

Rising Troponin Postpartum in a Patient with Severe Pre-Eclampsia : A Clinical Conundrum.

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BACKGROUND

Cardiovascular manifestations from pre-eclampsia are attributable to vascular endothelial activation and vasospasm resulting in hypertension and multi-organ hypoperfusion.

Elevated troponin, especially alongside severe pre-eclampsia, presents a clinical conundrum due to complexities differentiating between a wide differential diagnosis in pregnancy. We demonstrate the cruciality of a multidisciplinary approach for optimal patient management.

CASE DESCRIPTION



31-year-old primigravida
Presented at 34+1 weeks with headache and peripheral oedema to her local DGH.



Low risk midwifery care at booking.
No significant PMH. Booking BP 117/73 mmHg. Vaping.
Normal Anomaly Scan and UAD.



Fetal Medicine Unit Review at 32+0
Severe polyhydramnios (AFI 41.2cm). Small stomach bubble.
Normal TORCH screen and oral glucose tolerance test.
Plan for tertiary unit birth - ? tracheoesophageal fistula (TOF).



PC: Hypertension 152/110 mmHg and 2+ proteinuria.
Normal reflexes. Normal CTG.



Commenced Labetalol 200mg TDS + Nifedipine 20mg MR BD.



UPCR 414 mg/mmol.
PLGF <12 pg/nl.



Normal FBC, coagulation, renal function and liver function.



Plan for IUT in event of need for pre-term birth and neonatal surgery due to potential TOF.



Figure 1: Chest X-Ray (CXR) at 34+4 weeks
Pulmonary Oedema with slight cardiomegaly

PATIENT TIMELINE

Transfer to Tertiary Unit at 34+2

34+4 Pulmonary Oedema

Management

Birth at 34+6

Day 1 Postnatal (PN)

Day 6 Postnatal – New Chest Pain

BP controlled on Labetalol 300mg TDS + Nifedipine 20mg MR BD
Stable PET bloods

Dyspnoeic with O2 sats 90% on air
Chest auscultation: Reduced air entry bibasally with fine crepitations
CXR: Pulmonary oedema with slight cardiomegaly
ECG: T wave inversion V1-4
BNP: 1023 ng/l
O2 commenced

ECHO and Cardiology review:
Mildly dilated LV with EF 55%.
No pericardial effusion or valvular pathology

Diagnosis severe PET
Possible peripartum cardiomyopathy
IV Furosemide commenced
Fluid restriction started
MDT decision for delivery
Dexamethasone course completed
MGSO4 for fetal neuroprotection

Category 3 CS under Spinal anaesthetic
EBL 900ml

Baby girl 2330g (28th customised centile)
No evidence of TOF
Neonatal respiratory and feeding support

O2 requirement and oedema improved

Repeat ECHO:
Borderline mildly dilated LV - EF 55-60%
Normal systolic function with apical dyskinesia

Sudden onset, pleuritic chest pain
Normal observations – O2 Sats 96% on air
ECG: ST depression V2-V6
Bloods: Hb 104, WCC 9.8, **Troponin 1305.7**
CXR: Opacification right lower zone
CTPA: Small bilateral lower lobe consolidation, no Pulmonary Embolism

Cardiology review:
Managed as suspected myopericarditis

Investigation	Result
BNP (normal <400ng/l)	1023 ng/l (antenatal)
Troponin I (normal <15.7ng/l)	Day 6 PN – 1305.7 Day 7 PN – 2170.9 Day 11 PN – 4796
CT Coronary Angiogram (CTCA)	Normal
Cardiac MRI	Mildly impaired LV systolic function, no dilation No abnormal myocardial enhancement
Autoantibody screen	Antinuclear Antibody + Anticardiolipin IgG & IgM + IgG Beta-2 Glycoprotein + Antimitochondrial negative Anti dsDNA negative Anti Smooth Muscle negative
Thrombophilia screen	Negative

Figure 2: Investigations and Results

CORONARY CARE STAY

Day 7 PN

Transfer CCU Day 8 PN

MRI Brain Day 10 PN

Cardiac MRI Day 16 PN

Further IX

MDT Discussion

Transient, short lasting episodes of confusion
CT head - NAD

Rising troponin ?cause
CTCA Day 9 PN (?SCAD) - NAD

Tiny acute infarcts in an internal watershed distribution.
No neurological deficit
Stroke team review
Aspirin commenced

Mildly impaired LV systolic function, no dilation

Antinuclear antibody + Anticardiolipin IgG and IgM + IgG Beta-2 Glycoprotein +

Obstetrician, Obstetric Physician, Stroke Physician, Cardiologist and Haematologist

Working diagnosis:
Primary Antiphospholipid syndrome (APLS)

