Pregnancy-related pulmonary embolism: Clinical characteristics, management and outcomes in a South African academic hospital

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Background. Pulmonary embolism (PE) is a leading cause of death in pregnant and postpartum women.
Objective. To evaluate the clinical presentation, management and outcomes of pregnancy-related PE managed by a multidisciplinary team.
Methods. A retrospective review was conducted of pregnant and postpartum women diagnosed with PE between 2018 and 2024 at a tertiary hospital in Johannesburg, South Africa. Pretest probability scores (pregnancy-adapted YEARS and Geneva) were applied in a subgroup with D-dimers available.
Results. Seventy-seven women were included: 33 with antepartum and 44 with postpartum PE. The median (interquartile range) age was 29 (9) years, and most were of black African ethnicity. PE risk factors were present in 85% of antepartum and 96% of postpartum cases. Women with antepartum PE more frequently presented with chest pain, shortness of breath and palpitations ($p<0.05$). Pretest probability scores were assessed in a subgroup with D-dimers available. Based on the pregnancy-adapted YEARS and Geneva scores, imaging would have been required to rule out PE in 87.8% and 73.5% of cases, respectively. Computed tomography pulmonary angiography was the preferred diagnostic modality in 74.0%. Most women (97.4%) were treated as inpatients, and 57% required management in the intensive care and/or high care units. The median length of hospital stay was 14 (8) days. Low-molecular-weight heparin was the most frequently prescribed anticoagulant, with a median treatment duration of 3 (1) months. The live birth rate was 84.4%. One maternal death occurred due to sepsis, unrelated to venous thromboembolism. Antepartum/secondary postpartum major bleeding and primary postpartum major bleeding occurred in 6.5% and 3.9%, respectively.
Conclusion. Pregnancy-associated PE managed by a multidisciplinary team was associated with favourable maternal and fetal outcomes.

Keywords: pulmonary embolism, pregnancy, postpartum, maternal mortality, South Africa

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Venous thromboembolism (VTE) – comprising deep venous thrombosis (DVT) and pulmonary embolism (PE) – is a leading cause of maternal mortality globally.[1] Approximately 20 - 30% of VTE cases in pregnancy present as PE.[2] In the UK and Ireland (2020 - 2022), PE was identified as the leading direct cause of maternal death.[1] In South Africa (SA), a middle-income country, PE ranks among the top 10 causes of maternal mortality.[3] However, since PE is not a leading cause of maternal mortality in SA, awareness, education and research on pregnancy-related PE remain limited.[4] PE is associated with long-term complications, in particular VTE recurrence and chronic thromboembolic pulmonary hypertension (CTEPH).[5,6]

More than 50% of PE-related deaths are considered preventable. Risk factors for VTE during pregnancy and the postpartum period are well established. High-risk factors include a personal history of thrombosis and inherited thrombophilia,[7] for which thromboprophylaxis is recommended during pregnancy and up to 6 weeks postpartum.[8] There are also several clinical risk factors that are associated with an intermediate to low risk of VTE.[9] Despite the existence of evidence-based guidelines recommending thromboprophylaxis for high- and intermediate-risk women, adherence remains suboptimal.[8-11] A systematic review of African studies found that 25% of women at risk did not receive appropriate prophylaxis,[4] underscoring significant gaps in preventive care.

Diagnosis of VTE in pregnancy remains challenging owing to overlapping symptoms with normal pregnancy physiology, and concerns about radiation exposure. Recently, diagnostic algorithms have been developed to address this challenge.[12,13] The pregnancy-adapted YEARS algorithm,[13] which uses clinical criteria combined with D-dimer levels, has reduced the need for radiological imaging in 39% of pregnant women with suspected PE, with a *post hoc* analysis showing no VTE on follow-up (0%, 95% confidence interval (CI) 0 - 3.9).[14] Similarly, an adapted version of the Geneva score for pregnant women has been proposed,[12] incorporating seven diagnostic criteria to assess clinical probability. The risk-benefit ratio and cost-effectiveness of such algorithms could prove particularly useful in a low-resource setting; however, validation in the SA population is needed.

To address these knowledge gaps, we conducted a retrospective review of pregnancy-related PE cases managed by a multidisciplinary team at Charlotte Maxeke Johannesburg Academic Hospital (CMJAH) in Johannesburg, SA. Our aim was to describe the clinical presentation, management strategies and outcomes of affected women, thereby

contributing local data to inform best practices for managing this high-risk population in a middle-income country setting.

