonic antigen (CEA), were within normal limits. positron emission tomography–computed tomography (PET-CT) imaging showed a distended endometrial cavity with increased FDG uptake and an fluorodeoxyglucose (FDG)-avid heterogeneous soft tissue lesion in the left adnexal region measuring approximately 4 × 3 × 3.3 cm, with no evidence of metastatic disease.

The patient underwent total abdominal hysterectomy, bilateral salpingo-oophorectomy, bilateral pelvic lymph node dissection, omentectomy, and peritoneal biopsies. A frozen section of the left ovarian mass was reported as a granulosa cell tumour. Final histopathological analysis confirmed an adult granulosa cell tumour of the left ovary measuring 5 × 4.5 × 3.5 cm, with a mitotic count of 3–4/10 high-power fields (HPF) and a Ki-67 labelling index of 30%. The endometrium exhibited simple hyperplasia. Based on these findings, the tumour was staged as FIGO stage IA. The patient did not receive any adjuvant therapy and has remained disease-free to date. Table 1 provides the clinicopathological characteristics of patients with adult granulosa cell tumours.

Case 2

A 39-year-old female, para 1, live 1, presented in 2018 with

a large abdominal mass. Her tumour markers were raised: Ca125-404, Ca19-9- 74, and Inhibin b>1086 pg/ml, normal CEA. PET CT showed a large lobulated complex solid cystic abdominopelvic mass lesion measuring 12.8 × 17.8 × 24.3 cm with a solid component showing FDG avidity. It also showed the right iliac vein, left renal vein, and pulmonary artery thrombosis. There was no metastatic disease. Anticoagulant was started, and she was taken up for surgery after a month. She underwent exploratory laparotomy, excision of left ovarian mass with fallopian tube, frozen evaluation, which was reported as granulosa cell tumour, followed by hysterectomy, right salpingo-oophorectomy, infracolic omentectomy, and multiple peritoneal biopsies. Final histopathology was reported as an adult granulosa cell tumour of the left ovary of 24 × 20 × 11 cm showing scattered mitotic figures with a Ki67 proliferation index of ≤1%, FIGO stage IA. Endometrium showed benign nonsecretory endometrium. She did not receive adjuvant treatment, and clinical, biochemical, and radiological follow-up has been unremarkable.

Case 3

A 48-year-old female, para 3, live 1, presented in 2022 with continuous vaginal bleeding for 1.5 months. Endometrial

biopsy showed nonsecretory endometrium. The tumour markers were Ca125- 107 U/ml and inhibin b- >1050 pg/ml; the rest were normal. PET CT revealed a well-defined non-FDG avid soft tissue lesion with areas of internal necrosis in the left adnexal region measuring $4.6 \times 2.8 \times 3.3$ cm and no evidence of metastasis. She underwent exploratory laparotomy, excision of left ovarian mass with fallopian tube, frozen evaluation, which was reported as granulosa cell tumour, followed by a hysterectomy, right salpingo-oophorectomy, total omentectomy, bilateral pelvic node sampling with multiple peritoneal biopsies. Final histopathology showed an adult granulosa cell tumour of the left ovary, $\sim 5 \times 4 \times 2.5$ cm, with mitotic figures 4/10HPF, FIGO stage IA. Endometrium showed simple hyperplasia. She has been under regular follow-up and disease-free till now.

