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Biological work-up in patients with pregnancy-associated TMA	
ADAMTS13 activity ± anti-ADAMTS13 antibodies	P-TTP diagnosis is made if ADAMTS13 activity < 20%.
Serum soluble fms-like tyrosine kinase-1/Placental Growth Factor (sFlt1/PlGF)	sFlt-1/PlGF ratio > 85 before 34 weeks of gestation and (> 110 after 34 week) are strongly suggestive of preeclampsia/eclampsia or HELLP syndrome, whereas a ratio < 38 suggests an alternative diagnosis.
Complement tests. Serum levels of C3, C4, Factor H, Factor I and Factor B. Test for anti-Factor H antibodies. Sequencing of Factor H, Factor I, Membrane-Cofactor Protein, C3 and Factor B genes.	Pregnancy-associated HUS diagnosis is NOT based on complement test results[1-3]. Systemic markers of complement activation (low C3, elevated soluble C5b-9) are not synonymous of atypical HUS. Complement activation may be transient and self-limited in some pregnancy-associated TMA[4]. The detection of a pathogenic complement variant confirms retrospectively the diagnosis of complement-mediated HUS but negative genetic tests do not rule out the diagnosis of pregnancy-associated HUS[5,6] Genetics tests are very helpful for the decision to discontinue or not eculizumab[7-9].
Autoimmunity tests. Antinuclear antibodies, anti-native DNA antibodies, anti-cardiolipin antibodies, anti-b2GPI antibodies, lupus anticoagulant.	Pregnancy-associated TMA resulting from systemic autoimmune diseases require specific treatments.
Other tests Serum homocystein level. Prothrombin time, factor V, fibrinogen, D-dimers serum levels. Serum iron, vitamin B9 or B12 levels.	High homocystein serum level suggests cobalamin C deficiency-associated TMA, a diagnosis to rule out or confirm (specific treatment required)[10-12]. Disseminated intravascular coagulation is not suggestive of TMA. Consider alternative diagnoses, including acute fatty liver (predominant liver injury and coagulopathy)[13]. Deficiency in vitamin B9 and/or B12 may mimick TMA, including during pregnancy[14,15]. Deficiency in iron, vitamin B9 or B12 may explain low reticulocyte count despite hemolysis.

Supplemental Table S1: Initial biological work-up in patients with pregnancy-associated thrombotic microangiopathy (P-TMA).
Abbreviations: P-TTP, pregnancy-associated thrombotic thrombocytopenic purpura. P-HUS, pregnancy-associated hemolytic uremic syndrome.

