South Afr J Gynaecol Oncol. 2026;18:1-6
https://doi.org/10.36303/SAJGO.410

South Afr J Gynaecol Oncol
ISSN 2074-2835 EISSN 2220-105X

ORIGINAL RESEARCH

High-risk gestational trophoblastic neoplasia in a middle-income setting: a retrospective review of management and outcomes at a single tertiary institution in South Africa

SA Camroodien,[1] N Fakie,[2] N Mbatani[1]

[1] Department of Gynaecology Oncology, Groote Schuur Hospital, South Africa
[2] Department of Radiation Oncology, Groote Schuur Hospital, South Africa
Corresponding author, email: drcam84@gmail.com

Background: In South Africa, the incidence of high-risk gestational trophoblastic neoplasia (GTN) and the impact of chemotherapy outcomes are largely unknown. This study aimed to evaluate treatment outcomes and identify independent prognostic factors associated with chemotherapy failure in women with high-risk GTN treated at a single tertiary institution in South Africa.

Methods: This retrospective descriptive study reviewed all women with high-risk GTN managed at Groote Schuur Hospital (GSH), Cape Town, from January 2008 to December 2022. Clinical characteristics, treatment regimens, and outcomes were analysed. Univariate logistic regression was used to assess factors associated with mortality.

Results: Of the 95 women treated for GTN during the study period, 34 met the inclusion criteria for high-risk disease. The median age was 33 years, and 35.3% had ultra-high-risk scores (≥ 12). Most patients (76.5%) received EMA/CO (etoposide, methotrexate, and actinomycin D alternating weekly with cyclophosphamide and vincristine) as first-line chemotherapy, with a complete sustained remission rate of 88.2%. Mortality occurred in 11.8% of patients (two early and two late deaths), with ultra-high-risk disease accounting for 50% of all deaths. Risk factors associated with mortality included non-molar antecedent pregnancy, high metastatic burden, stage IV disease, and World Health Organization (WHO) scores ≥ 12. Paclitaxel and etoposide alternating biweekly with paclitaxel and cisplatin (TE/TP) was used in select patients, with favourable remission rates and manageable toxicity. The study noted delays in diagnosis and treatment initiation in over one-third of patients, likely due to socio-economic barriers and health system limitations.

Conclusion: High-risk GTN can be effectively treated in a resource-limited setting, with remission rates comparable to international standards. However, early deaths and treatment-related toxicity remain concerning. Identifying patients at risk for poor outcomes – particularly those with ultra-high-risk disease – can guide induction regimens and support tailored management strategies. This is our institution's first analysis of high-risk GTN, focusing specifically on treatment patterns, remission, and mortality among women with high-risk GTN at a tertiary referral centre in South Africa, highlighting the need for improved diagnostic timelines and expanded treatment capacity.

Keywords: high-risk gestational trophoblastic neoplasia, chemotherapy outcomes, prognostic factors, resource-limited setting, South Africa

Introduction

Gestational trophoblastic disease (GTD) is a heterogeneous group of rare tumours characterised by the abnormal proliferation of trophoblastic tissue. The malignant tumours, specifically, are commonly referred to as GTN.[1] Quantification of human chorionic gonadotropin (hCG) reflects the total viable tumour burden and is relevant for GTN diagnosis and treatment monitoring. Before initiating chemotherapy, GTN staging is critical. Two systems are used to categorise GTN: the International Federation of Gynaecology and Obstetrics (FIGO) anatomical stage and the WHO prognostic score. They both correlate with clinical outcomes and identify patients at risk for treatment failure. Currently, the 2000 FIGO staging system is the standard classification and is anatomically based. Patients are also assigned a modified WHO prognostic index score based on prognostic factors.[2-4]