Case 4

A 37-year-old female, para1, live 1, presented in 2022 with irregular menstrual cycles for 2.5 years and an ultrasound showing a right ovarian mass. All her tumour markers, i.e., Ca125, CEA, CA19-9, lactate dehydrogenase, (LDH) Alpha-fetoprotein m (AFP), beta human chorionic gonadotropin (β -HCG), were normal except inhibin b, which was raised >1050 pg/ml. magnetic resonance imaging (MRI) of the abdomen showed a large well, well-circumscribed, solid cystic mass from the right ovary $\sim 8.3 \times 7.9 \times 5$ cm with diffusion restriction in the solid component. She underwent fertility preservation surgery- exploratory laparotomy, excision of the right ovarian mass with fallopian tube, frozen evaluation, which was reported as granulosa cell tumour, followed by total omentectomy, bilateral pelvic lymph node dissection, multiple peritoneal biopsies, and endometrial biopsy. Final histopathology revealed an adult granulosa cell tumour of the right ovary, $\sim 8 \times 7.5 \times 5$ cm with focal capsule breach, thus, FIGO stage IC2. Mitosis varied from 8-10/10HPF and immunohistochemistry (IHC) as shown in Figure 1." Endometrial biopsy showed proliferative endometrium. She conceived spontaneously and delivered a healthy baby by cesarean section in January 2023. She was taken up for completion surgery- laparoscopic evaluation, robotic-assisted type 1 hysterectomy, left salpingo-oophorectomy, and adhesiolysis. Final histopathology revealed no residual tumour. No adjuvant treatment was given. Routine follow-up has been uneventful to date.

Case 5

A 57-year-old female, para 2, live 2, presented with postmenopausal bleeding in 2021. MRI of the pelvis showed a well-defined right adnexal mass of $\sim 4.8 \times 3.4$ cm along the right lateral wall of the uterus. Broad ligament fibroid with cystic degeneration. PET CT showed a large, well-defined, solid cystic mass from the right adnexa, $\sim 7.5 \times 6.9$ cm with mild FDG uptake and no evidence of metastasis. Preoperative

serum CA125 was 20.7 U/L. She underwent exploratory laparotomy, excision of the right ovarian mass with fallopian tube, frozen evaluation, which was reported as granulosa cell tumour, followed by a hysterectomy, left salpingo-oophorectomy, bilateral pelvic lymph node dissection, para-aortic lymph node sampling, total omentectomy, and multiple peritoneal biopsies. Final histopathology reported a right ovarian adult granulosa cell tumour $\sim 8 \times 5.5 \times 4$ cm with capsule breach, Ki67- 3%; thus, FIGO stage IC2 and endometrium showed proliferative endometrium. She was kept on observation and regular follow-up and is disease-free.

Case 6

A 34-year-old female, para 2, live 2, was referred to us after eight months of surgery, i.e., total abdominal hysterectomy, bilateral salpingo-oophorectomy, frozen section of left ovarian mass, and omental biopsy done. She had initial complaints of pain in her lower abdomen and irregular menses for 1 year. Intraoperative findings were reported as a normal uterus and right adnexa. Left ovarian cystic mass $\sim 10 \times 7$ cm and no lymphadenopathy and metastasis. The frozen section was reported as a malignant tumour. Histopathology showed an adult granulosa cell tumour, left ovary, Ki67-20%, FIGO stage IA, and endometrium showed endometrial hyperplasia without atypia. Preoperative serum CA125 was 1.72 U/L. Blocks were reviewed and reported as the same. PET-CT was done after one year of surgery and showed no evidence of disease. The patient is under regular follow-up and is disease-free clinically, radiologically, and biochemically.

Case 7

A 33-year-old female, para 1, live 1, had a history of laparoscopic right oophorectomy and omental biopsy done outside in 2008, given abdominal pain with clinical suspicion of the dermoid cyst (USG- right adnexal cyst $\sim 8 \times 6$ cm, CA125-14.7). She also had a history of ovarian drilling for infertility in 2002. Histopathology was reported as adult granulosa cell tumour, mitotic figures 2-3/10 HPF with intact capsule, FIGO stage IA. She was asymptomatic for 8 years until, in 2016, she presented to another institute with complaints of lower abdominal pain and menstrual irregularity for 1 year. Tumour markers were: CA125- 37.1 U/mL, Inhibin A- 8.6 pg/mL and CT abdomen showed peritoneal deposits, largest in the subphrenic space $\sim 4.7 \times 6 \times 9$ cm. She underwent total abdominal hysterectomy, left salpingo-oophorectomy, omentectomy, and pelvic cystic mass excision on 31.05.2016. The cystic mass on the frozen section was reported as a hemorrhagic cyst. Final histopathology was reported as left ovarian adult granulosa cell tumour $\sim 3.5 \times 3 \times 1$ cm, mitosis 2-4/10 HPF, pelvic cystic mass- metastatic

