

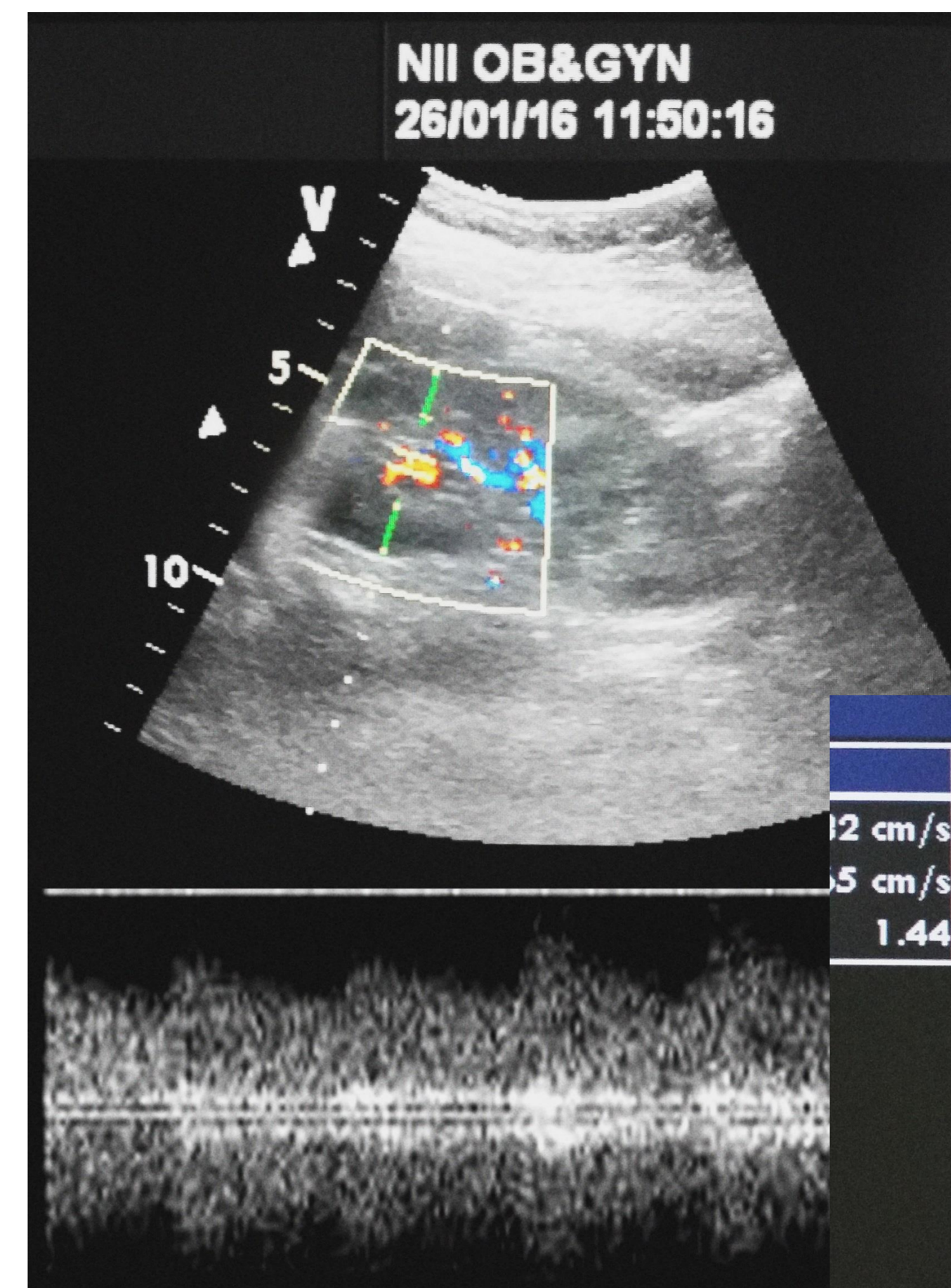


FEATURES OF INTRARENAL BLOOD FLOW (IBF) IN PATIENTS WITH PREECLAMPSIA (PE), HELLP-SYNDROME AND PREGNANCY-ASSOCIATED ATYPICAL HEMOLYTIC UREMIC SYNDROME (P-AHUS)

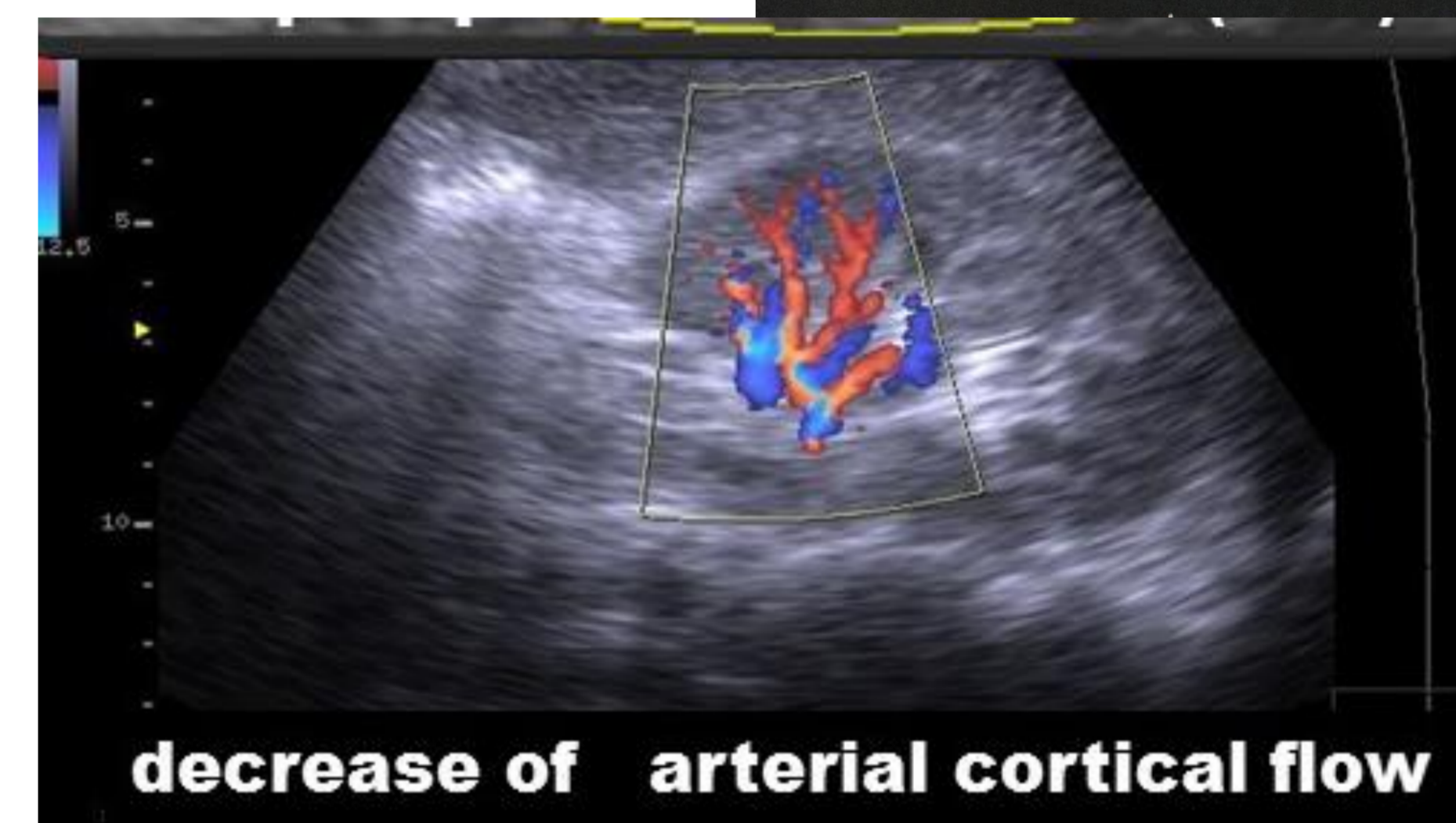
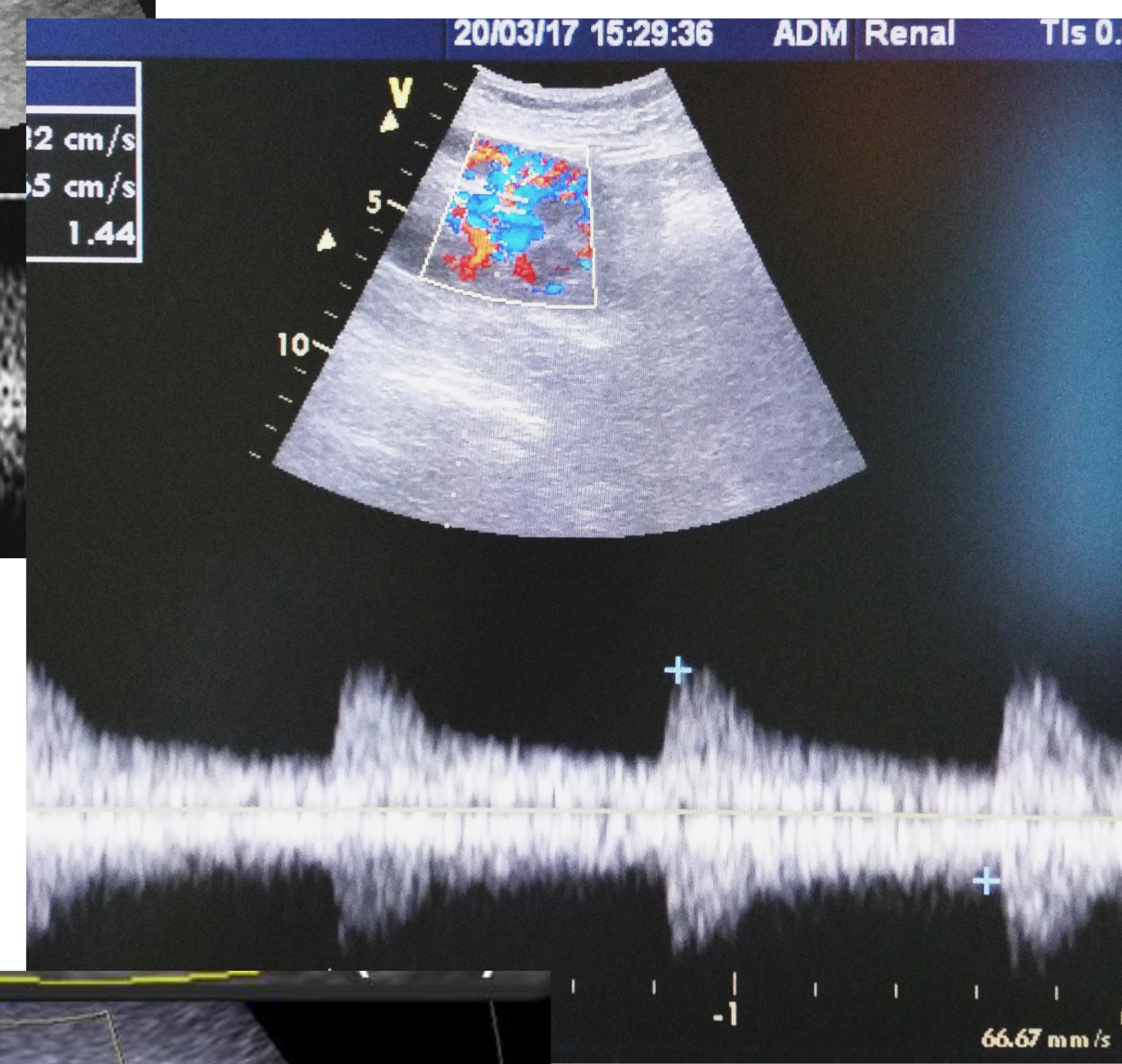
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A number of conditions in pregnancy present with microangiopathic hemolytic anemia and thrombocytopenia (MAHAT), including HELLP-syndrome. Recent evidence and clinical