Methods

Study population

The study included women with a confirmed clinical and radiological diagnosis of PE in pregnancy and up to 6 weeks postpartum. Women with other identifiable causes of VTE, such as confirmed DVT alone, superficial vein thrombosis, cerebral sinus vein thrombosis and valve thrombosis, and those who delivered outside our centre, were excluded. The study was approved by the University of the Witwatersrand Human Research Ethics Committee (Medical) (ref. no. M221187).

Data collection

Women diagnosed with pregnancy-related PE at CMJAH from January 2018 to September 2024 were identified from the statistical records in the Department of Obstetrics. Data were collected from the hospital records at presentation, on follow-up and on completion of treatment. Clinical data, relevant medical and obstetric history and corresponding data on diagnosis, treatment and laboratory investigations were collected. Clinical records were verified with laboratory and radiology records. Antepartum and postpartum risk factors as per the SA Society of Thrombosis Haemostasis Guideline (modified Royal College of Obstetricians and Gynaecologists guidelines) were also collected.[8] Women were classified into low-, intermediate- and high-risk groups for PE in accordance with these recommendations.[8,10] Complications, including chronic thromboembolic pulmonary hypertension and post-thrombotic syndrome, as well as outcomes including recurrent thrombosis and bleeding, were documented. Bleeding was assessed using the International Society of Thrombosis and Haemostasis Scientific and Standardisation Subcommittee on Control of Anticoagulation bleeding assessment tool for minor, clinically relevant non-major (CRNMB) and major bleeding events related to the use of anticoagulants during pregnancy and the postpartum period.[15] All bleeding events were reviewed and confirmed by an independent adjudication committee.

Risk scores were calculated using the pregnancy-adapted YEARS algorithm and pregnancy-adapted Geneva score according to clinical signs and symptoms, relevant medical history and D-dimer levels, where available, at presentation with PE.

Computed tomography pulmonary angiography (CTPA), ventilation-perfusion (V/Q) scanning, echocardiogram and compression ultrasound for the diagnosis of VTE were collected at presentation. All PEs were objectively verified, and all diagnoses were independently reviewed by the study investigators.

The data were anonymised and coded once all relevant information was collected.

Data analysis

Statistical analysis was performed using Statistica Software version 13.2 (Statistica, USA). Normally distributed continuous data were presented as means (standard deviations (SDs)), and variables with non-Gaussian distribution as medians (interquartile ranges (IQRs)). Categorical data were presented as frequencies and percentages. Statistical significance was set at $p<0.05$. As this was a descriptive study on the study population, there was no control group.

Table 1. Baseline characteristics in pregnant and postpartum women with pulmonary embolism (*N*=77)

Characteristic	Total (*N*=77)	Pregnant (*n*=33)	Postpartum (*n*=44)
Baseline clinical characteristic			
Age at diagnosis, years, median (IQR)	29.0 (9.0)	30.0 (9.0)	29.0 (9.0)
BMI, kg/m², median (IQR)	28.6 (12.5)	28.6 (10.9)	28.6 (13.0)
Black African, *n* (%)	73 (94.8)	31 (93.9)	42 (95.5)
White, *n* (%)	1 (1.3)	1(3.0)	0 (0.0)
Coloured, *n* (%)	1 (1.3)	0 (0.0)	1 (2.3)
Indian, *n* (%)	2 (2.6)	1 (3.0)	1 (2.3)
Parity, median (IQR)	2.0 (2.0)	1.0 (2.0)	2.0 (2.0)
Gravidity, median (IQR)	3.0 (2.0)	3.0 (2.0)	2.0 (2.0)
Previous miscarriages, *n* (%)	20 (26.0)	7 (21.2)	13 (29.5)
Smoker, *n* (%)	1 (1.3)	0 (0)	1 (2.3)
Baseline laboratory characteristic			
Living with HIV, *n* (%)	20 (26.0)	10 (30.3)	10 (22.7)
CD4 count of HIV infected, × 10⁶/L, median (IQR)	327.5 (372.0)	353.5 (284.0)	296.0 (520.0)
HIV viral load <50 (copies/ml) of HIV infected, *n* (%)	8 (40)	4 (40.0)	4 (40.0)
White cell count, × 10⁹/L, median (IQR) (ref. 3.9 - 12.6)	10.4 (4.3)	8.6 (2.8)**	11.4 (6.4)**
Haemoglobin, g/dL, median (IQR)	10.3 (2.7)	10.7 (2.7)	10.3 (2.8)
Platelet count, × 10⁹/L, median (IQR)			
Pre LMWH	259.0 (128.0)	258.0 (116.0)	260.0 (155.0)
Post LMWH	307.0 (237.0)	235.0 (180.0)*	345.0 (212.0)*
Creatinine (μmol/L), median (IQR)	60 (20.0)	57.5 (16)*	64 (23)*
D-dimer (mg/L), median (IQR)			
Presentation (*n*=47)	3.4 (4.4)	3.3 (4.7)	3.4 (4.0)
Completion of treatment (*n*=33)	0.2 (0.1)	0.2 (0.1)	0.22 (0.0)
Troponin T (ng/L), median (IQR)	19.0 (36.0)	16.0 (54.0)	27.5 (21.0)

