

Postpartum Microangiopathy-Thinking of Differentials

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Antepartum

- 33yrs, BMI 20.6
- Para 0, 40+6 weeks
- Low risk , no significant past history
- Spontaneous labour

Intrapartum

- Intrapartum pyrexia
- Sepsis 6 commenced - IV Augmentin
- Pathological trace- fetal tachycardia @ 180/min, reduced variability- no response to fluids
- Straight forward LSCS for failure to progress at 4 cm, pathological trace and maternal sepsis
- Birth Weight- 3160 gms
- Blood Loss - 1060 ml
- Cord pH- 7.1

Postpartum

- **Blood culture- E coli**
- Placental culture- no growth
- HVS- anaerobic growth
- MSSU- no growth

- **Differential diagnosis-**
AKI with multisystem involvement
? Sepsis driven presentation of vasculitis
? HUS
? HELLP
? DIC, severe sepsis

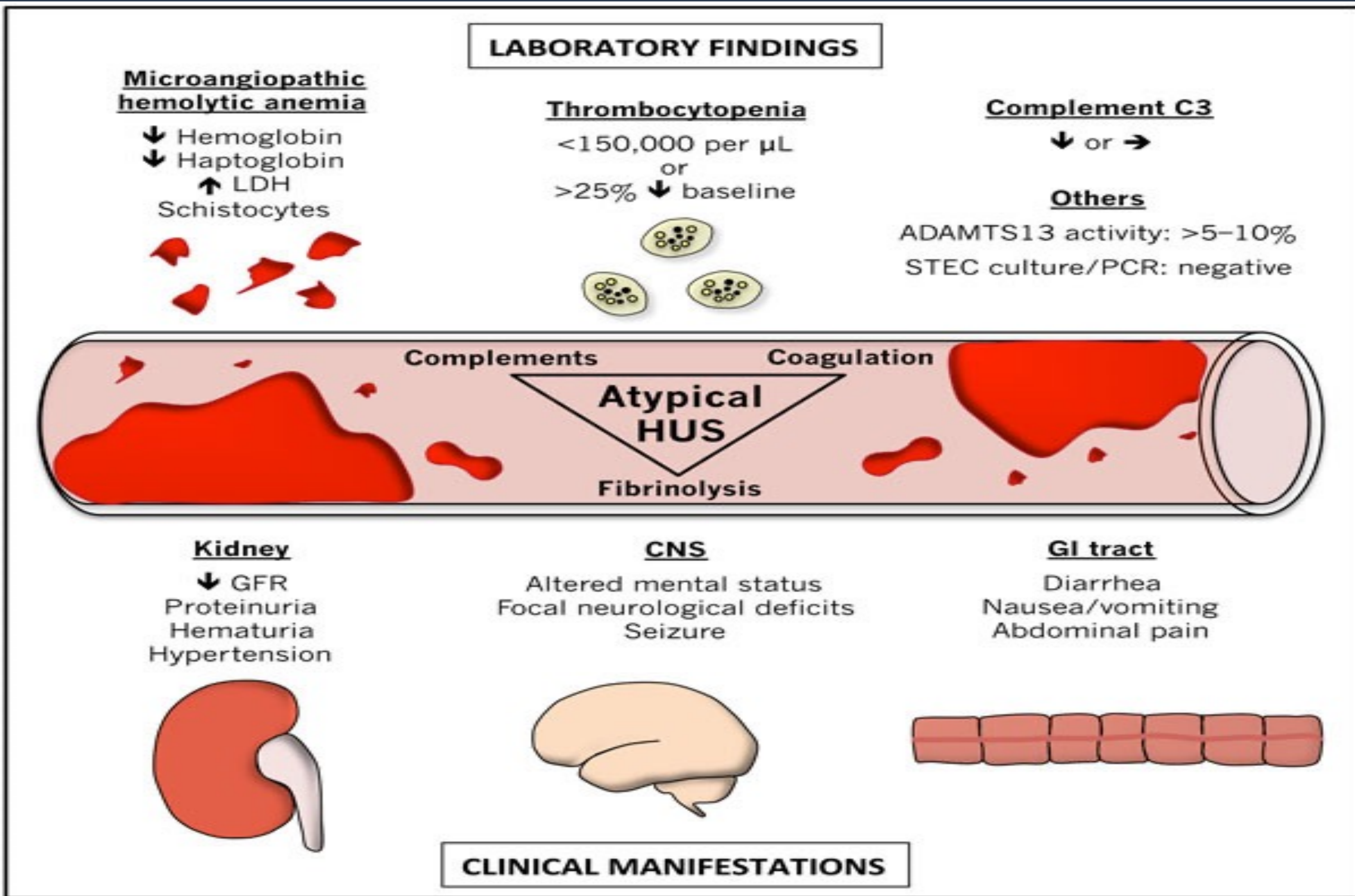
Investigations

Bloods Week 1								
	20/8 (pre-op)	20/8 (post-op)	21/8 (6hrs)	21/8 (12hrs)	22/8	23/8	24/8	25/8
Hb	126	97	86	87	78	89	82	84
Platelets	146	83	65	69	93	111	120	128
WBC	2.3	8.9	15.4	23.3	34.1	36.4	22.5	13.0
CRP	46	57	122	187		174	60	96
Lactate	3.1	4.1	2.3	2.4	1.4	1.5	1.5	1.2
LDH					1225	1101	837	742
BU	4.7	4.9	7.3	8.2	20.9	23.5	27.6	33.1
Scr	101	140	219	262	437	487	493	515
EGFR	>59	43	25	20	11	9	9	9
S bil	9	18	11	9		4	3	3
ALT	37	32	63	80		79	62	71
AST				183				
PT ratio		1.3	1.2			0.9	0.9	0.9
PTT ratio		1.4	1.3			0.9	0.8	0.7
		1.9	1.8			6.4	4.5	4.0

- Blood film- occasional red cell fragments
- PCR - 74 , Repeat - 26
- ADAMTS 13- 47.1 (low)
- ANA- neg , RF- neg
- Anti GBM- neg
- Myeloperoxidase ab – neg
- Vit 12, FA- normal
- Haptoglobin- 3.2
- C3, C4 levels- normal

Bloods Week 2						
	27/8	28/8	30/8	31/8	01/9	04/9
Hb	84	91	98	97	101	98
Platelets	240	329	502	501	520	558
WBC	20.2	18.9	12.6	12.8	9.5	6.8
CRP	207	158	62	51	31	12
Lactate		0.6	0.7		0.8	
LDH	538	521	400	338	451	270
BU			28.4	29	25	14
Scr			471	398	333	195
EGFR			10	12	15	29
S bil		3	3	4	<3	
ALT		46	33	33	32	
AST			16			

Interpretation



