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(CASE REPORT)

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Management of Delayed Diagnosis of Uterine Rupture: A Case Report

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Abstract

Uterine rupture is commonly described as an obstetrics catastrophy due to its associated increase in maternal as well as perinatal morbidity and mortality. The diagnosis of uterine rupture is usually clinical especially when there are background obvious risk factors. However, when risk factors are not obvious or classical clinical features or bleeding per vaginam more especially after delivery is assumed to be due to cervical or perineal tear, occurrence of uterine rupture could be missed and hence delay in the diagnosis. In this case report, we describe a case of missed uterine rupture which was diagnosed after 24 hours of vaginal delivery in a tertiary hospital.

Keywords: Induction of labour; Vaginal delivery; Primary postpartum haemorrhage; Delayed/missed diagnosis of uterine rupture; Uterine repair

1. Introduction

Uterine rupture is an obstetrics emergency usually described as an obstetric catastrophy due to the associated increased perinatal and maternal morbidity and mortality [1]. When the diagnosis is not made immediately after rupture leading to delays of up to a day or several days, the morbidity and mortality is much worse. This delay usually occurs when the common risk factors for uterine rupture previous uterine scar, high parity, use of oxcytocics in labour, instrumental vaginal deliveries and uterine manipulations during the peripartum periods [2] are not readily evident or where associated bleeding from the rupture is slow and not revealed through the vagina. The common symptoms of complete uterine rupture which include but not limited to foetal heart rate abnormalities, abdominal pains and distension, bleeding per vaginam [3] and signs of shock such as hypotension, maternal tachycardia and tachypnea may also be present depending on the interval between the occurrence of the rupture and the diagnosis. These symptoms and signs usually develop slowly and become obvious later after several hours or days culminating in delayed recognition of uterine rupture. Rottenstreich et al identified unscarred uterus, epidural analgesia during labour and grandmultiparity as independent risk factors for delayed diagnosis of uterine rupture [4]. When other causes of primary postpartum haemorrhage occur at the period of delivery, the initial bleeding may only be attributed to this leading to missed diagnosis and the delay in the diagnosis. Our patient had cervical laceration with active bleeding which was repaired and the bleeding stopped. This possibly masked the co-existing uterine rupture and then the associated delay in the diagnosis. Maternal outcomes equally showed that parturients with a delayed diagnosis had significantly higher rates of blood transfusions, puerperal fever, and hysterectomy [4]. Sepsis and intra-abdominal abscess collection are also common findings in cases of missed or delayed diagnosis of uterine rupture [5,6,7]. These were not seen in our patient as the delay in diagnosis within 24 hours and these could not have developed. We present a case of uterine rupture in an unscarred uterus which diagnosis was missed for over a duration of 24 hours after vaginal delivery in a tertiary hospital.

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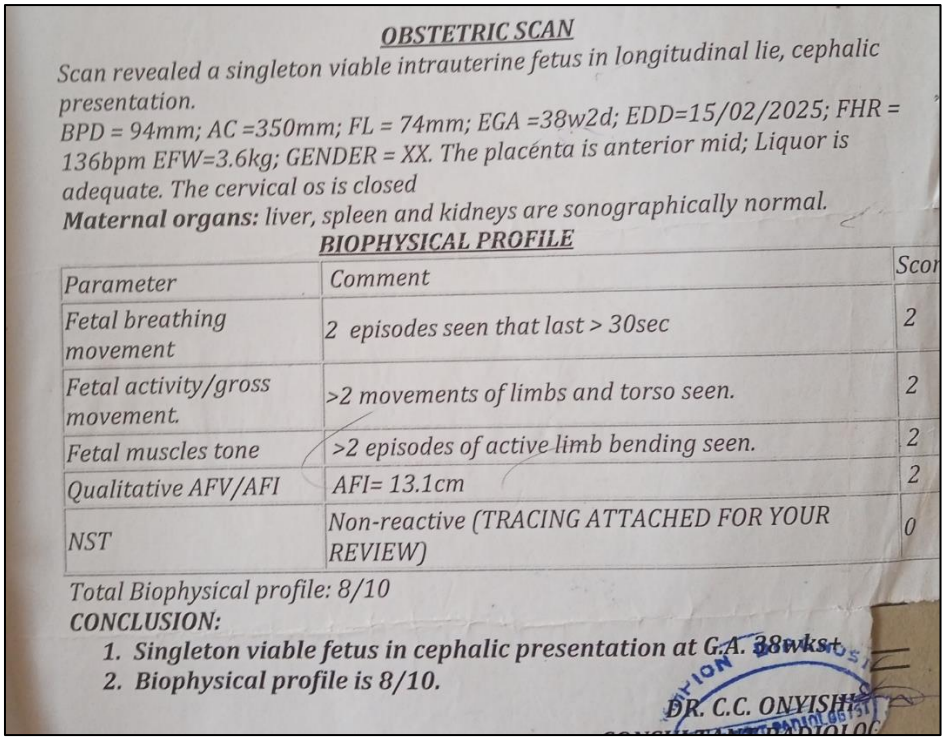
2. Case