IQR = inter-quartile range; BMI = body mass index; LMWH = low-molecular-weight heparin.

*$p<0.05$;**$p<0.001$.

Results

Seventy-seven (77) women had an objectively diagnosed PE during the study period, of whom 33 (42.9%) were diagnosed during pregnancy and 44 (57.1%) in the postpartum period. The median (IQR) age was 29.0 (9.0) years, and the majority (*n*=73, 94.8%) were of black African ethnicity. There were 15 women (19.5 %) living with HIV on antiretroviral therapy, and an additional 5 (6.5%) who were diagnosed in the current pregnancy. The baseline clinical and laboratory characteristics in the pregnant and postpartum PE women are described in Table 1.

Table 2 lists the antepartum and postpartum risk factors in this cohort. Of the 59 women with a recorded body mass index (BMI) at first antenatal presentation, 20 (33.9%) had a BMI ≥30 kg/m². Thrombophilia testing was performed in 44 women (57.1%) post treatment. Testing revealed a protein S deficiency in 4 (9.1%), and heterozygosity for the non-classical prothrombin gene mutation in 1 (2.3%). There were 4 (12.1%) pregnant and 4 (9.1%) postpartum women classified as high risk for VTE. Of these, only 3 (37.5%) women received thromboprophylaxis in the postpartum period, as per guideline recommendations (Table 3). Among the women with postpartum PE, 31 (70.5%) underwent an emergency caesarean delivery, and only 1 (2.3%) received postpartum thromboprophylaxis.

The live birth rate in this study cohort was 84.4% (Table 4). At delivery, 14/24 (58.3%) women with an antepartum PE underwent a scheduled delivery. The median time interval between the last low-molecular-weight heparin (LMWH) dose and delivery/neuraxial anaesthesia was 24 (15) hours. There was no significant difference in the time intervals between women with scheduled and unscheduled delivery modes (31 (15) hours v. 24 (16) hours, *p*=0.483). Similarly, the rates of neuraxial anaesthesia did not differ significantly between the two subgroups (*p*=0.615).

The baseline PE characteristics and testing performed are presented in Table 5. There were 50 (64.9%) women with a tachycardia >110 beats per minute. Signs of DVT were present in 10 (13.0%) women. Compression ultrasound was performed in 14 (18.1%) at diagnosis, and the presence of a DVT was verified in 6 (7.8%) of the 77 women. The diagnosis was radiologically confirmed using CTPA in 57 women (74.0%), and V/Q scan in 27 women (35.1%), with 7 women undergoing both investigations. Reasons for carrying out both investigations included inconclusive findings on initial investigation (*n*=5), or negative findings on initial investigation in women with strong clinical suspicion of a PE (*n*=2). On CTPA, a segmental PE was the most common type of PE (*n*=42, 54.5%). A diagnosis of multiple PE was confirmed in 35 (45.5%) women, of whom 13 (39.4%) were pregnant and 22 (50.0%) were postpartum.

According to the revised Geneva score, 37 (54.4%) of the pregnant and postpartum women were classified as intermediate risk. D-dimer tests were performed in 23 of the intermediate-risk women, and >0.5 mg/L was recorded in 20 (87.0%). Similarly, D-dimers were >0.5 mg/L in 9 (90.0%) of the low-risk women. There was no significant difference in the median (IQR) scores of pregnant and postpartum women (5 (2.5) and 5 (0.5), respectively; *p*=0.857). For the application of the YEARS algorithm, D-dimer tests were not performed in 28 (36.3%) women. Among the remaining 49 women, according

Table 2. Risk factors for pulmonary embolism in pregnant and postpartum women (*N*=77)