Authors	Age (y)	History of aHUS	Complement gene pathogenic variant identified	Timing of pregnancy-aHUS (pregnancy rank)	Characteristics before eculizumab start	Time from admission to eculizumab initiation	Response to eculizumab	Neonate Status	Eculizumab Stopped	Last follow—up (duration)
aHUS during pregnancy										
Andries et al, 2017[16]	30	-	None in CFH,CFI, MCP,C3,CFB, THBD,DGKE	10 GW (11th /5 miscarriages)	Plt, 15 G/L SCr 707 µmol/L Dialysis Severe HT	4 days (Resistance to 4 PE)	At day 15 Ptl 150 G/L SCr 215 µmol/L Dialysis stopped At 4 months SCr 46 µmol/L	Healthy infant (36 GW)	-	HUS remission Normal renal function (20 months)
Mandala et al, 2014[17]	29	-	None in CFH,CFI, MCP,C3,CFB, THBD, DGKE	16 GW* (ND)	Plt 52 G/L SCr 92 µmol/L Severe HT	47 days (resistance to daily PE)	Immediate improvement of all parameters	ND	-	HUS remission SCr 62 µmol/L (ND)
Ardissino et al, 2013[18] Mussoni et al, 2014[19]	26	+ (2 y earlier)	CFH Homozygous p. Arg53Cys	17 GW (1st)	Ptlt 102 G/L PU >3g/L SCr 56 µmol/L Severe HT	6 weeks (resistance to 30 PE)	At day 5 Ptl 163 G/L Normal SCr Reduced PU	Healthy infant (38 GW)	+ (after delivery/38 GW)	ND
Demir et al, 2016[20]	17	-	None in CFH or CFI	17 GW (ND)	Plt, 52 G/L SCr 252 µmol/L	5 days (Resistance to 5 PE)	Within few days Plt 202 G/L SCr 71 µmol/L	Caesarean section at 31GW (fetal distress) Neonatal respiratory distress Then healthy infant	-	HUS remission Normal renal function (6 months)
Tsai et al, 2016[21]	22	+ Pregnancy-HUS 2 y earlier	CFH p. Arg1203Trp	22 GW (3rd) (HT during 1st pregnancy HUS at 2nd pregnancy)	Plt 56 G/L SCr 265 µmol/L	5 weeks	At day 3 Normal Plt At 3 months Normal SCr	Pre-term infant (Induced delivery/ 22 GW)	-	ND
Kourouklaris et al, 2014[22]	23	-	ND	Preeclampsia at 31 GW HUS 5 days after caesarean section (ND)	Plt 80 G/L SCr 707 µmol/L Dialysis Neurological complications	6 months (Transient benefit from PE)	At 6 weeks Plt 141 G/L SCr 247 µmol/L	ND	+ after 6 weeks**	HUS relapse 5 months after stopping eculizumab Ptl 75G/L SCr 486 µmol/L 12 months after restoring eculizumab HUS remission

Shanmugalingam et al, 2018[23]	27	-	None in MCP	32 GW Preeclampsia /HUS (1st)	Ptl 44 G/L SCr 544 µmol/L Dialysis Severe HT	3 days (Resistance to PE)	At day 3 Plt 158 G/L Dialysis stopped At day 19 SCr 105 µmol/L	Pre-term healthy infant (induced delivery at 32GW)	-	SCr 132 µmol/L HUS remission SCr 110 µmol/L (2 months)
Asif et al, 2017[24]	33	-	ND	33 GW Extensive blood loss after caesarean section /hysterectomy (ND)	Ptl, 39 G/L SCr 530 µmol/L Dialysis	~ 6 days (resistance to 5 PE)	At 2 weeks Ptl, 147 G/L SCr 300 µmol/L Dialysis stopped	In utero foetal death (33 GW)	-	HUS remission Normal renal function (6 months)
Chua et al, 2017[25]	34	-	ND	33 GW (ND)	Ptl, 88 G/L SCr 300 µmol/L Dialysis Neurological, intestinal and vascular complications	~ 6 days (Resistance to 5 PE)	At day 4 Hemolysis improved At day 40 Dialysis stopped	In utero foetal death (33 GW)	-	HUS remission Normal renal function (8.5 months)
Cravero et al, 2016[26]	33	-	None in CFH,CFI, MCP,C3,CFB No anti-CFH Ab	36 GW (1st)	Ptl 200 G/L (under PE) Anuria Dialysis	5 days (Renal resistance to PE)	At ~ 1 week SCr 62 µmol/L	Caesarean section (36 GW) (foetal distress) Healthy infant	-	HUS remission Normal renal function (9 months)
Chua et al, 2017[25]	29	-	ND	37 GW (ND)	Ptl, 70 G/L SCr 495 µmol/L	2 days (after 2 PE)	At 2.5w Hemolysis markers normalized At 1m Screat 80 µmol/L	In utero fetal death (37 GW)	+ after 3 weeks***	HUS remission Normal renal function (7 months)****
Yamaguchi et al, 2017[27]	25	-	CFH Homozygous p.R1215G	Preeclampsia at 37.5 GW HUS 2 days after caesarean section (1st)	Ptl 110 G/L SCr 177 µmol/L Neurological complications Carotid arteries stenosis	74 days (Transient improvement under PE)	At ~ 2 weeks Ptl 250 G/L SCr 72 µmol/L	Healthy infant (37.5 GW)	-	HUS remission Normal renal function (7.5 months)