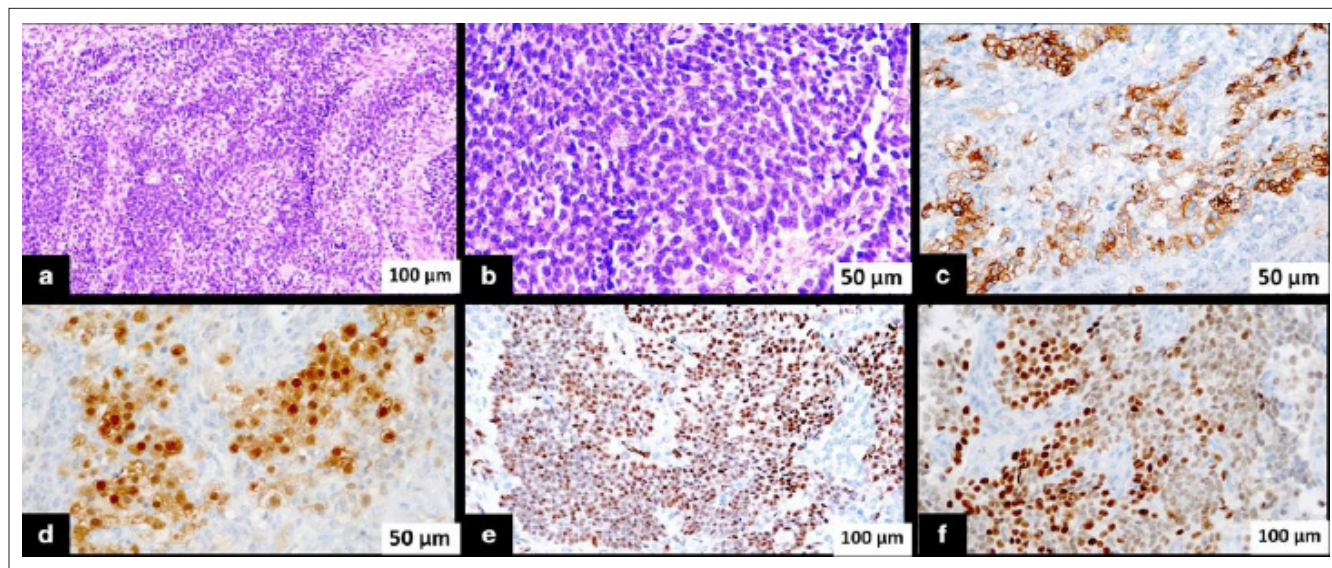


Figure 1: (A) Hematoxylin and Eosin (H and E) stain (100x) shows tumour cells arranged in a trabecular and microfollicular pattern. (B) Hematoxylin and eosin (H and E) stain - higher magnification 200x shows individual tumour cells have scant to moderate indistinct cytoplasm with round to oval nuclei and small nucleoli. Occasional tumour cells show nuclear grooving. Immunohistochemistry for (C) Inhibin, (D) Calretinin, (E) WT1 and (F) SF1 are positive.

deposit of tumour cells, and omentum also showed metastatic tumour deposits. She was referred to us for completion surgery. She was taken up for cytoreductive surgery (completion surgery) on 27.06.2016 with the following intraoperative findings: omental adhesions with anterior abdominal wall, bowel loops adherent to pelvic peritoneum, inter bowel loop adhesions, right retrocolic mass (4-6 cm) adherent to paracolic gutter peritoneum, mass in colonic mesentery at splenic flexure ~5 × 6 cm, nodular mass on ligamentum teres ~2cm, lesser omentum 1-1.5 cm, multiple nodular deposits ~3-3.5 cm on greater omentum & small bowel mesentery, disease deposit over appendix ~0.5-1cm, enlarged pelvic lymph nodes ~2 cm and para-aortic lymph nodes ~1.5 cm. Cytoreductive surgery was done to no gross residual disease. Final histopathology showed tumour cells compatible with granulosa cell tumour over omentum, appendix, small bowel mesenteric deposit, ligamentum teres, bowel adhesion, right paracolic gutter deposit, and left a colonic mesenteric deposit. All the nodes were negative. Immunohistochemistry showed ER-60% and PR- 70% positivity. She received 6 cycles of adjuvant chemotherapy (carboplatin and paclitaxel). She recurred while on chemotherapy and was started on tablet Tamoxifen 20 mg. PET CT showed scalloping disease deposits around the liver. Secondary cytoreductive surgery with excision of a six cm-sized tumour deposit from the right posteroinferior liver surface adherent to the right diaphragm was done. Histopathology again showed granulosa cell tumour, ER/PR- 60%. Tamoxifen was increased to 40 mg and continued for 1 year, then she was shifted to letrozole 2.5 mg. She followed up regularly till 2019 with clinically no disease and has been lost to follow-up since then. Table 2