Variable	*n* (%)
Pregnancy (*n*=33)	
Personal history of VTE	4 (12.1)
Positive family history for VTE	1 (3.0)
Inherited thrombophilia*	4 (17.4)
APLS†	2 (9.5)
Medical comorbidities‡	13 (42.4)
Systemic infection	6 (18.2)
Age >35 years	8 (24.2)
Parity ≥3	8 (24.2)
Multiple pregnancy	3 (9.1)
Pre-eclampsia with FGR	3 (9.1)
Hospital admission or immobility	4 (12.1)
Gross varicose veins	1 (3.0)
BMI ≥30 kg/m²§	10 (37.0)
Dehydration/hyperemesis	0 (0)
Postpartum period (*n*=44)	
Personal history of VTE	2 (4.5)
Positive family history for VTE	0 (0)
Inherited thrombophilia*	1 (4.8)
APLS²	2 (9.1)
Medical comorbidities‡	4 (9.1)
Systemic infection	18 (40.9)
Age >35 years	11 (25.0)
Parity ≥3	12 (27.3)
Multiple pregnancy	6 (13.6)
Pre-eclampsia with FGR	17 (38.6)
Non-obstetric surgery	5 (11.4)
Hospital admission or immobility	16 (36.4)
Gross varicose veins	1 (2.3)
BMI ≥40 kg/m²§	6 (20.0)
Preterm delivery	18 (40.9)
Stillbirth	3 (6.8)
Elective caesarean delivery	6 (14.6)
Emergency caesarean delivery	31 (75.6)
Prolonged labour	2 (4.5)
Postpartum haemorrhage	7 (15.9)

VTE = venous thromboembolism; APLS = antiphospholipid syndrome; ART = antiretroviral therapy; FGR = fetal growth restriction; BMI = body mass index; SLE = systemic lupus erythematosus; DM = diabetes mellitus.
*Testing was performed in 23 women with PE in pregnancy and 21 women with postpartum PE.
†Testing was performed in 21 women with PE in pregnancy and 22 women with postpartum PE.
‡Medical comorbidities: antepartum included cardiac disease (*n*=10), active SLE (*n*=1), DM (*n*=2); postpartum included cardiac disease (*n*=3), DM (*n*=1), autoimmune hepatitis (*n*=1).
§BMI was recorded in 27 women with PE in pregnancy and 30 women with postpartum PE.

Table 3. Risk stratification of study participants according to SASTH guidelines[8] (*N*=77)

Antepartum (*n*=33)	*n* (%)	Received thromboprophylaxis, *n* (%)
High risk	4 (12.1)	0 (0)
Intermediate risk or >4 low-risk factors	13 (39.4)	3 (27.3)
Low risk	11 (33.3)	0 (0)
No risk factors	5 (15.2)	0 (0)
Postpartum (*n*=44)		
High risk	4 (9.1)	3 (75.0)
Intermediate risk or ≥2 low-risk factors	32 (72.7)	8 (25.0)
Low risk	6 (13.6)	2 (33.3)
No risk factors	2 (4.5)	0 (0)

SASTH = Southern African Society of Thrombosis and Haemostasis.

Table 4. Obstetric and delivery characteristics (*N*=77)

Clinical characteristic	Total (*N*=77)	Pregnant (*n*=33)	Postpartum (*n*=44)
Hypertension, *n* (%)	32 (41.6)	7 (21.2)	25 (56.8)*
Gestational	9 (11.7)	2 (6.1)	7 (15.9)
Chronic	3 (3.9)	2 (6.1)	1 (2.3)
Pre-eclampsia and FGR	20 (26.0)	3 (9.1)	17 (38.6)
Gestational diabetes, *n* (%)	3 (3.9)	2 (6.1)	1 (2.3)
Delivery characteristic			
Live births, *n* (%)	65 (84.4)	24 (72.7)	41 (93.2)
Intrauterine fetal death, *n* (%)	5 (6.5)	2 (6.1)	3 (6.8)
Miscarriage, *n* (%)	1 (1.3)	1 (3.0)	0 (0.0)
Medical termination of pregnancy, *n* (%)	6 (7.8)	6 (18.2)	0 (0.0)
Gestational age delivery (weeks), median (IQR)†	37.0 (4.0)	37.5 (2.5)	37.0 (6.0)
Birthweight (g), median (IQR)†	2 547.5 (1 075.0)	2 547.5 (831.5)	2 550.0 (1 455.0)
Mode of delivery, *n* (%)†			
Normal vaginal delivery, *n* (%)	13 (20)	7 (29.2)	6 (14.6)
Elective caesarean delivery	11 (16.9)	5 (20.8)	6 (14.6)
Emergency caesarean delivery	41 (63.1)	10 (15.4)	31 (75.6)
Anaesthesia, *n* (%)‡			
General	13 (31.7)	6 (46.2)	7 (25.0)
Epidural/spinal	28 (68.3)	7 (53.8)	21 (75.0)
Estimated blood loss at delivery, mL, median (IQR)†	500 (300)	500 (400)	600 (225)
Wound sepsis, *n* (%)	2 (2.6)	0 (0.0)	2 (4.5)
Postpartum characteristic			
Contraception, *n* (%)	41 (53.2)	14 (42.4)	27 (61.4)*
Contraceptive injection	14 (18.2)	5 (15.2)	9 (20.4)
Bilateral tubal ligation	16 (20.8)	8 (24.2)	8 (18.2)
Intrauterine contraceptive – IUD/IUS	1 (1.3)	1 (3.0)	0 (0.0)
Condom	7 (9.1)	0 (0.0)	7 (15.9)
Oral contraception	3 (3.9)	0 (0.0)	3 (6.8)