High-risk disease (prognostic score ≥ 7) is typically treated with multi-agent chemotherapy.[5] Ultra-high-risk disease includes extensive liver, lung, or brain metastases, a WHO score > 12, major bleeding, and an hCG level > 1 000 000 IU/L.[6] Early deaths occur within four weeks of treatment, mostly due to respiratory compromise resulting from haemorrhage within the thorax, intraperitoneal or intracranial spaces, and secondary to the tumour burden and resultant rapid tumour destruction that occurs with chemotherapy.[6]

EMA/CO was first described by the Charing Cross Group in 1986. Over time, this regimen has become the most common and preferred first-line chemotherapy. Etoposide, methotrexate, and actinomycin D, with etoposide and cisplatin (EMA/EP), was initially developed as a second-line regimen and is often reserved for first-line therapy in ultra-high-risk GTN due to greater relative toxicity. The Charing Cross Group reported the use of induction low-dose etoposide (100 mg/m^2) and cisplatin (20 mg/m^2) on

days one and two every seven days, in select patients with a high tumour burden. This almost eliminated the early death rate from respiratory compromise and haemorrhage from 7.8% to 0.7%. They also report a 94% remission rate with EMA/CO, achieved by carefully excluding non-gestational tumours through genetic analysis.[7]

However, EMA/CO and EMA/EP are costly and toxic regimens, requiring 1–2 nights of hospitalisation per cycle, which contributes to financial and psychosocial costs for patients. High-risk GTN is responsive to a variety of multi-agent chemotherapies. The efficacy of multiple regimens is also an important consideration, as offering EMA/CO or EMA/EP to patients may not be accessible or practically feasible in all cases worldwide, given differences in available resources.[5]

The overall survival for GTN is high. EMA/CO results in complete response rates (71–78%) and has long-term survival rates (85–94%).[3] However, 14% of high-risk patients who receive EMA/CO chemotherapy will require further treatment. Various salvage chemotherapy regimens have been used, including EMA/EP. The response rates for this regimen are high, with 75% of EMA/CO failures (excluding placental site trophoblastic tumours [PSTT]), achieving long-term survival with or without additional surgery.[8] However, toxicity is a major problem.

The potential value of the TE/TP regimen is well documented, especially in pretreated patients with GTN. The survival in this group was not surprisingly low at 45%, because most patients received two or more prior therapies or already failed a platinum-based treatment (EMA/EP or bleomycin, etoposide, cisplatin [BEP] chemotherapy). Survival in patients not previously receiving platinum-based therapy but who had failed EMA/CO was much better at 70%. Moreover, survival with TE/TP was also good (75%) in patients needing to change treatment because of toxicity to their previous therapy, which included EMA/EP. The toxicity profile of TE/TP appears less problematic. Thus, grade 3 or 4 neutropoenia and thrombocytopaenia occurred less frequently in patients receiving TE/TP compared with EMA/EP. Moreover, none of the patients receiving TE/TP experienced neutropoenic sepsis, and none required dose delays.[8]

Despite the success of primary therapy with EMA/CO, roughly 30–40% of women with high-risk GTN will have an incomplete response to first-line therapy or will relapse from remission and require additional multi-agent chemotherapy, with or without other treatment modalities. Risk factors for resistant or relapse disease in most studies seem related to a large initial tumour burden, inadequate primary therapy, and patients who defaulted on potential treatments or were noncompliant in follow-up.[9]

In South Africa, the frequency of high-risk GTN is unknown. There are no institutional studies on prognostic factors of treatment response. Although previous studies by South African institutions have reported on GTD, they did not focus on high-risk GTN.[6,10-13] In addition, the mortality rates (up to 40%) among high-risk GTN patients from developing countries are high and frequently associated with metastases, haemorrhagic complications, septic shock, and disease progression.[14] In this scenario, social and economic factors and barriers to healthcare may lead to patient presentation at advanced disease stages, with death occurring within the first weeks of treatment.[14]

Because GTN is potentially curable and affects young women of reproductive age, it is extremely important to identify prognostic factors to provide effective treatment and a greater chance of survival.[14] This study aimed to evaluate the response to treatment in women with high-risk GTN who were treated at a single tertiary institution in South Africa, and identify independent risk factors for chemotherapy treatment failure.