similarities suggest a link to P-aHUS, but we have no early non-invasive method for verification TMA. HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets) is a severe variant of PE that leads to severe morbidity and mortality to both the mother and fetus. Delivery is the treatment of choice of PE and HELLP, but can lead to progression in case of aHUS. PaHUS - life-threatening disease with uncontrolled complement activation, resulting in complement-mediated thrombotic microangiopathy (TMA), defined by the occurrence of microangiopathic hemolytic anemia and thrombocytopenia without ADAMTS13 deficiency. Triggered by pregnancy women develop the syndrome, leading to a disastrous hemolytic disease characterized by diffuse endothelial damage and platelet consumption. This disease requires prompt diagnosis and therapy. An estimated incidence of P-aHUS is 1 in 25,000 pregnancies. The estimated PaHUS-mortality is 30%. Diagnostic of early TMA-signs (confirmed!!) can prevent PaHUS-development due to quickly treatment.



arteriovenous fistulas



AIMS: to investigate renal perfusion in patients with PE, HELLP and PaHUS by Doppler ultrasonography (DUS)

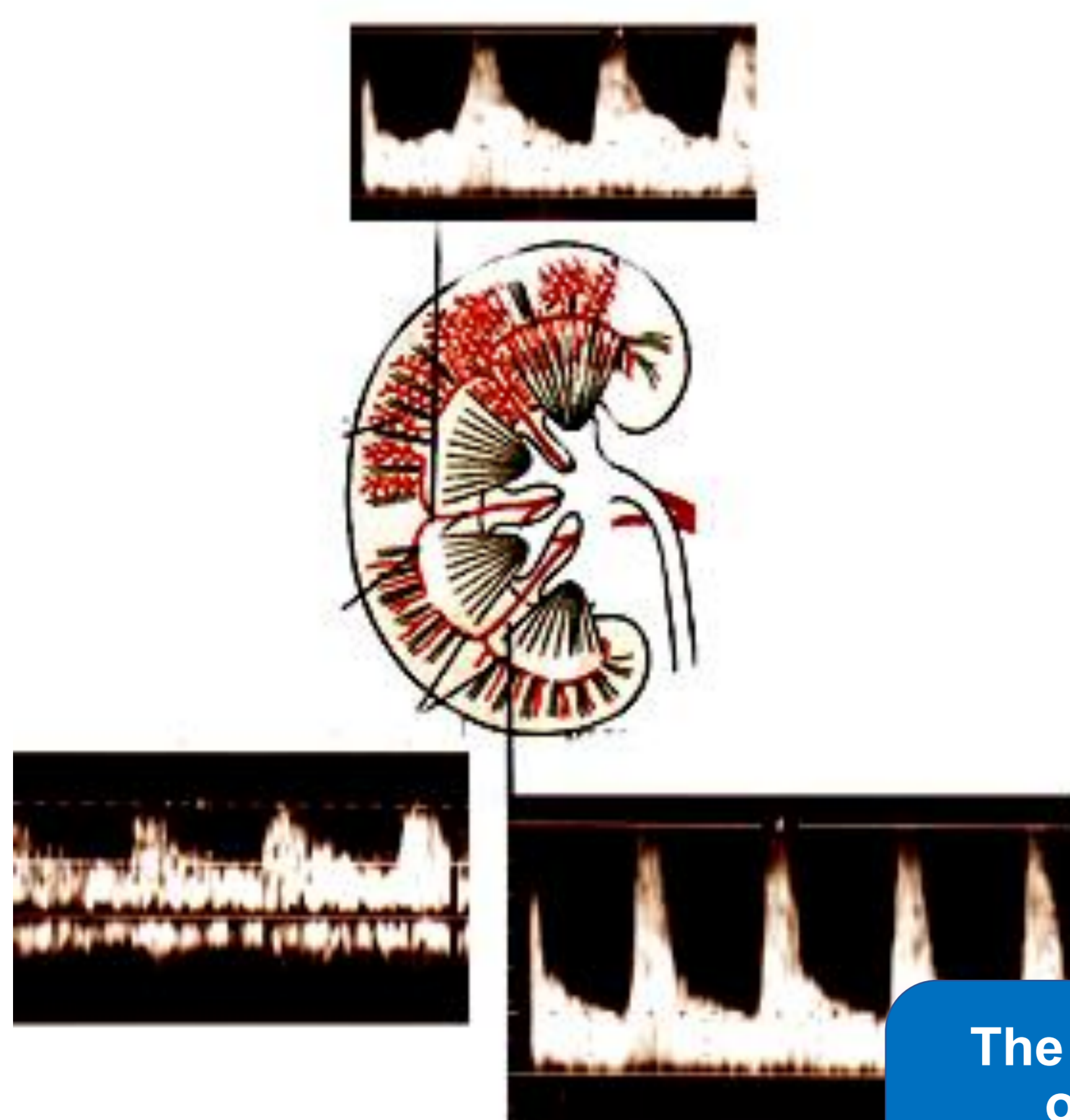
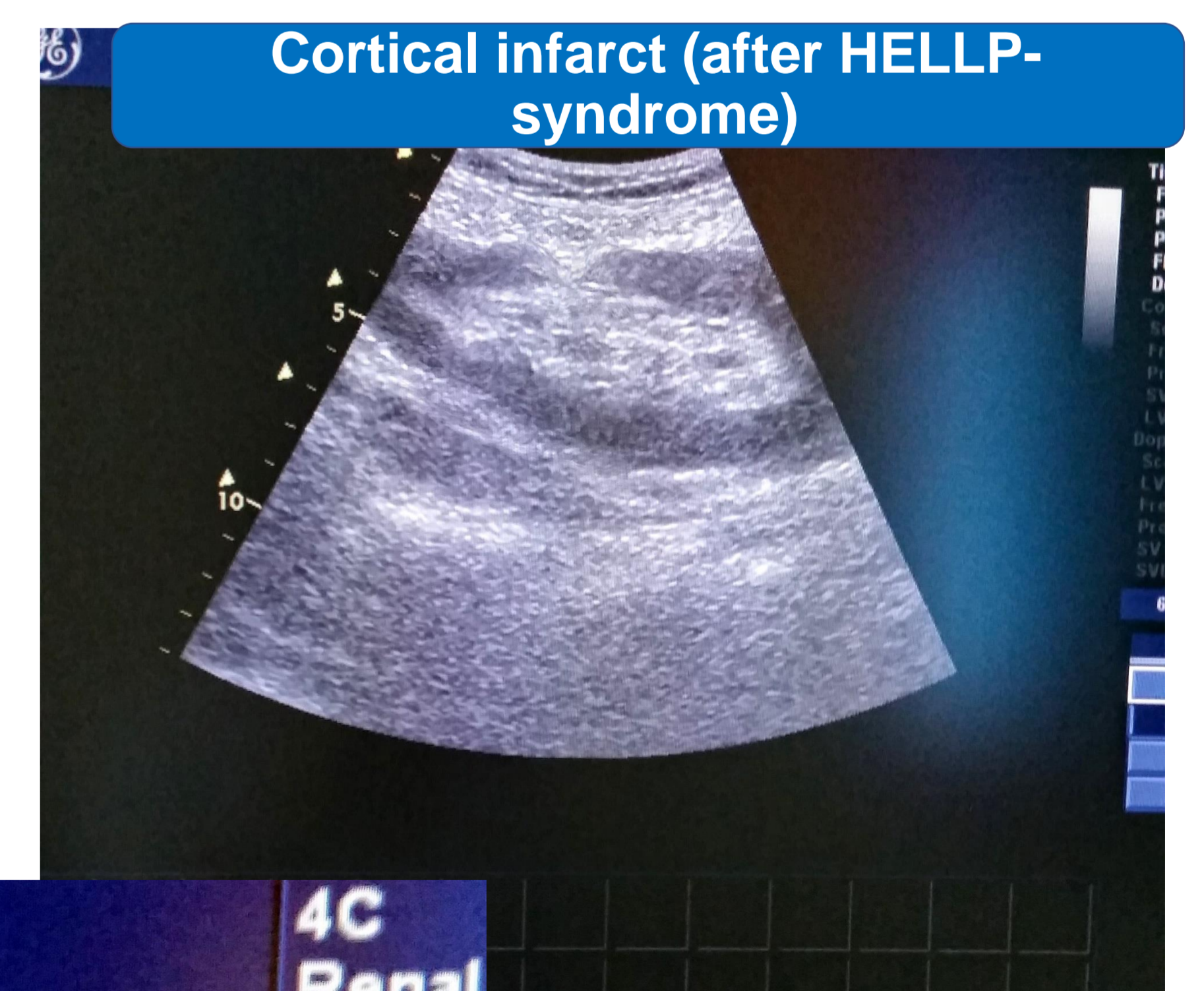
METHODS: 54 pregnant 21-46 years: 24 severe PE (24-36 weeks)-gr.1, 12-HELLP-syndrome (27-34)-gr.2, 4 P-aHUS (26-39 weeks)-gr.3, 12 volunteers (gr.4) 38-40 weeks. Severe PE diagnosed in accordance with the WHO criteria of 2008, HELLP-Tennessee criteria, aHUS- PaTMA without ADAMTS 13 deficiency, progressive