- Uncontrolled terminal complement activation leads to systemic thrombotic microangiopathy (TMA)
- Pregnancy can trigger aHUS
- May progress to end-stage renal disease

Thrombotic microangiopathies of pregnancy

Table 5. Clinical and laboratory features of microangiopathies of pregnancy							
Feature	Preeclampsia	HELLP	AFLP	aHUS	TTP	CAPS	SLE
Hypertension	+++	+++	+	++	+	+	++
Proteinuria	+++	++	+/-	+++	+/-	+	+++
Nausea/vomiting	+	+	++	+/-	+/-	+/-	+/-
Abdominal pain	+/-	++	++	+/-	+/-	+/-	+/-
Jaundice	+/-	+/-	++	+/-	+/-	+/-	+/-
Neurologic symptoms	+	+	+	+/-	++	++	+
Thrombocytopenia	+	+++	+	+++	+++	++	+
Hemolysis	+/-	+++	+	+++	+++	+/-	+
Raised bilirubin	+/-	+++	+++	+++	+++	+/-	+/-
Renal impairment	+/-	+	++	+++	+	++	++
DIC	+/-	++	+++	+/-	+/-	+/-	+/-
Hypoglycemia	+/-	+/-	+++	+/-	+/-	+/-	+/-
Elevated ammonia	+/-	+/-	+	+/-	+/-	+/-	+/-
Elevated transaminases	+	+++	+++	+/-	+/-	+/-	+
Peak time of onset	Third trimester	Third trimester	Third trimester	Postpartum	Second or third trimester	Anytime	Anytime

CAPS indicates catastrophic antiphospholipid syndrome; +/-, rare or absent (0%-20% of cases); +, fairly common (20%-50% of cases); ++, common (50%-80% of cases); and +++, very common or constant feature (80%-100% of cases).

Conclusion

- Laboratory parameters in HELLP syndrome begin to resolve spontaneously within 3 to 4 days postpartum
- They continue to worsen after delivery in the setting of aHUS, often progressing to renal failure and requiring dialysis (65%) or complement inhibition (35%)
- In the postpartum period, marked elevation of both serum creatinine and LDH is highly specific for aHUS and is uncommon in HELLP syndrome
- Hemolysis and renal failure may occur transiently after delivery, their persistence for >72-hour postpartum should raise suspicion for aHUS provoked by pregnancy.
- Useful to keep in mind the differential diagnosis so that timely referral can be done if required
- Importance of multidisciplinary care and team work
- Complement genetic testing may be considered