Mrs EU, a 37 year old G4P3A3 who was admitted to the prenatal ward at a gestational age of 41 weeks + 2 days with elevated blood pressure of 160/100mmHg with lower limb oedema that extended to involve the lower abdomen. She had no other symptoms of pre-eclampsia with severe features. She had postdate pregnancy in the second and the third pregnancy was complicated with pre-eclampsia. She had induction of labour in the second and the third pregnancy on account of postdatism and pre-eclampsia respectively. All her deliveries were via the vaginal route and with normal puerperal periods. Her medical history was not significant and she had no history of uterine surgery.

On admission, she had no uterine contractions. Vaginal examination showed Bishop Scores of 3/13. Her urinalysis showed proteinuria of 2++. The biophysical profile and nonstress test done showed a score of 8/10 (figure 1). Nonstress test was 0. Her platelet counts was normal. The liver function test as well as serum electrolyte urea and creatinine showed normal findings (figure 2). She was commenced on MgSo4 according to the Pritchart regimen-hospital protocol. She was equally commenced on oral antihypertensives; oral Alpha methyldopa 500mg 8 hourly and oral Nifedipine 20mg daily. The blood pressure thereafter remained fairly controlled between 150/90-140/90 mmHg. She was counseled on the options of delivery route. She declined caesarean section citing that she had induction of labour in the previous pregnancy for similar condition and had good outcome. She was counseled on the risks and she consented to induction of labour with close foeto-maternal surveillance. Her packed cell volume was 37% and 2 units of blood were cross-matched for her. She was then commenced on cervical ripening with 25µg trans-vaginal misoprostol and this transited her to labour pains after 2 hours of administration. The continuous electronic foetal monitoring was instituted which showed essentially normal findings throughout the labour period. She expressed irresistible urge to bear down 4 hours later and subsequently had vaginal delivery of a live female neonate with APGAR Scores of 8^{1} and 10^{5}. Birth weight was 3.4kg. The third stage of labour was managed actively. Significant vaginal bleeding was noticed shortly after the delivery of the placenta and assessment of primary postpartum haemorrhage due to uterine atony and genital tracts lacerations which included cervical laceration was made. She received 80 iu oxytocin infusion. She had EUA with cervical and posterior vaginal wall repair and bleeding per vaginam stopped. She received a unit of whole blood during the procedure. She was admitted to the High Dependency unit of the labour ward where she received additional 2 units of whole blood. About 20 hours later she was transferred to the postnatal ward in a fair clinical condition but with tachycardia. About 3 hours after transfer to the postnatal ward, she complained of abdominal pain and generalized body weakness. She was in obvious painful distress, pale, afebrile with a temperature of 36.5^{0}c. Other vital signs were; pulse rate of 118 beats per minute, blood pressure of 140/80mmHg, respiratory rate of 22 cycles per minute and SPO2 of 98% at roam air. Her abdomen was distended with generalized tenderness. Abdominal tap showed positive aspirate of 4ml of non-clotting blood and suspicion of uterine rupture was made. Urgent abdominopelvic ultrasound showed moderate to massive intra-peritoneal fluid with echoes and floating bowel loops of normal caliber with impression of haemo-peritoneum (figure 3). Diagnosis of ruptured uterus was then made. Patient was counseled on the findings and need for emergency exploratory laparotomy and possible hysterectomy. Written informed consent was obtained. Urgent packed cell volume was 18% (Figure 4). Clotting profile showed normal parameters (figure 5). Four units of blood were cross-matched. She subsequently had exploratory laparotomy and findings showed haemo-peritoneum of 2500ml with a right lateral lower uterine segment tear about 8cm long extending to the cervix with laceration of the right uterine vessel with active bleeding. There was an ipsilateral broad ligament haematoma of 10cm in dimension. The broad ligament haematoma was evacuated and the uterine laceration repaired using number 2 vicryl suture in 2 layers and haemostasis achieved. She received 3 units of blood intra-operation. Immediate post-operative condition was satisfactory. She received 3 additional units of blood in the post-operative period making a total of 9 units of blood in the course of the treatment. Her general clinical condition improved gradually and was discharged in satisfactory condition 5 day post-operation with haematocrit level of 29%.