after delivery. IBF was investigated by DUS (VIVID7): systolic (Vps) and diastolic (Ved) velocities (m/sec) in segmental, interlobar (IA) and arcuate (AA) arteries. $RI = (Vps - Ved) / Vps$

RESULTS: There was no any difference in Vps and RI in renal and segmental arteries in patients of all groups. We noted decrease of arterial and increase the venous flow of IA, AA (gr. 1-3) with maximal decrease/absence of arterial flow (gr3). Gr.1-3 irregular blood flow in distal renal vessels. 10/24 gr. 1 and 5/12, 2/4 -arteriovenous fistulas (AVF), 3/24, 1/12, 1/4 spleno-renal fistulas (SRF)

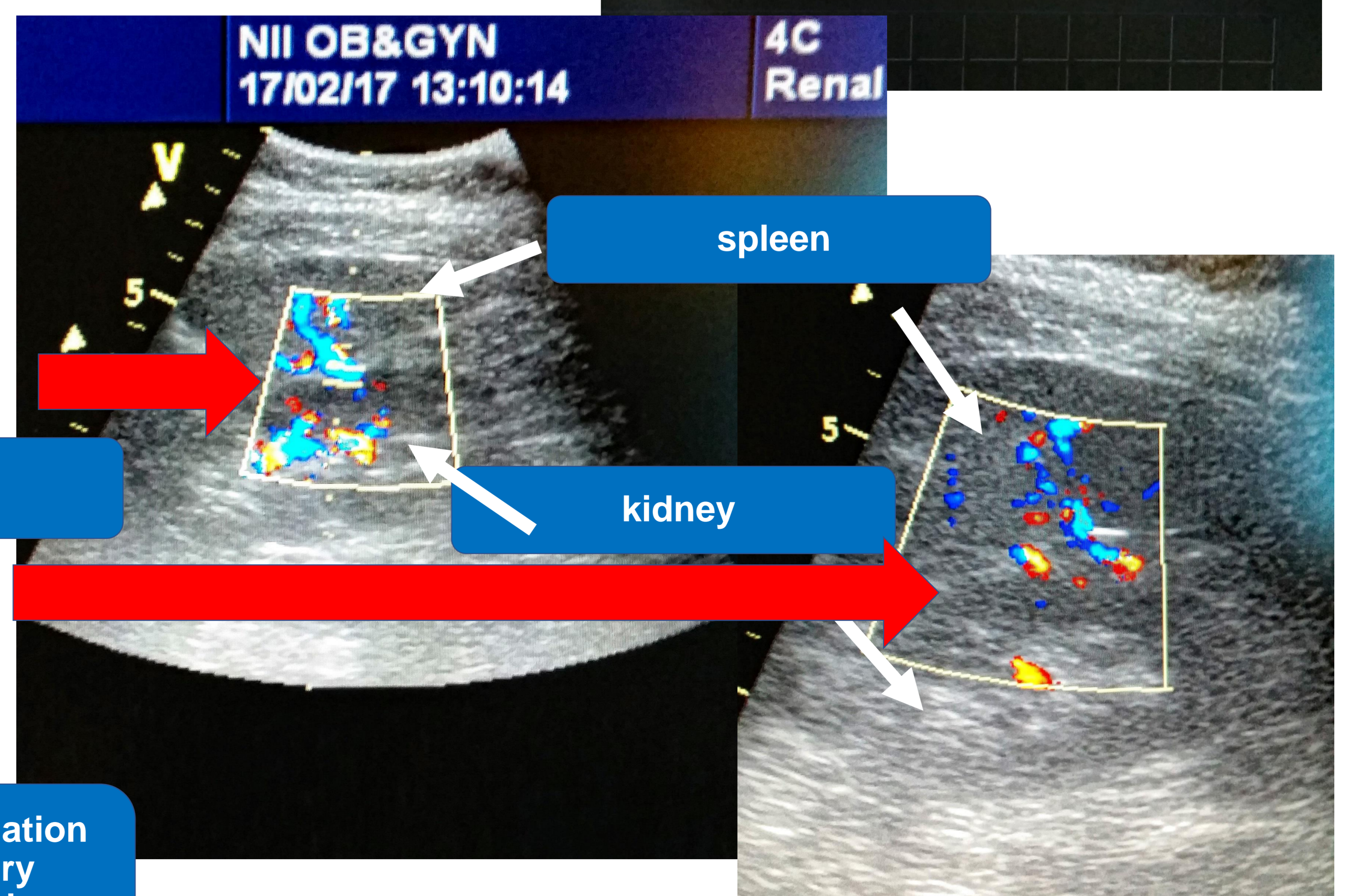
	Gr1(PE)	Gr2(HELLP)	Gr.3(PaHUS)	Gr4(volunteers)	
IA	Vps	0,56±0,1	0,42±0,04	0,17±0,03	0,38±0,07
p1,2, 2,4<0,05, p1,3, p 2,3 ,3,4<0,01					
RI max		0,72±0,035	0,9±0,05	0,59± 0,04	
RI min		0,43± 0,033	0,56±0,02		
AA	Vps	0,30±0,08	0,29±0,03	0,07±0,01	0,32±0,1
p1,4, 2,4, 3,4 <0,01					
RI max		0,56±0,024	0,89±0,02	0,58 ± 0,04	
RI min		0,48± 0,02	0,6±0,03		

Conclusions:

decrease of arterial flow - DUS-marker of renal ischemia. The "patchy" RI suggests the presence of occlusive lesions and glomerular endotheliosis - swallow endothelial cells (high RI) and arteriovenous bypassing in more distal vessels (low RI). AVF and SRM - adaptive phenomena to renal ischemia. DUS - early non-invasive method for verification PA-TMA.



spleno-renal fistulas



The "patchy" RI suggests regular alternation of ischemic lesions and compensatory hyperfiltration - the presence of occlusive lesions and glomerular endotheliosis