Materials and methods

This was a retrospective descriptive study of all women with high-risk GTN referred to our tertiary gynaecological oncology unit, Department of Obstetrics and Gynaecology, at GSH in Cape Town, Western Cape Province, South Africa, from the referring hospitals in the Metro West from January 2008 to December 2022.

Inclusion criteria were patients registered and followed up at the combined gynaecological oncology and radiation oncology clinic at GSH with a WHO prognostic score ≥ 7. We excluded all patients with WHO prognostic scores < 7, PSTT, epithelioid

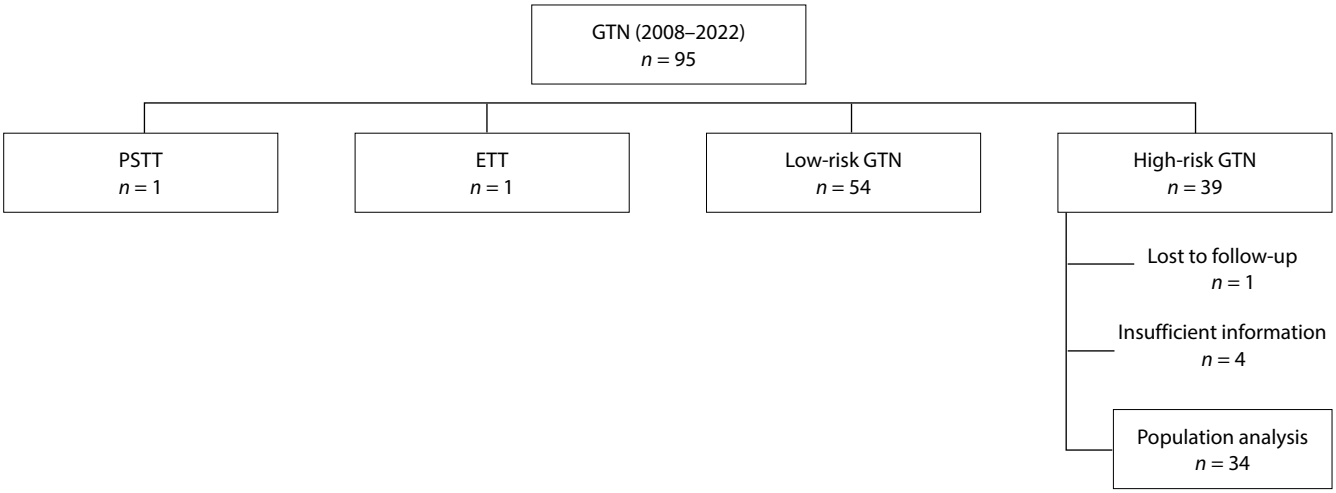

Figure 1: Selection of patients with high-risk gestational trophoblastic neoplasia
ETT – epithelioid trophoblastic tumour, GTN – gestational trophoblastic neoplasia, PSTT – placental site trophoblastic tumour

trophoblastic tumours, and patients lost to follow-up or whose files had insufficient or incomplete clinical information.

Statistical analysis

Continuous variables were summarised as median (range) and categorical variables as count (per cent). Univariate logistic regression was performed to determine factors associated with death. Odds ratios (OR) were reported as measures of association, along with the corresponding 95% confidence intervals (CI). Statistical significance was set at $p < 0.05$. Stata 18 (StataCorp, College Station, United States) was used for data analysis.