OBSTETRIC SCAN

Scan revealed a singleton viable intrauterine fetus in longitudinal lie, cephalic presentation.
BPD = 94mm; AC =350mm; FL = 74mm; EGA =38w2d; EDD=15/02/2025; FHR = 136bpm EFW=3.6kg; GENDER = XX. The placenta is anterior mid; Liquor is adequate. The cervical os is closed
Maternal organs: *liver, spleen and kidneys are sonographically normal.*

BIOPHYSICAL PROFILE

Parameter	Comment	Scor
Fetal breathing movement	2 episodes seen that last > 30sec	2
Fetal activity/gross movement.	>2 movements of limbs and torso seen.	2
Fetal muscles tone	>2 episodes of active limb bending seen.	2
Qualitative AFV/AFI	AFI= 13.1cm	2
NST	Non-reactive (TRACING ATTACHED FOR YOUR REVIEW)	0

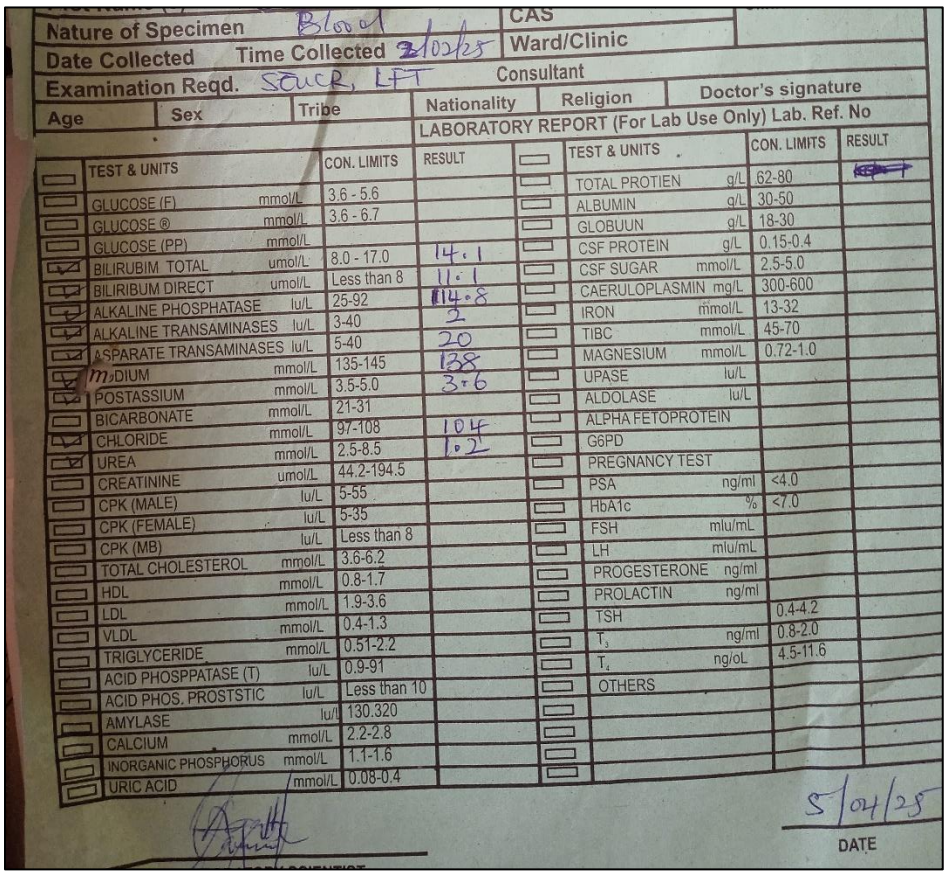
Total Biophysical profile: 8/10

CONCLUSION:

1. ***Singleton viable fetus in cephalic presentation at G.A. 38wks+***
2. ***Biophysical profile is 8/10.***