provides a summary of clinical and pathological features of adult granulosa cell tumours in the present series.

Table 2: Summary of clinical and pathological features of adult granulosa cell tumours in the present series

Parameter	Present study (n = 7)
Median age (range)	39 years (33–62)
Menopausal status	Premenopausal: 5 (71.4%); Postmenopausal: 2 (28.6%)
Median tumour size (range)	8 cm (3.5–24 cm)
FIGO stage	Stage IA: 4 (57.1%); Stage IC2: 2 (28.6%); Advanced/recurrent: 1 (14.3%)
Endometrial changes	Hyperplasia/proliferative in 4 (57.1%)
Median mitotic index	3–4/10 HPF (range: 2–10)
Recurrence	1 (14.3%)
Median follow-up duration	4 years (range: 1–8 years)
Disease status at last follow-up	Disease-free: 6; lost to follow-up: 1

FIGO: International Federation of Gynecology and Obstetrics, HPF: High power field

DISCUSSION

Granulosa cell tumours (GCTs) are the most common type of sex-cord stromal tumours, making up 2–5% of all ovarian neoplasms.^[1,2] These tumours originate from granulosa cells in late preovulatory follicles and exhibit similar biochemical, morphological, and hormonal features, such as the production of inhibin, estrogen, and Müllerian inhibiting substances.^[1,7]

These hormones contribute to clinical symptoms and are important markers for monitoring disease progression and treatment response.

Patterns observed in this case series reveal distinct presentations. Women of reproductive age frequently exhibit menstrual irregularities alongside adnexal cysts, as noted in cases 2, 3, 4, and 6. Conversely, postmenopausal bleeding was a prominent symptom in menopausal patients, as seen in cases 1 and 5, likely due to estrogen production by the tumour, which induces proliferative or hyperplastic changes in the endometrium. Unopposed estrogen can lead to Type 1 endometrial carcinoma. Although rare, some GCTs may secrete androgens, causing hyperandrogenic symptoms such as clitoromegaly or hirsutism, but no such cases were reported in this series.^[8]

Positive immunohistochemical staining for inhibin is a key diagnostic feature of GCTs. As demonstrated in these cases, adult-type GCTs display varied histological patterns, with the classic "coffee-bean" nuclear grooves and Call-Exner bodies seen in the microfollicular pattern. Although GCTs typically have low mitotic activity, a mitotic rate of ≥ 4 MF/10 HPF is associated with poorer outcomes and increased recurrence risk.^[9] For instance, case 4 in this series showed a high mitotic index (8–10 MF/10 HPF), placing it at greater risk of recurrence.

Research indicates 70–97% of adult GCTs harbour a specific somatic FOXL2 c.402C>G point mutation.^[10–12]

This mutation makes FOXL2 a highly sensitive and specific diagnostic marker for distinguishing adult GCTs from juvenile types and other sex-cord stromal tumours.^[13] Furthermore, FOXL2 expression may have prognostic significance, as higher expression levels are linked to poorer outcomes and reduced disease-free survival.^[14]

Surgical management

GCTs are classified as neoplasms with low malignant potential, with about 80% detected at FIGO stage I. This early detection contributes to a five-year survival rate exceeding 90%.^[15] In young patients with early-stage disease who desire fertility preservation, fertility-sparing surgery (unilateral salpingo-oophorectomy with staging) is an option, as seen in case 4.^[16] However, completion surgery (hysterectomy and contralateral oophorectomy) is advised later in life. Endometrial biopsy should be performed in patients undergoing fertility-sparing surgery to exclude hyperplasia or carcinoma.^[17] A retrospective study of FIGO stage I GCTs found endometrial alterations in 47% of cases, emphasizing the need for thorough endometrial assessment during initial treatment.^[18] This series identified Endometrial hyperplasia in cases 1, 3, and 6.