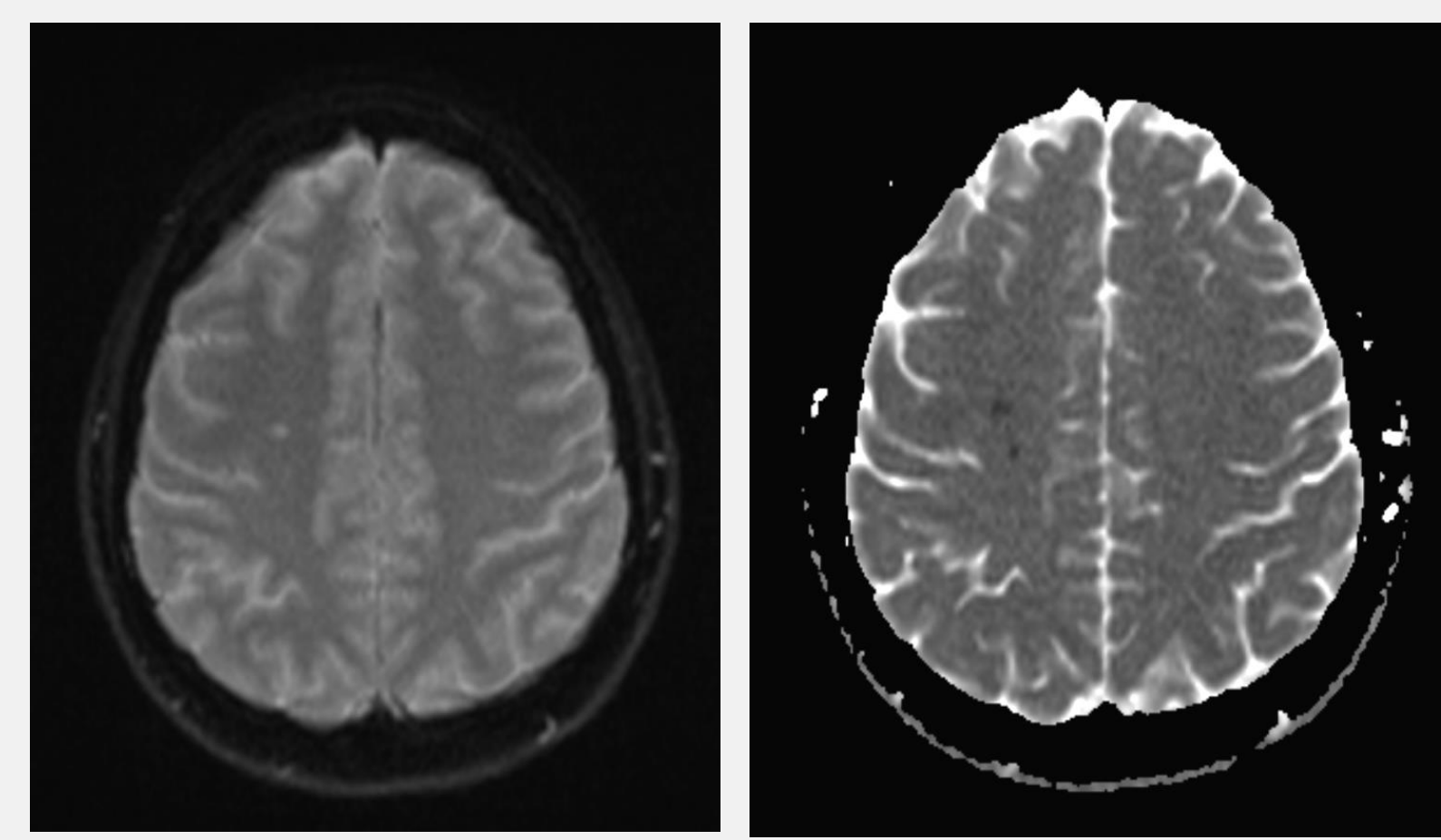


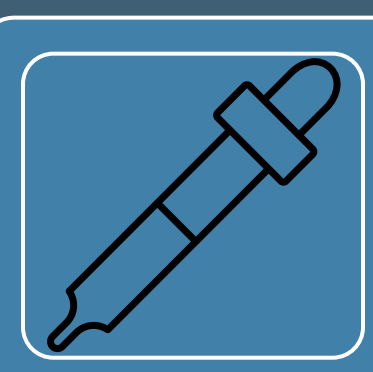
Figure 3: MRI Head – Day 10 PN
Tiny acute infarcts in the right centrum semiovale in an internal watershed distribution. No evidence of PRES or VST.

DISCUSSION



Raised troponin postpartum can be seen in:

- Myocardial infarction
- Stroke
- Aortic dissection
- Pulmonary embolism
- Thrombotic microangiopathies



Assay interference due to raised anticardiolipin antibodies can cause troponin rise.



Consideration of catastrophic APLS is important in patients presenting with APLS and multi-organ involvement, particularly microthrombosis.

CONCLUSION

This case highlights the essentiality of a nuanced approach to interpreting troponin in the context of pre-eclampsia and the importance of multidisciplinary teamwork between obstetricians, obstetric physicians, anaesthetists, cardiologists and haematologists to provide individualised, patient-centred care which is the cornerstone of obstetric medicine.

REFERENCES

1. Melchiorre K. (2014). 'Cardiovascular Implications in Preeclampsia: An Overview', *Circulation*, 130 (8). Available at: <https://doi.org/10.1161/CIRCULATIONAHA.113.00366>. (Accessed 3rd June 2025).

CASE OUTCOME

Day 16 PN
Stepped down to postnatal ward

Day 18 PN
Commenced warfarin with bridging LMWH

Day 21 PN
Discharged home with baby
Breastfeeding

5/52 PN
Obstetric medicine postnatal appointment.
All antihypertensives stopped due to persistent hypotension.
No residual limb weakness or cognitive deficit.

Plan for repeat anti-phospholipid antibody testing in 3 months