DR. C.C. ONYISHI

Figure 1 Biophysical profile and non stress test



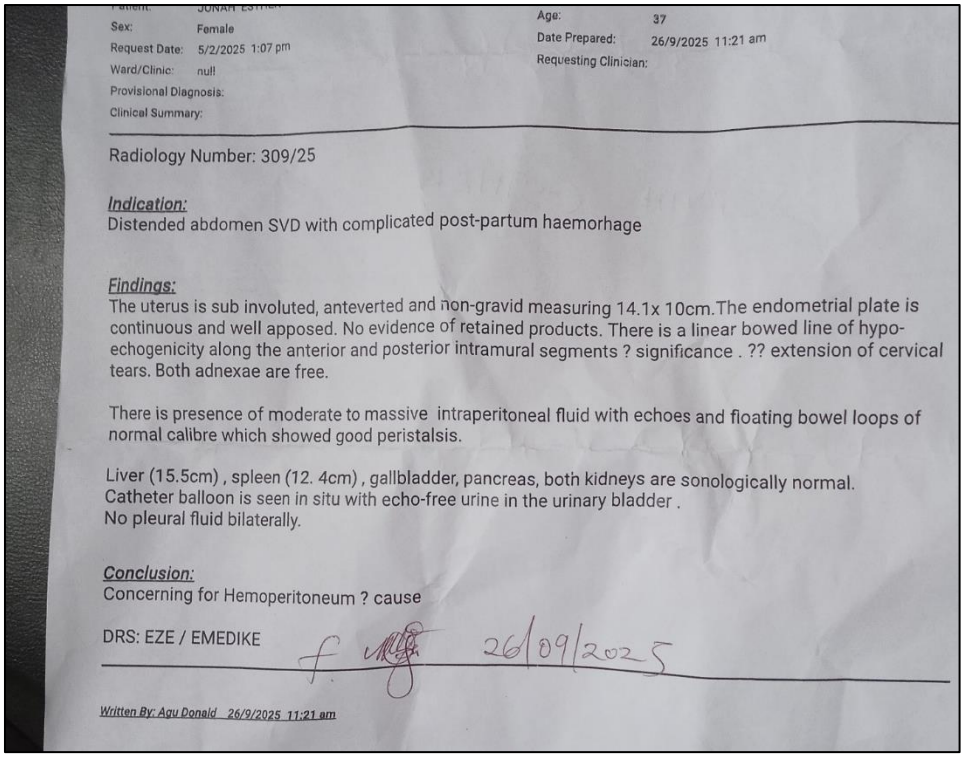
Nature of Specimen: Blood — CAS
Date Collected: 3/02/25 — Time Collected — Ward/Clinic
Examination Reqd.: SOUCR, LFT — Consultant
Age | Sex | Tribe | Nationality | Religion | Doctor's signature
LABORATORY REPORT (For Lab Use Only) Lab. Ref. No

TEST & UNITS	CON. LIMITS	RESULT
GLUCOSE (F) mmol/L	3.6 - 5.6	
GLUCOSE ® mmol/L	3.6 - 6.7	
GLUCOSE (PP) mmol/L		
BILIRUBIM TOTAL umol/L	8.0 - 17.0	14.1
BILIRIBUM DIRECT umol/L	Less than 8	11.1
ALKALINE PHOSPHATASE iu/L	25-92	114.8
ALKALINE TRANSAMINASES iu/L	3-40	3
ASPARATE TRANSAMINASES iu/L	5-40	20
SODIUM mmol/L	135-145	138
POSTASSIUM mmol/L	3.5-5.0	3.6
BICARBONATE mmol/L	21-31	
CHLORIDE mmol/L	97-108	104
UREA mmol/L	2.5-8.5	1.02
CREATININE umol/L	44.2-194.5	
CPK (MALE) iu/L	5-55	
CPK (FEMALE) iu/L	5-35	
CPK (MB) iu/L	Less than 8	
TOTAL CHOLESTEROL mmol/L	3.6-6.2	
HDL mmol/L	0.8-1.7	
LDL mmol/L	1.9-3.6	
VLDL mmol/L	0.4-1.3	
TRIGLYCERIDE mmol/L	0.51-2.2	
ACID PHOSPPATASE (T) iu/L	0.9-91	
ACID PHOS. PROSTSTIC iu/L	Less than 10	
AMYLASE iu/l	130.320	
CALCIUM mmol/L	2.2-2.8	
INORGANIC PHOSPHORUS mmol/L	1.1-1.6	
URIC ACID mmol/L	0.08-0.4	