FGR = fetal growth restriction; IQR = interquartile range; IUD = intrauterine device; IUS = intrauterine system.
*$p<0.01$.
†Live births (total = 65, pregnant = 24, postpartum = 41).
‡Caesarean deliveries.

to the algorithm, imaging was indicated to rule out PE in 43 (87.8%). The most common clinical criterion, which was present in 40 (81.6%), was 'PE as the most likely diagnosis'. No significant difference between pregnant and postpartum women was observed (p=0.907).

The anticoagulant characteristics are presented in Table 6. All women with PE (n=77, 100%) were managed as inpatients. For initial management, 35 (45.5%) were admitted to high care, and 9 (11.7%) to intensive care, with no significant difference between women with antepartum PE and postpartum PE. The median (IQR) length of hospital admission was 14 (8) days, with no difference between women with antepartum and postpartum PE (p=0.387). In the antepartum period, all pregnant women were treated with LMWH. In the postpartum period, 76 women initially received LMWH for a median (IQR) duration of 11 (6) days. Thereafter, 35 (79.5%) and 9 (20.5%) women were treated with warfarin and rivaroxaban, respectively.

There was one maternal death, in a 38-year-old woman who presented with PE at 26 weeks' gestation. A hysterotomy was performed at 29 weeks due to intrauterine fetal death, complicated by uterine necrosis, necessitating a total abdominal hysterectomy. During her admission, she developed sepsis with organ failure, and died of sepsis-related complications. The incidence of major antepartum/secondary postpartum bleeding was 1 6.5% (95% CI 2.5 - 14.7) (Appendix Table S1). The incidence of major primary postpartum bleeding was 3.9% (95% CI 0.9 - 11.3).

Discussion

In this study, more than half (57.1%) of pregnancy-related PE events occurred within the first 6 weeks postpartum, a pattern consistent with findings from the Registro Informatisado de Enfermedad TromboEmbólica (RIETE) registry.[2] Risk factors for VTE were prevalent in this study population, with 85% of women having at least one antepartum risk factor, and 96% having at least one postpartum risk factor. Similarly, in a study of 21 019 Irish women, 75% had at least one postpartum risk factor, and ~40% had ≥2. [16]

In this cohort, the most common antepartum risk factors were medical comorbidities and obesity. Among black African women, the risk of VTE has been linked to factors including cardiac disease, HIV infection, chronic hypertension, obesity, sickle cell disease and inherited thrombophilias, such as protein S deficiency and antithrombin deficiency.[17,18] In this cohort, 23.3% of women presented with cardiac disease as an antepartum risk factor for VTE, compared with only 0.2% in the RIETE registry.[2] Our study was conducted at an academic obstetric unit, which may account for the higher prevalence of cardiac disease. Additionally, peripartum and hypertension-related cardiomyopathy in African populations is a contributing risk factor for VTE.[19] Obesity, another major contributor to VTE risk, reflects the broader epidemiological transition in developing regions toward non-communicable diseases. In our cohort, 37% of women with antepartumPE had a BMI ≥30 kg/m². Obesity poses an additional challenge in managing thrombosis, as

Table 5. Baseline clinical characteristics at pulmonary embolism diagnosis (*N*=77)