Recurrence occurs in about 25% of GCT patients, often years after initial treatment, as illustrated by case 7, where recurrence

occurred eight years post-surgery.^[19] Secondary cytoreductive surgery is the preferred treatment for recurrence.^[20] Evidence from studies by Karalok *et al.* and Mangili *et al.* highlights the importance of complete tumour resection in both primary and secondary surgeries to minimize recurrence risk.^[17,18]

Chemotherapy and hormonal therapy

Chemotherapy regimens such as bleomycin, etoposide, cisplatin (BEP), or carboplatin with paclitaxel are commonly used for advanced or recurrent diseases.^[21,22] National Comprehensive Cancer Network (NCCN) guidelines recommend taxane-based chemotherapy for recurrent tumours.^[23] However, studies like the MITO-9 trial indicate a limited impact of chemotherapy on overall survival despite improved recurrence-free survival.^[18]

Hormonal therapy offers another avenue for managing recurrent or advanced GCTs due to their hormone receptor expression. Aromatase inhibitors, such as letrozole, anastrozole, and exemestane, have effectively inhibited tumour growth.^[24] Case 7 in this series was treated with hormonal therapy. Targeted therapies, such as tyrosine kinase inhibitors (e.g., imatinib), are also emerging as potential treatments, with promising results in stabilizing recurrent tumours.

Our findings are consistent with prior reports. For instance, Khosla *et al.* (2014) reported a median age of 44 years, stage I disease in 77%, and recurrence in 23% of patients.^[16] Similarly, Mangili *et al.* (2013) observed recurrence in 20–25% of cases with a median follow-up of 7 years.^[18] In comparison, our study showed a younger median age (39 years), stage I disease in 86%, and recurrence in 14%, highlighting the variable presentation and relatively favourable outcomes in this Indian cohort.

Although the number of patients in this series is small, it adds to the limited body of literature from the Indian subcontinent. Regional experiences are essential, as tumour biology, treatment choices, and follow-up adherence often vary across populations. Our study highlights unique aspects, including fertility-sparing surgery with successful pregnancy (Case 4), a very late recurrence after 8 years (Case 7), and a high mitotic index in one patient (Case 4), all of which carry clinical significance. Therefore, this series emphasizes the importance of tailoring treatment, recognizing diverse presentations, and ensuring long-term follow-up in patients with adult granulosa cell tumours.

Patients consent: Written informed consent for participation and use of anonymized clinical data was obtained from all patients at the time of diagnosis or treatment. For the patient who was later lost to follow-up, consent had been taken at the time of initial surgery and treatment; however, subsequent follow-up data.

CONCLUSION

Surgical intervention remains the cornerstone of treatment for both primary and recurrent GCTs. Adjuvant chemotherapy is reserved for advanced or unresectable cases due to its toxicity and limited benefits. Hormonal therapy shows promise but requires further evaluation. Advances in identifying therapeutic targets and developing targeted therapies are essential to improve outcomes, particularly for aggressive and recurrent tumours. Given the risk of late recurrence, extended surveillance with clinical, biochemical, and imaging assessments is crucial after initial treatment.

Author contributions: TS: Conceived and designed the study, supervised data collection, and critically revised the manuscript for important intellectual content; SD: Reviewed histopathological slides, performed and interpreted immunohistochemical analyses, contributed to data interpretation, and critically revised the manuscript; SM: was involved in patient recruitment, data acquisition, and drafting of the initial manuscript; MS: Assisted in literature review, data verification, and preparation of tables and figures; RRB: Contributed to manuscript editing and final approval of the version to be published.

All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to accuracy or integrity of any part of the work are appropriately investigated and resolved.

Ethical approval: Institutional Review Board approval is not required.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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