TEST & UNITS	CON. LIMITS	RESULT
TOTAL PROTIEN g/L	62-80	
ALBUMIN g/L	30-50	
GLOBUUN g/L	18-30	
CSF PROTEIN g/L	0.15-0.4	
CSF SUGAR mmol/L	2.5-5.0	
CAERULOPLASMIN mg/L	300-600	
IRON mmol/L	13-32	
TIBC mmol/L	45-70	
MAGNESIUM mmol/L	0.72-1.0	
UPASE iu/L		
ALDOLASE iu/L		
ALPHA FETOPROTEIN		
G6PD		
PREGNANCY TEST		
PSA ng/ml	<4.0	
HbA1c %	<7.0	
FSH mlu/mL		
LH mlu/mL		
PROGESTERONE ng/ml		
PROLACTIN ng/ml		
TSH	0.4-4.2	
T_3 ng/ml	0.8-2.0	
T_4 ng/oL	4.5-11.6	
OTHERS		

5/04/25
DATE

Figure 2 Liver and kidney function tests before exploratory laparotomy



Sex: Female
Age: 37
Request Date: 5/2/2025 1:07 pm
Date Prepared: 26/9/2025 11:21 am
Ward/Clinic: null
Requesting Clinician:
Provisional Diagnosis:
Clinical Summary:

Radiology Number: 309/25

Indication:
Distended abdomen SVD with complicated post-partum haemorhage

Findings:
The uterus is sub involuted, anteverted and non-gravid measuring 14.1x 10cm.The endometrial plate is continuous and well apposed. No evidence of retained products. There is a linear bowed line of hypo-echogenicity along the anterior and posterior intramural segments ? significance . ?? extension of cervical tears. Both adnexae are free.

There is presence of moderate to massive intraperitoneal fluid with echoes and floating bowel loops of normal calibre which showed good peristalsis.

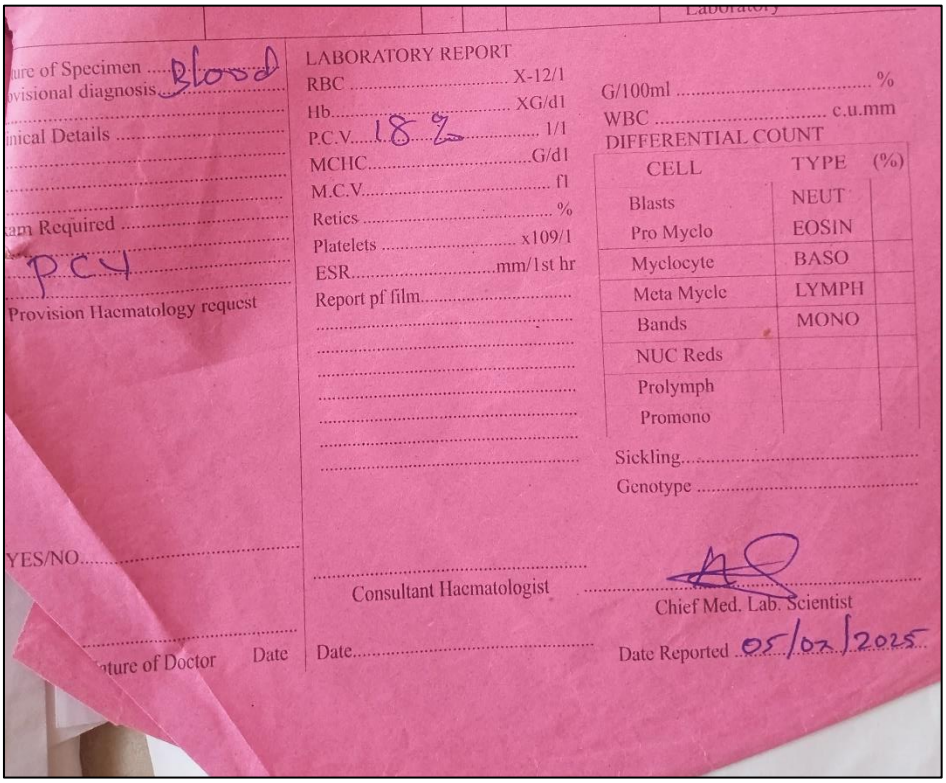
Liver (15.5cm) , spleen (12. 4cm) , gallbladder, pancreas, both kidneys are sonologically normal.
Catheter balloon is seen in situ with echo-free urine in the urinary bladder .
No pleural fluid bilaterally.