aHUS after delivery										
Canigral et al, 2013[28]	32	-	None identified in CFH,CFI, MCP	At delivery (severe bleeding after caesarean section) (ND)	Plt 30 G/L SCr 390 µmol/L	3 days (resistance to PE)	At 7 days Ptl 150G/L At ~ 2 months Normal SCr	ND	+, after 6 months	HUS remission Normal renal function (6 months)****
Gately et al, 2017[29]	32	-	ND	At delivery at 40 GW (severe post-partum bleeding) (1st)	Plt 44 G/L SCr 170 µmol/L Dialysis at 4 days post-partum	6 days (renal resistance to 4 PE)	At 9 days Dialysis stopped	ND	-	HUS remission SCr 145 µmol/L (1 month)
Saad et al, 2015[30]	16	-	MCP p.T383I	1 day after delivery (39 GW) (1st)	Before PE Plt 50 G/L SCr 210 µmol/L	46 days (Plt 180 G/L SCr 124 µmol/L after 14 PE)	Maintenance of normal Plt and improved SCr	ND	-	HUS remission (ND)
Zschiedrich et al, 2013[31]	31	-	CFI p.Gly263AlafsX37	3 days after delivery (41GW) (ND)	Plt 88 G/L SCR 400 µmol/L Dialysis Delirium	18 days (resistance to 27 PE)	Within 2 weeks Plt normalized SCr 88 µmol/L	Healthy infant	-	HUS remission SCr 88 µmol/L (2.5 months)
De Sousa-Amorim et al, 2015[32]	41	-	None in CFH,CFI, MCP,C3,CFB. No anti-CFH Ab	4 days after full term delivery (1st)	Plt 105 G/L SCr 292 µmol/L Dialysis Severe HT	12 days (resistance to 5 PE)	At 4 days Plt 130 G/L Dialysis stopped At 4 weeks Plt 180 G/L SCr 97 µmol/L	Healthy infant	+ after 11 months	HUS remission Normal renal function (1 year)****
Carr et al, 2012[33]	20	-	CFH (variant ND)	7 days after caesarean section (39 GW) (ND)	Plt 28 G/L SCr 723 µmol/L Dialysis	7 days (resistance to PE)	At 2 weeks Plt normalized At 6 weeks Dialysis stopped At 12 weeks Normal SCr	ND	+, after 9 m	HUS relapse 6 months after stopping Ec Ptl 54 G/L SCr 447 µmol/L Dialysis Remission after eculizumab restoring
Delmas et al, 2013[34] Fakhouri et al, 2014[35] (patient 1)	26	-	CFH p.Lys1186Thr CFI p.Ile322Thr	1w week after full term delivery (1st)	Plt 49 G/L SCr 550 µmol/L Dialysis	3 days (resistance to PE)	At 15 days Dialysis stopped At 42 days Ptl normalized At 6 months	Healthy infant	+ after 18 m	Remission of HUS 5 y after stopping eculizumab Normal renal function 2nd pregnancy, 34 GW Plt 110 G/L, proteinuria

							SCr 75 µmol/L				Eculizumab reinitiated Caesarean section (foetal stagnation) at 36 GW Eculizumab stopped after 6 months Sustained remission and normal renal function during subsequent year
Baghli et al, 2017[36]	23	-	C3 p.Arg735Tr	2 weeks after delivery (38 GW) (1st)	Plt 177 G/L SCr 377 µmol/L Dialysis	~ 14 days (resistance to few PE)	At 4 months Plt 201 G/L SCr 123 µmol/L Dialysis stopped	ND	-	HUS remission (8 months)