Clinical characteristic	Total (*N*=77)	Pregnant (*n*=33)	Postpartum (*n*=44)
Gestational age at diagnosis, weeks, median (IQR)	28.0 (22.0)	28.0 (22.0)	-
Number of days postpartum, median (IQR)	3.0 (6.0)	-	3.0 (6.0)
Symptom, *n* (%)			
Chest pain	21 (27.3)	14 (42.4)*	7 (15.9)
Shortness of breath	44 (57.1)	24 (72.7)**	20 (45.5)
Palpitations	5 (6.5)	5 (15.2)**	0 (0.0)
Haemoptysis	2 (2.6)	1 (3.0)	1 (2.3)
Symptoms of DVT	7 (9.1)	5 (15.1)	2 (4.5)
Sign			
Heart rate, bpm, median (IQR)	120 (17)	120 (20)	120 (16)
SBP (mmHg), median (IQR)	126 (25)	126 (21)	126 (25)
DBP (mmHg), median (IQR)	83 (19)	83 (20)	84 (19)
SPO_2 (%), median (IQR)	95 (5)	94 (4)	96 (7)
RR, rpm, median (IQR)	20 (10)	20 (8)	22 (6)
Temperature, °C, median (IQR)	36.5 (0.3)	36.5 (0.2)	36.5 (0.6)
GCS, median (IQR)	15 (0)	15 (0)	15 (0)
Signs of DVT, *n* (%)	10 (13.0)	7 (21.2)	3 (6.8)
Scoring			
Revised Geneva score, *n* (%)			
Low (0 - 1 point)	10 (14.7)	6 (21.4)	4 (10.0)
Intermediate (2 - 6 points)	37 (54.4)	11 (39.3)	26 (65.0)**
High clinical (≥7 points)	21 (30.9)	11 (39.3)	10 (25.0)
Insufficient data to complete scoring, *n*=9			
YEARS algorithm, *n* (%)			
No clinical criteria and D-dimer <1.0 mg/L	0 (0.0)	0 (0.0)	0 (0.0)
No clinical criteria and D-dimer ≥1.0 mg/L	4 (8.2)	1 (4.5)	3 (11.1)
1 - 3 clinical criteria and D-dimer <0.5 mg/L	6 (12.2)	3 (13.6)	3 (11.1)
1 - 3 clinical criteria and D-dimer ≥0.5mg/L	39 (79.6)	18 (81.8)	21 (77.8)
Insufficient data to complete scoring, *n*=28			
Diagnostic investigation, *n* (%)			
Chest X-ray	52 (67.5)	22 (66.7)	30 (68.2)
Electrocardiogram	68 (88.3)	32 (97.0)	36 (81.8)
Echocardiogram	44 (57.1)	27 (81.8)	17 (38.6)
CTPA	57 (74.0)	24 (72.7)	33 (75.0)
V:Q	29 (37.7)	16 (48.5)	13 (29.5)
Compression ultrasound	14 (18.2)	12 (36.4)	2 (4.5)
D-dimer	48 (62.3)	21 (63.6)	27 (61.4)
Baseline CTPA (*n*=57), *n* (%)			
Pulmonary trunk PE	5 (8.8)	3 (12.5)	2 (6.1)
Main pulmonary artery PE	4 (7.0)	2 (8.3)	2 (6.1)
Lobar pulmonary artery PE	11 (19.3)	4 (16.7)	7 (21.2)
Segmental pulmonary artery PE	42 (73.7)	15 (62.5)	27 (81.8)
Subsegmental PE	13 (22.8)	6 (25.0)	7 (21.2)
Pulmonary trunk size, ≥30 mm	18 (23.4)	10 (30.3)	8 (18.2)
Baseline echocardiogram (*n*=44)			
Tricuspid annular plane systolic excursion (mm), median (IQR)	23 (9)	21 (10)	23 (5)
Pulmonary artery pressure, mm, median (IQR)	52 (70)	52 (72)	51 (59)
Right ventricular systolic dysfunction, *n* (%)	10 (22.7)	9 (33.3)	1 (5.9)

IQR = interquartile range; DVT = deep-vein thrombosis; SBP = systolic blood pressure; DBP = diastolic blood pressure; SPO_2 = oxygen saturation; RR = respiratory rate; rpm = respiration per minute; GCS = Glasgow Coma Scale, CTPA = computed tomography pulmonary angiogram; V:Q = ventilation-perfusion scan; PE = pulmonary embolism.
*p<0.01; **p<0.05.