Conclusion:
Concerning for Hemoperitoneum ? cause

DRS: EZE / EMEDIKE 26/09/2025

Written By: Agu Donald 26/9/2025 11:21 am

Figure 3 Abdominopelvic ultrasound scan that suggested uterine rupture



ure of Specimen Blood
visional diagnosis
nical Details
am Required
PCV
Provision Haematology request

YES/NO
nture of Doctor Date

LABORATORY REPORT
RBC X-12/1
Hb XG/d1
P.C.V. 18% 1/1
MCHC G/d1
M.C.V. fl
Retics %
Platelets x109/1
ESR mm/1st hr
Report pf film

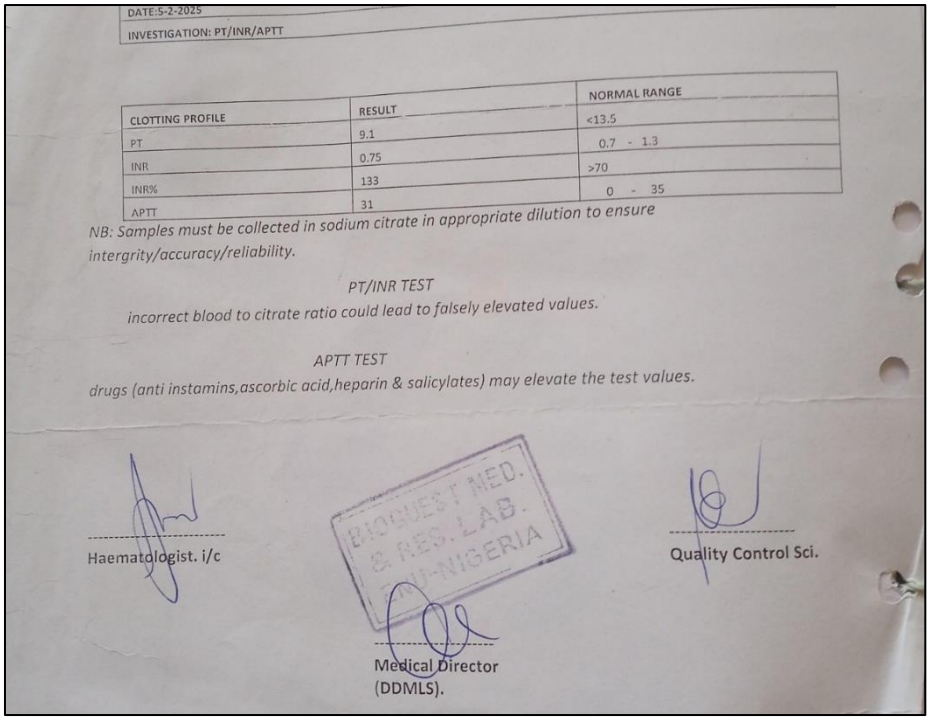
G/100ml %
WBC c.u.mm
DIFFERENTIAL COUNT

CELL	TYPE	(%)
Blasts	NEUT	
Pro Myclo	EOSIN	
Myclocyte	BASO	
Meta Mycle	LYMPH	
Bands	MONO	
NUC Reds		
Prolymph		
Promono		

Sickling
Genotype

Consultant Haematologist
Date
Chief Med. Lab. Scientist
Date Reported 05/02/2025

Figure 4 Pre-operative packed cell volume



DATE:5-2-2025

INVESTIGATION: PT/INR/APTT

CLOTTING PROFILE	RESULT	NORMAL RANGE
PT	9.1	<13.5
INR	0.75	0.7 - 1.3
INR%	133	>70
APTT	31	0 - 35

NB: Samples must be collected in sodium citrate in appropriate dilution to ensure intergrity/accuracy/reliability.

PT/INR TEST

incorrect blood to citrate ratio could lead to falsely elevated values.

APTT TEST

drugs (anti instamins,ascorbic acid,heparin & salicylates) may elevate the test values.

Haematologist. i/c

BIOMEDT MED. & RES. LAB. -NIGERIA

Quality Control Sci.

Medical Director (DDMLS).