Results

Of the 95 women with GTN treated between 2008 and 2022 at our institution, 39 (41%) had high-risk GTN. A final study population of 34 patients was identified (Figure 1). The patients' median age at diagnosis was 33 years (range 14–59); 5.9% were adolescents, and 29.4% were aged ≥ 40 years. The median parity was 1 (range 0 – 8). Index pregnancy type was complete mole in 23.5%, term pregnancy in 41.1%, and spontaneous abortion in 26.5% of women. The median pretreatment hCG level was 153 858 IU/L (25th and 75th percentiles: 2750 – 3 264 390 IU/L). At diagnosis, pulmonary metastasis was observed in 58.8%, liver/gastrointestinal tract metastasis was seen in 14.7%, and brain metastasis was found in 8.8% of patients.

According to the 2000 FIGO staging system, 14.7% of the high-risk GTN cases were classified as stage I, 2.9% as stage II, 58.8% as stage III, and 23.5% as stage IV. The median prognostic WHO score was 10 (range 7–19), with 35.3% of women having ultra-high-risk scores (≥ 12). The interval between index pregnancy and treatment initiation was ≥ 12 months in 38.2% of cases (Table I).

First-line multi-agent chemotherapy consisted mainly of non-platinum-based regimens (76.5%), principally EMA/CO, followed by platinum-based regimens (23.5%) (Table II). Two patients (5.9%) received EP (etoposide and cisplatin) induction (1–3 courses) due to the large extent of the disease at presentation. No patients received EMA/EP, BEP, or methotrexate and actinomycin D (MAC).

A total of four adjuvant surgeries were performed in 11.8% of patients: hysterectomy ($n = 2$) and adnexectomy ($n = 2$). Adjuvant radiotherapy was administered to three patients (8.8%): whole-brain radiation at 24 Gy in 2 Gy fractions ($n = 2$) and spine radiation at 16 Gy in 2 Gy fractions ($n = 1$).

Complete sustained remission was achieved in 88.2% of patients, while death occurred in 11.8% (two early and two late deaths), with a median follow-up of three years. Patients with WHO scores ≥ 12 had a mortality rate of 16.7% and accounted for 50% of the total deaths in the cohort. One early death was associated with ultra-high-risk GTN, and in one patient with a FIGO/WHO score of 7–11. Early death was associated with heavy disease burden, indicated by their high initial hCG values, and both were characterised by non-molar antecedent pregnancies. Between 2008 and 2022, there were 12 patients with ultra-high-risk GTN and one early death. Late deaths were caused by chemotherapy

Table I: Clinical characteristics of 34 South African high-risk gestational trophoblastic neoplasia patients

Variable	*n*	% or range
Age, median (minimum to maximum)	33 (14–59)	
Age (years)		
< 20	2	5.9
20–39	22	64.7
≥ 40	10	29.4
Parity, median (range)	1 (0–8)	
Antecedent pregnancy		
Complete mole	8	23.5
Term pregnancy	14	41.1
Spontaneous abortion	9	26.5
Partial mole	1	2.9
Ectopic	2	5.9
Pretreatment hCG (IU/L), median (IQR)	153 858 (2 750–3 264 390)	
Metastatic disease at diagnosis		
None	6	17.6
Lung	20	58.8
Liver/gastrointestinal	5	14.7
Brain	3	8.8
Metastases (*n*)		
0	3	8.8
1–4	10	29.4
5–8	8	23.5
> 8	13	38.2
Largest tumour, including uterus (cm)		
< 3	5	14.7
3–5	7	20.6
> 5	22	64.7
FIGO stage (median)	3 (1–4)	
FIGO stage		
I	5	14.7
II	1	2.9
III	20	58.8
IV	8	23.5
WHO score (median)	10 (7–19)	
WHO score		
< 12	22	64.7
≥ 12	12	35.3
Interval from antecedent pregnancy to chemotherapy (months)		
< 4	13	38.2
4–6	3	8.8
7–11	5	14.7
≥ 12	13	38.2
HIV status		
Positive	13	38.2
Negative	21	61.8