Supplemental Table S2: Characteristics and outcome of 20 individual cases of pregnancy and postpartum-associated atypical HUS treated with eculizumab. Fifteen additional patients with pregnancy-aHUS treated with eculizumab are indicated in 3 series: 4 patients in Bruel et al (2017)[5], 10 in Huerta et al (2018)[6] and 1 in Gaggl et al (2018)[37], with an excellent response in all. Eculizumab discontinuation was reported in 7 patients (Huerta 2018) after a median time of 10 months (IQR: 6,11), including 2 carriers of pathogenic variants. One of the patients with a pathogenic variant (Hybrid CFH) had recurrence of HUS after 7 months, the other was relapse-free after 481 days follow-up. One of the patients without variant had recurrence 5 months after discontinuation. Both patients with recurrence were in remission after restoring eculizumab treatment.

Abbreviations: Ab, antibodies; aHUS, atypical haemolytic uremic syndrome; CFB, complement factor B; CFH, complement factor H; CFI, complement factor I; d, day; Ec, eculizumab; GW, gestational weeks; m, month; MCP, membrane cofactor protein (CD46); ND, not documented; PE, plasma exchanges; Ptl, platelet count; Screat, serum creatinine; THDB, thrombomodulin.

*, The patient experienced severe bleeding after caesarean section (17 GW). **, the patient received 6 doses of eculizumab. ***, the patient received 4 doses of eculizumab.****, follow-up after eculizumab discontinuation.

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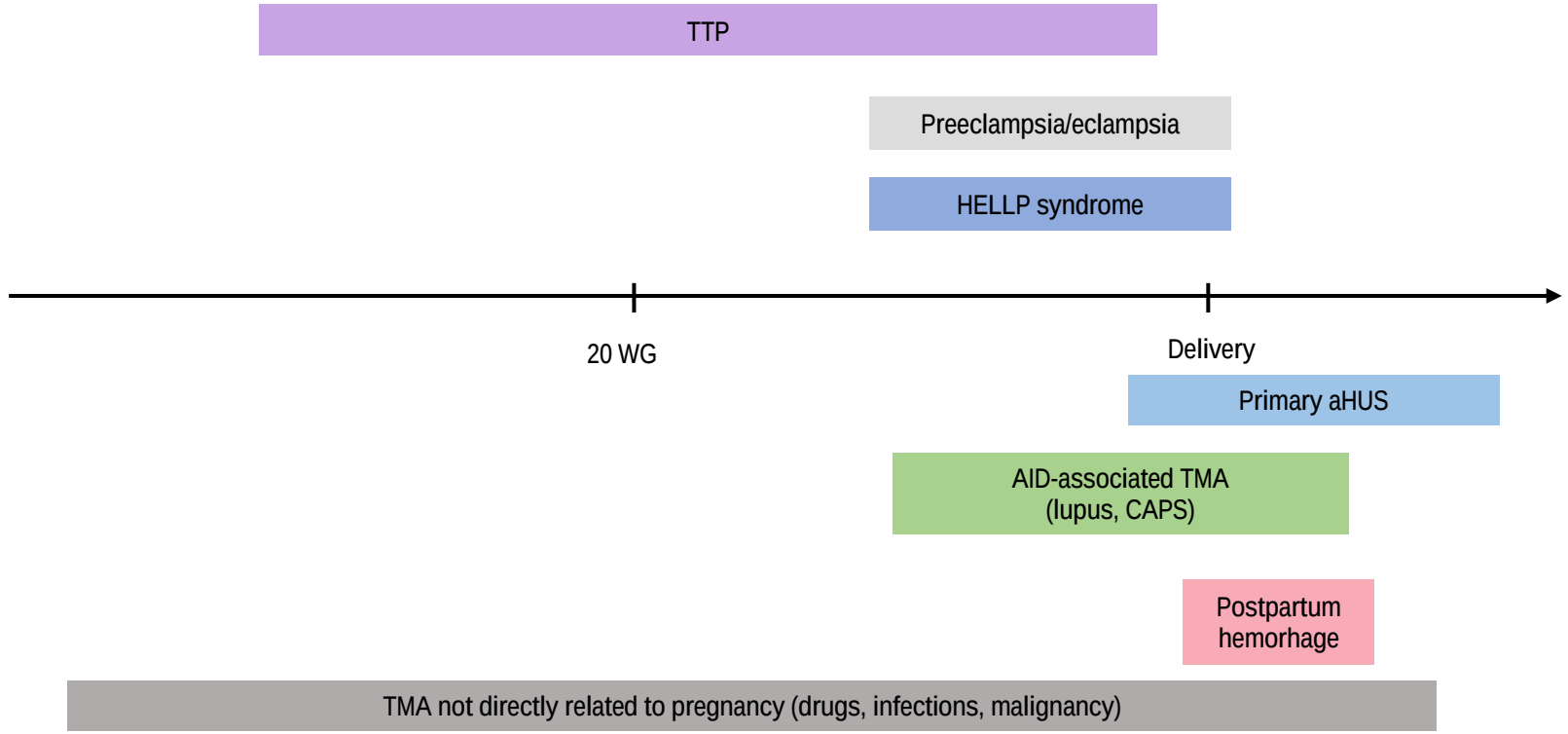