the altered pharmacokinetics may necessitate dose adjustments, which are recommended in those at the extremes of weight, together with anti-Xa monitoring of LMWH to ensure efficacy. Furthermore, 30.3% of women with antepartum PE were living with HIV. While this is consistent with national antenatal prevalence rates, 60% had a high viral load (>50 copies/mL). Further study of unsuppressed HIV as a prothrombotic condition is required in this setting.[18] Inherited thrombophilia were less common, observed in only 17.4% of women,

Table 6. Anticoagulation characteristics (*N*=77)

Characteristic	Total, *N*=77	Pregnant, *n*=33	Postpartum, *n*=44
Hospital setting (initial management), *n* (%)			
High-care inpatient	35 (45.5)	14 (42.4)	21 (47.7)
ICU inpatient	9 (11.7)	5 (15.2)	4 (9.1)
Length of stay, median (IQR)			
High-care inpatient	4 (4)	5 (7)	4 (3)
ICU inpatient	7 (2)	7 (1)	7 (5.5)
General ward inpatient	11 (10)	10 (9)	13 (10)
Anticoagulant			
Medical thrombolysis, *n* (%)	4 (5.2)	3 (9.1)	1 (2.3)
Catheter-directed, *n* (%)	0 (0)	0 (0)	0 (0)
LMWH, *n* (%)	76 (98.7)	33 (100)	43 (97.7)
Dose LMWH (mg bd subcutaneously), mean (SD)	80 (20)	80 (20)	80 (20)
Weight adjusted LMWH, *n* (%)	49 (64.5)	17 (51.5)	32 (74.4)
Anti-Xa adjusted LMWH, *n* (%)	27 (35.5)	16 (48.5)	11 (25.6)
Anti-Xa antepartum (IU/mL), mean (SD)	-	0.6 ± 0.3	-
Warfarin postpartum, *n* (%)	-	-	35 (79.5)
Warfarin daily dose at discharge, mg, median (IQR)	5 (2.5)	5 (1.9)	5 (2.5)
TTR, median (IQR)	-	-	45.3 (55.1)
Rivaroxaban postpartum, *n* (%)	-		9 (20.5)
Duration of treatment (months), median (IQR)	3 (1)	3 (1)	3 (2)
Complication, *n* (%)			
Skin reactions/bruising	2 (2.6)	2 (6.1)	0 (0.0)
Treatment changes due to drug hypersensitivity*	2 (2.6)	2 (6.1)	0 (0.0)
CTEPH†	3 (8.3)	3 (15.8)	0 (0.0)
Post-thrombotic syndrome‡	0 (0.0)	0 (0.0)	0 (0.0)
Outcome, *n* (%)			
Bleeding			
Minor	3 (21.4)	3 (27.3)	0 (0.0)
CRNMB	3 (21.4)	2 (18.2)	1 (33.3)
Major	8 (57.1)	6 (54.5)	2 (66.7)
Recurrent VTE	0 (0.0)	0 (0.0)	0 (0.0)
Death	1 (1.3)	1 (3.0)	0 (0.0)

ICU = intensive care unit; IQR = interquartile range; LMWH = low-molecular-weight heparin; SD = standard deviation; TTR = time in therapeutic range; CTEPH = chronic thromboembolic pulmonary hypertension; CRNMB = clinically relevant non-major bleeding; VTE - venous thromboembolism.
*Platelet count dropped >50% after 5 days. LMWH switched to fondaparinux (30 weeks' gestation).
The platelet count dropped >50% after 5 days. UFH switched to LMWH.
†Assessed in 36 participants
‡Assessed in 6 participants with deep-vein thrombosis.

compared with 55% in the RIETE registry. Key postpartum risk factors identified included emergency caesarean delivery (CD), preterm delivery and systemic infection. To date, clinical risk factors have been associated with a modest effect on VTE risk,[10] and a large population-based case-controlled study is needed to determine the significance of clinical risk factors for antepartum and postpartum VTE in black African populations.

A striking finding was the lack of thromboprophylaxis among high-risk antepartum women, despite the presence of well-defined risk factors. This is in contrast to the postpartum women in our study, 75% of whom received appropriate thromboprophylaxis. Furthermore, postpartum thromboprophylaxis was not routinely prescribed to women who underwent emergency CD – who are recommended to receive 7 - 10 days of thromboprophylaxis – despite existing guideline recommendations (class IIb, level C).[11] Contributing factors may include the absence of structured VTE risk assessments during routine antenatal care, limited clinician familiarity with guidelines and concerns about bleeding risk, particularly in women requiring neuraxial anaesthesia or with high CD rates. Nonetheless, the absence of bleeding risk assessments in this study may have influenced decisions regarding thromboprophylaxis use and its appropriateness in this high-risk population. Resource constraints and patient adherence challenges may also play a role, although this needs to be investigated further.