FIGO – International Federation of Gynaecology and Obstetrics, hCG – human chorionic gonadotropin, HIV – human immunodeficiency virus, IQR – interquartile range (25th to 75th percentile)

Table II: Treatment type and outcomes for high-risk gestational trophoblastic neoplasia in 34 South African patients

Variable	*n*	%
First-line multi-agent chemotherapy		
EMA/CO	26	76.5
Paclitaxel / Etoposide alternating with Paclitaxel / Cisplatin	8	23.5
Chemotherapy induction (EP)	2	5.9
Number of cycles of first-line chemotherapy (median)	4 (1–8)	
Regimens, *n* (median)	1 (1–4)	
Prior chemotherapy		
Yes	1	2.9
No	33	97.1
Second-line chemotherapy		
Yes	14	41.1
No	20	58.8
Primary hysterectomy		
Yes	4	11.8
No	30	88.2
Adjuvant surgery		
Yes	4	11.8
No	30	88.2
Adjuvant radiotherapy		
Yes	3	8.8
No	31	91.2
Treatment outcome		
Complete sustained remission	30	88.2
Death	4	11.8
Death in women with WHO score ≥ 12	2/12	16.7
Relapse		
Yes	2	5.9
No	32	94.1

EMA/CO – etoposide, methotrexate, actinomycin D alternating weekly with cyclophosphamide and vincristine, EP – etoposide and cisplatin, TE/TP – paclitaxel and etoposide alternating with paclitaxel and cisplatin, WHO – World Health Organization

complications (two cases of bone marrow suppression/neutropoenic sepsis).

Univariate logistic regression was used to assess factors strongly associated with the risk of death. These included non-molar antecedent pregnancy (OR 1.09, 95% CI 0.10 to 12.07), number of metastases ≥ 8 (OR 4.0, 95% CI 0.46 to 34.49), presence of liver or brain metastasis (OR 4.0, 95% CI 0.46 to 34.49), FIGO stage IV (OR 4.0, 95% CI 0.46 to 34.49), and an ultra-high-risk prognostic risk score (OR 2.0, 95% CI 0.24 to 16.36). The odds of death for patients with WHO scores ≥ 12 were double the odds of those with WHO scores < 12 ($p = 0.05$). The odds of death increased non-significantly by 17% for each additional unit increase in the WHO score ($p = 0.25$). Although the odds of death seem higher among women with WHO scores ≥ 12 (OR 2.0), this finding is based on only four deaths and should be interpreted cautiously.

Discussion

This study shows that our trophoblastic disease centre in South Africa achieved a high remission rate in high-risk GTN. It is noteworthy that this is the first such study in our institution and shows what can be accomplished in treating high-risk GTN in trophoblastic centres in the developing world. In our population, death was associated with non-molar antecedent pregnancy, greater metastatic burden, WHO score ≥ 12, and FIGO stage IV disease. Women with a FIGO prognostic score ≥ 12 accounted for 35.3% of our patients. In the literature, the reported proportion of these cases ranges from 14.3% to 21%.[7,15] Ultra-high-risk disease, which is associated with poor survival, was only associated with one early and one late death in our study.[15-18]

The proportion of high-risk GTN (41%) in this cohort was higher than rates in developed countries (2.7–6.3%), though lower than those in other developing countries (58–74%).[19-22] The rate of non-molar antecedent pregnancy among our patients was as high as in developed countries.[7,23] Regression analysis showed the odds of death non-significantly increased by 9% for non-molar antecedent pregnancy ($p = 0.94$), supporting the previous reports of it being associated with an increased risk for treatment failure, with significantly lower cure rates of GTN following term pregnancy, abortion, or ectopic pregnancy.