Supplemental Figure S1: Timing during pregnancy and postpartum of the main causes of pregnancy-associated thrombotic microangiopathies (TMA).
In a patient presenting with TMA before 20 weeks of gestation, the most likely diagnosis is TTP. The differential diagnosis aims to rule out the less likely diagnosis, particularly aHUS and autoimmune diseases-associated TMA. In a patient presenting with TMA during pregnancy after 20 weeks of gestation, PE/eclampsia and HELLP syndrome are the most likely diagnoses. In a patient presenting with TMA in the postpartum of an uneventful pregnancy, the most likely diagnoses are PE/E and aHUS. TMA that develops several weeks after delivery is highly suggestive of aHUS. Other differential diagnoses such as HELLP, postpartum hemorrhage, sepsis, TTP, autoimmune diseases exacerbated by pregnancy (although disorders may be inter-linked, e.g. CAPS following HELLP syndrome[38]) or TMA not directly related to pregnancy are less likely.

Abbreviations: TTP, thrombotic thrombocytopenic purpura. HELLP, Hemolysis, Elevated Liver enzymes and Low platelet count. aHUS, atypical haemolytic uremic syndrome. AID, auto-immune diseases.

Consensus process

Fadi Fakhouri, Marie Scully and Vassilis Tsatsaris conceived the idea of an international consensus on pregnancy-associated thrombotic microangiopathy and established the framework of the manuscript. All participants were invited to join the working group (The International Working Group on Pregnancy-Related Thrombotic Microangiopathies) based on their internationally recognized expertise in the field of pregnancy and thrombotic microangiopathies.

Marie Scully, Paul Coppo and Agnès Veyradier reviewed the relevant literature and wrote the draft of the section related to the diagnosis and treatment of pregnancy-associated thrombotic thrombocytopenic purpura.

Miquel Blasco, Fadi Fakhouri and Véronique Frémeaux-Bacchi reviewed the relevant literature and wrote the draft of the section related to the diagnosis of pregnancy-associated hemolytic uremic syndrome.

Giuseppe Remuzzi and Marina Noris reviewed the relevant literature and wrote the draft of the section related to the treatment of pregnancy-associated hemolytic uremic syndrome.

Chantal Loirat and Fadi Fakhouri wrote the draft of the section related to the counselling of a patient with a history of atypical HUS.

Vassilis Tsatasris and Norbert Winer reviewed the relevant literature and wrote the draft of the sections related to preeclampsia/eclampsia and HELLP syndrome.

François Provôt reviewed the relevant literature and wrote the draft of the sections related to renal cortical necrosis in the setting of pregnancy.

Miquel Blasco designed Figure 1 and Fadi Fakhouri the remaining Figures.

The manuscript was circulated several times among all the authors who reviewed it and included substantial modifications. All authors have approved the initial and revised versions of the manuscript.