Figure 5 Clotting profile picture before exploratory laparotomy

3. Discussion

Uterine rupture is a cause of major morbidity and mortality [1]. Commonly, uterine rupture is an intra-partum event occurring following induction/augmentation of labour, instrumental vaginal delivery, in a high parity or those with previous uterine surgeries or congenital uterine anomalies[2]and the diagnosis of rupture can be made promptly. The diagnosis of uterine rupture can be missed or delayed shortly or several days after delivery where some of these risk factors are not evident commonly in parturients with unscarred uterus, high parity and use of epidural analgesia in labour [4,5,6,7]. Where delay was prolonged, there is occasionally associated sepsis and intra-abdominal abscess [5,6,7]. Sepsis and intra-abdominal abscess collections were not seen in our patient as diagnosis was made within 24 hours of occurrence. In Portugal, Rosado and the study group reported delayed diagnosis of uterine rupture in an unscarred uterus for a duration of 6 days after uncomplicated vaginal delivery for which the patient subsequently had hysterectomy [5]. While she was para 3 just like our patient, her labour was spontaneous unlike our patient that received prostaglandin. The rupture could have been due to the delivery of foetal macrosomia. The absence of common risk factor such as previous caesarean section, induction or augmentation of labour could have been responsible for the delay in diagnosis unlike our patient that received prostaglandin. Similar delayed diagnosis of uterine rupture of 23 days after uncomplicated vaginal delivery with previous caesarean section was reported by Ali and his colleagues [6]. The vaginal delivery was supervised at home by a midwife. The delay in the diagnosis despite bleeding per vaginam that developed shortly after vaginal delivery and finding of over 2 litres of peritoneal haemorrhage, could have been due to the possible poor surveillance in the midwife led home delivery compared to better surveillance in an obstetrician led care in teaching hospital setting where our patient was managed with earlier diagnosis. The delay could have also been as a result of slow bleeding from previous uterine scar which was the only point of rupture unlike in our patient where rupture was on intact uterus and bleeding expected to be more rapid leading to earlier deterioration of patient's condition with earlier diagnosis. In Brooklyn, New York City, Narasimhulu and his colleagues reported delay of 3 days in the diagnosis of uterine rupture in a parturient with a previous caesarean who had uncomplicated vaginal delivery at term [7]. The diagnosis of uterine rupture was with the aid of abdominal ultrasound and computed tomography (CT) which revealed uterine defect and pelvic fluid collection tracking into the right paracolic gutters. The patient had CT guided drainage of this collection with complete recovery but the uterine defect was left unrepaired. The delay in the diagnosis here was possibly due to patient's major complaints of high grade fever, hypotension, tachypnea which were more suggestive of sepsis unlike in our patient where hypotension and abdominal distension suggesting more of intraperitoneal blood collection leading to earlier suspicion of uterine rupture and diagnosis. Rupture at the previous scar and possible associated slow rate of haemorrhage could have led to the delay in diagnosis compared to our patient that had rupture of the intact uterus. While diagnosis was largely via radiologic evaluation, definitive diagnosis of uterine rupture was made during laparotomy in our patient. In Palestine, Iqnaibi and the study group reported a missed

diagnosis of uterine rupture which occurred 5 days after uneventful vaginal delivery in a parturient with previous caesarean section [8]. While high parity and previous caesarean section were possible risk factors here, our patient had no previous uterine scar and was only para 3. The delayed diagnosis could be due to slow bleeding from previous scar and major complaints which were mainly suggestive of sepsis. The delay in the diagnosis in our patient was the initial believe that the postpartum haemorrhage was due only to the cervical laceration which she had that was promptly repaired.

4. Conclusion

Uterine rupture remains an obstetrics catastrophy. In an unscarred uterus, early diagnosis can be missed. High index of suspicion and close postpartum vital sign monitoring is therefore very imperative in early detection of uterine rupture to prevent associated increased maternal morbidity and mortality in delayed or missed diagnosis especially in cases of high parity, augmentation or induction of labour.

Compliance with ethical standards

Acknowledgement

The authors acknowledge the help of the radiology department in the care of the patient

Disclosure of conflict of interest

The authors report no conflict of interest

Statement of ethical approval

Ethical approval was obtained according to institutional protocols. Approval number: ESUCOM/FBMS/ETR/2026/ 006. Privacy and confidentiality of the patient was fully observed.

Statement of informed consent

The woman whose story was reviewed in this case report had provided consent for its publication

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