Consistent with other studies in developing countries, the interval between the antecedent pregnancy and treatment initiation was > 12 months in 38.2% of our patients.[16,21] A longer interval from antecedent pregnancy is associated with a higher disease burden and lower survival rates.[15,24,25] A long symptom-free period may delay diagnosis. Moreover, even when symptoms are present, delays in diagnosis may occur due to

Table III: Univariate logistic regression analysis of clinical factors associated with death in 34 South African patients

Variable	OR	95% CI		*p*-value
Parity	0.37	0.09	1.47	0.16
Non-molar pregnancy	1.09	0.10	12.07	0.94
Pretreatment hCG (IU/L)	1.00	1.00	1.00	0.05*
Number of metastases ≥ 8	4.00	0.46	34.49	0.20
Liver or brain metastasis	4.00	0.46	34.49	0.20
FIGO stage IV	4.00	0.46	34.49	0.20
WHO score > 6	1.17	0.89	1.54	0.25
WHO score ≥ 12	2.00	0.24	16.36	0.05*
Interval between antecedent pregnancy and beginning chemotherapy > 12 (months)	0.50	0.05	5.39	0.57

* $p < 0.05$

CI – confidence interval, FIGO – International Federation of Gynaecology and Obstetrics, hCG – human chorionic gonadotropin, OR – odds ratio, WHO – World Health Organization

social and economic conditions, limited access to medical care, and physicians' unfamiliarity with GTN.

EP induction chemotherapy was used in only 5.9% of patients. EP induction chemotherapy was introduced in 1995, when the experience of the Charing Cross Gestational Trophoblastic Disease Centre was reported by Alifrangis et al.[7] They demonstrated that for women with a high disease burden at presentation, EP induction chemotherapy significantly improved survival and reduced early deaths from 7.2% to 0.7%.[7] In our study, the small number of patients receiving EP induction precludes a meaningful evaluation of the effect of this type of treatment on the occurrence of early death. Hopefully, the continued use of EP induction will also reduce early deaths at our institution.

Regarding high-risk patients, the protocols adopted worldwide for chemotherapy effectiveness (a remission rate of 90.6%) consist of EMA/CO.[26] In our study, EMA/CO was used as first-line chemotherapy 76.5% of the time, with a remission rate of 92.3%. Notably, 53.8% of patients receiving first-line EMA/CO required second-line chemotherapy to achieve remission.

TE/TP have encouraging results in heavily pretreated patients. It appears to have tolerable toxicity, though experience remains quite limited.[3] Eight patients were treated with TE/TP as first-line multi-agent chemotherapy in our population. Reasons for TE/TP use included logistical challenges, COVID-19, and poor performance status. The most common side effect of this regimen was haematological, with 75% of patients achieving remission.

Study strengths and limitations

The major limitation of this study is its retrospective design, which may introduce selection bias. Because all patients in this study received care at a trophoblastic disease centre, this may also have introduced selection bias. The sample size was small, affecting statistical significance analysis. However, the study was the first analysis of high-risk GTN at our institution and highlighted gaps in care, enabling improvements to our treatment protocols and surveillance tools. It also contributes to local literature and may be relevant to other low- and middle-income settings.

Conclusion

This study demonstrates that high-risk GTN can be successfully managed at a tertiary care setting in South Africa, with remission rates comparable to those reported internationally. Despite the challenges posed by advanced disease at presentation, limited resources, and delayed treatment initiation, complete and sustained remission was achieved in most patients. Mortality was primarily associated with ultra-high-risk features, such as non-molar antecedent pregnancies, extensive metastases, high WHO scores, and FIGO stage IV disease. These findings underscore the importance of early diagnosis, timely referral to specialised centres, and the potential role of induction chemotherapy in select patients to reduce early mortality. As the first study of its kind from our institution, it highlights both the successes and gaps in current care, providing a foundation for improved treatment protocols, follow-up strategies, and patient education – both locally and in similar low- and middle-income settings. Further prospective, multicentre studies are needed to better define optimal management strategies for high-risk and ultra-high-risk GTN in resource-constrained environments.

Acknowledgements

The authors acknowledge Dr Moleen Zunza's contribution to data analysis and the provision of statistical results.