The diagnosis of VTE in pregnancy can be challenging. The clinical presenting signs and symptoms in the study population, which most commonly included tachycardia and dyspnoea, overlap with the physiological changes of pregnancy. An unusual finding in this study was that only 13% of patients presented with signs and symptoms of DVT. This contrasts with the expected concomitant DVT rates of 27% in antepartum and 42% in postpartum women with PE, as reported in the RIETE registry.[2] One possible explanation for this discrepancy is that compression ultrasound was not frequently performed, with utilisation in only 18.1% of the study population. The preferred specific investigation for the diagnosis of PE in this study was CTPA. CTPA is widely available in most hospitals, and is the first-line investigation in many centres in pregnancy. However, the radiation dose protocols vary widely. Recent advances have focused on integrating D-dimer testing with clinical decision rules to predict PE in pregnant and

postpartum women.[12,13] In our cohort, the pregnancy-adapted YEARS algorithm was evaluated in 49 women. A high proportion (87.8%) met the criterion 'PE as the most likely diagnosis', and among these, 86% had positive D-dimer results. The adapted Geneva score was assessed in 68 women, but it was less effective in identifying high clinical probability; only 30.9% were categorised as high clinical probability for PE. Nonetheless, when combined with D-dimer testing, 87.9% of low- or intermediate-risk women would still have required imaging, suggesting a potential role for such cost-effective strategies in resource-limited settings.[20] Updated guidelines from the European Society of Cardiology[21] recommend the use of pretest probability scores and D-dimer testing (class IIa recommendation) to safely rule out PE in pregnancy. However, a prospective validation of these diagnostic algorithms in pregnant SA populations is needed to assess safety and cost-effectiveness.

Management predominantly involved weight-adjusted LMWH, reflecting guideline recommendations.[9-11] Owing to the low incidence of recurrent VTE or major bleeding events, the utility of anti-Xa monitoring could not be fully assessed. Future studies are warranted to determine the safety and efficacy of anti-Xa adjusted LMWH, especially in women at the extremes of weight or with renal impairment.[22] Postpartum treatment typically transitioned from LMWH to warfarin, in line with current practice guidelines, while direct oral anticoagulants (DOACs) were rarely used owing to concerns about safety during breastfeeding.[9]

The maternal mortality directly attributable to PE in this cohort was zero. One maternal death occurred due to sepsis-related complications in a woman with multiple comorbidities and postpartum PE. Major bleeding events were observed in 6.5% of cases during the antepartum or secondary postpartum period, consistent with similar studies.[23] Primary postpartum major bleeding occurred in 3.9% of cases. Notably, the rates of both elective and emergency CD were high at this referral centre for high-risk pregnancies, at 17.6% and 60.3%, respectively. A scheduled vaginal delivery is the preferred mode of delivery in most situations, with the timing and approach guided by obstetric indications.

Study limitations

Several limitations must be acknowledged. First, the study was conducted at a single tertiary referral centre, potentially limiting generalisability to the broader SA population. Second, D-dimer testing was performed in only 61.0% of the participants at presentation, which limited interpretation of the pretest probability scores. Third, the study was not designed to assess for long-term complications, in particular VTE recurrence, post-thrombotic syndrome and CTEPH. Nevertheless, the observed CTEPH rate of 8.3%, higher than previously reported in non-pregnant populations, warrants further investigation.[6] Finally, the lack of a control group precluded a comparative analysis, which could have provided additional insights into maternal and fetal outcomes.

Conclusion

This study provides important insights into the clinical characteristics, management and outcomes of pregnancy-associated PE in a SA tertiary centre. Risk factors for PE were found in the majority of patients, which can provide guidance for the rational use of antepartum and postpartum thromboprophylaxis. The use of a D-dimer test in conjunction with pre-test probability scores offers a potentially cost-effective approach for low-resource settings. Future prospective studies are needed to validate these strategies and to better characterise long-term outcomes in the SA pregnant population.

Data availability. The data sets generated and analysed during the present study are available from the corresponding author (ES) on reasonable request.

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