Conflict of interest

The authors declare no conflict of interest.

Funding source

No funding source to be declared.

Ethical approval

Ethical approval was obtained from the University of Cape Town, Faculty of Health Sciences Human Research Ethics Committee (reference: 054/2024).

ORCID

SA Camroodien https://orcid.org/0009-0001-2222-7498
N Fakie https://orcid.org/0000-0001-8356-0764
NH Mbatani https://orcid.org/0000-0001-8826-5182

References

1. Lukinovic N, Malovrh EP, Takac I, Sobocan M, Knez J. Advances in diagnostics and management of gestational trophoblastic disease. Radiol Oncol. 2022;56(4):430-9. https://doi.org/10.2478/raon-2022-0038.
2. Lurain JR. Gestational trophoblastic disease II: classification and management of gestational trophoblastic neoplasia. Am J Obstet Gynecol. 2011;204(1):11-8. https://doi.org/10.1016/j.ajog.2010.06.072.
3. Braga A, Elias KM, Horowitz NS, Berkowitz RS. Treatment of high-risk gestational trophoblastic neoplasia and chemoresistance/relapsed disease. Best Pract Res Clin Obstet Gynaecol. 2021;74:81-96. https://doi.org/10.1016/j.bpobgyn.2021.01.005.
4. FIGO Oncology Committee. FIGO staging for gestational trophoblastic neoplasia 2000. Int J Gynaecol Obstet. 2002;77(3):285-7. https://doi.org/10.1016/S0020-7292(02)00063-2.
5. Albright BB, Ellett T, Knochenhauer HE, et al. Treatments and outcomes in high-risk gestational trophoblastic neoplasia: a systematic review and meta-analysis. BJOG. 2023;130(5):443-53. https://doi.org/10.1111/1471-0528.17374.
6. Barnardt P. Managing gestational trophoblastic neoplasm (GTN) and people living with HIV (PLWH). South Afr J Gynaecol Oncol. 2019;11(1):21-4.
7. Alifrangis C, Agarwal R, Short D, et al. EMA/CO for high-risk gestational trophoblastic neoplasia: good outcomes with induction low-dose etoposide-cisplatin and genetic analysis. J Clin Oncol. 2013;31(2):280-6. https://doi.org/10.1200/JCO.2012.43.1817.
8. Wang J, Short D, Sebire NJ, et al. Salvage chemotherapy of relapsed or high-risk gestational trophoblastic neoplasia (GTN) with paclitaxel/cisplatin alternating with paclitaxel/etoposide (TP/TE). Ann Oncol. 2008;19(9):1578-83. https://doi.org/10.1093/annonc/mdn181.
9. Powles T, Savage PM, Stebbing J, et al. A comparison of patients with relapsed and chemo-refractory gestational trophoblastic neoplasia. Br J Cancer. 2007;96(5):732-7. https://doi.org/10.1038/sj.bjc.6603608.
10. Snyman LC. Gestational trophoblastic disease: an overview. South Afr J Gynaecol Oncol. 2009;1(1):32-7. https://doi.org/10.1080/20742835.2009.11441132.
11. Makhathini BS, Dreyer G, Buchmann EJ. Gestational trophoblastic disease managed at Grey's Tertiary Hospital: a five-year descriptive study. South Afr J Gynaecol Oncol. 2019;11(2):15-9. https://doi.org/10.1080/20742835.2019.1667627.
12. Tayib S, van Wijk L, Denny L. Gestational trophoblastic neoplasia and human immunodeficiency virus infection: a 10-year review. Int J Gynecol Cancer. 2011;21(9):1684-91. https://doi.org/10.1097/IGC.0b013e31822d8ffd.

13. Moodley M, Tunkyi K, Moodley J. Gestational trophoblastic syndrome: an audit of 112 patients. A South African experience. Int J Gynecol Cancer. 2003;13(2):234-9. https://doi.org/10.1046/j.1525-1438.2003.13027.x.
14. Maestá I, de Freitas Segalla Moreira M, Rezende-Filho J, et al. Outcomes in the management of high-risk gestational trophoblastic neoplasia in trophoblastic disease centers in South America. Int J Gynecol Cancer. 2020;30(9):1366-71. https://doi.org/10.1136/ijgc-2020-001237
15. Bolze P-A, Riedl C, Massardier J, et al. Mortality rate of gestational trophoblastic neoplasia with a FIGO score of ≥13. Am J Obstet Gynecol. 2016;214(3):390.e1-8. https://doi.org/10.1016/j.ajog.2015.09.083.
16. Kong Y, Yang J, Jiang F, et al. Clinical characteristics and prognosis of ultra high-risk gestational trophoblastic neoplasia patients: a retrospective cohort study. Gynecol Oncol. 2017;146(1):81-6. https://doi.org/10.1016/j.ygyno.2017.04.010.
17. Ngan HYS, Seckl MJ, Berkowitz RS, et al. Update on the diagnosis and management of gestational trophoblastic disease. Int J Gynaecol Obstet. 2018;143(Suppl 2):79-85. https://doi.org/10.1002/ijgo.12615.
18. Li J, Yue H, Wang X, Chen R, Lu X. Chemotherapy for gestational trophoblastic neoplasia patients with a FIGO score of 12 or greater: a multistudy analysis. Eur J Obstet Gynecol Reprod Biol. 2019;238:164-9. https://doi.org/10.1016/j.ejogrb.2019.05.023.
19. Sita-Lumsden A, Short D, Lindsay I, et al. Treatment outcomes for 618 women with gestational trophoblastic tumours following a molar pregnancy at the Charing Cross Hospital, 2000-2009. Br J Cancer. 2012;107(11):1810-4. https://doi.org/10.1038/bjc.2012.462.
20. Maestá I, Growdon WB, Goldstein DP, et al. Prognostic factors associated with time to hCG remission in patients with low-risk postmolar gestational trophoblastic neoplasia. Gynecol Oncol. 2013;130(2):312-6. https://doi.org/10.1016/j.ygyno.2013.05.017.
21. Nzayisenga I, Segal R, Pritchett N, et al. Gestational trophoblastic neoplasia treatment at the Butaro Cancer Center of Excellence in Rwanda. J Glob Oncol. 2016;2(6):365-74. https://doi.org/10.1200/JGO.2015.002568.
22. Hussain A, Shiekh AA, Bhat GM, Lone AR. Gestational trophoblastic neoplasia, management as per risk stratification in a developing country. Indian J Med Paediatr Oncol. 2016;37(1):28-31. https://doi.org/10.4103/0971-5851.177012.
23. Lurain JR, Singh DK, Schink JC. Management of metastatic high-risk gestational trophoblastic neoplasia: FIGO stages II-IV: risk factor score > or = 7. J Reprod Med. 2010;55(5-6):199-207.
24. Lybol C, Centen DW, Thomas CMG, et al. Fatal cases of gestational trophoblastic neoplasia over four decades in the Netherlands: a retrospective cohort study. BJOG. 2012;119(12):1465-72. https://doi.org/10.1111/j.1471-0528.2012.03480.x.
25. Neubauer NL, Strohl AE, Schink JC, Lurain JR. Fatal gestational trophoblastic neoplasia: an analysis of treatment failures at the Brewer Trophoblastic Disease Center from 1979-2012 compared to 1962-1978. Gynecol Oncol. 2015;138(2):339-42. https://doi.org/10.1016/j.ygyno.2015.05.041.
26. Babaier A, Jim B, Ghatage P. Management of EMA-CO resistant/refractory gestational trophoblastic neoplasia. Cancer Rep Rev. 2019;3. https://doi.org/10.15761/CRR.1000179.