Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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Inflammation-Driven Pro-Thrombotic State in Hemodialysis: The Functional Role of the VWF/ADAMTS13 Axis

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Objectives : Hemodialysis (HD) patients paradoxically experience both bleeding tendencies and thrombotic events, including vascular access thrombosis. According to Virchow's triad, endothelial injury, hypercoagulability, and hemodynamic changes are key drivers of thrombosis. This study evaluates the pro-thrombotic state by assessing the von Willebrand factor (VWF)/ADAMTS13 axis in relation to chronic inflammation and endothelial injury.
Methods : We retrospectively analyzed 90 patients on maintenance HD. Endothelial injury was evaluated by measuring VWF antigen (VWF:Ag), VWF ristocetin cofactor activity (VWF:RCo), ADAMTS13 activity, and thrombomodulin (TM). High-sensitivity CRP and D-dimer were measured to assess chronic inflammation and active thrombogenesis. Patients with possible chronic DIC due to aortic aneurysm (D-dimer ≥ 10.0 μg/mL) were excluded from correlation analyses (n = 3).
Results : Patients exhibited severe endothelial injury. Notably, 62.2% and 38.9% of cases exceeded the upper normal reference range for VWF:Ag (median 171.7%) and VWF:RCo (median 145.7%), respectively. Simultaneously, 48.9% exhibited abnormally diminished ADAMTS13 activity (median 51.3%). Strictly normal hepatic markers confirmed that ADAMTS13 deficiency is independent of liver dysfunction. Elevated CRP correlated with increased VWF:Ag ($r = 0.34$, $p < 0.01$) and decreased ADAMTS13 ($r = -0.38$, $p < 0.01$), evidencing inflammation-driven damage. Although markedly elevated TM (median 33.5 TU/mL) confirmed baseline damage, it showed no correlation with D-dimer ($r = -0.06$, $p = 0.61$). Conversely, reduced ADAMTS13 significantly correlated with increased D-dimer ($r = -0.31$, $p < 0.01$), linking the VWF/ADAMTS13 imbalance to thrombin generation. Despite this pro-thrombotic profile, vascular access intervention frequency showed no correlation with these markers, suggesting complex compensation involving uremic platelet dysfunction and potential qualitative VWF alterations, such as multimer loss.
Conclusions : HD patients exist in a precarious, highly pro-thrombotic state driven by chronic inflammation and endothelial injury, both of which induce the VWF/ADAMTS13 imbalance. This fulfills Virchow's triad, whereas qualitative changes like multimer loss might paradoxically mitigate overt clinical thrombosis.

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Table 1. Patient Characteristics and Biomarker Profiles (N=87)

Parameter	Median (IQR) / n (%)	Abnormal Rate (%)	Reference / Cut-off
Patient Characteristics			
Age (years)	65.0 (54.5 - 74.0)	-	-
Male sex, n (%)	46 (52.9 %)	-	-
Dialysis vintage (months)	88.0 (40.0 - 180.5)	-	-
Blood type (A / B / O / AB / Unknown)	33 / 16 / 25 / 10 / 3	-	-
Hepatic Function			
AST (U/L)	12 (8 - 16)	-	> 40
ALT (U/L)	10 (9 - 13)	-	> 45
Total Bilirubin (mg/dL)	0.3 (0.2 - 0.3)	-	> 1.2
Endothelial & Coagulation Profile			
VWF:Ag (%)	171.7 (138.8 - 216.5)	62.1 %	≥ 150
VWF:RCo (%)	145.7 (104.5 - 200.7)	37.9 %	≥ 170
ADAMTS13 Activity (%)	51.3 (41.4 - 60.0)	47.1 %	< 50
Thrombomodulin (TU/mL)	33.5 (28.4 - 39.0)	96.5 %	> 22.7
D-dimer (μg/mL)	1.6 (1.0 - 2.1)	66.7 %	> 1.0

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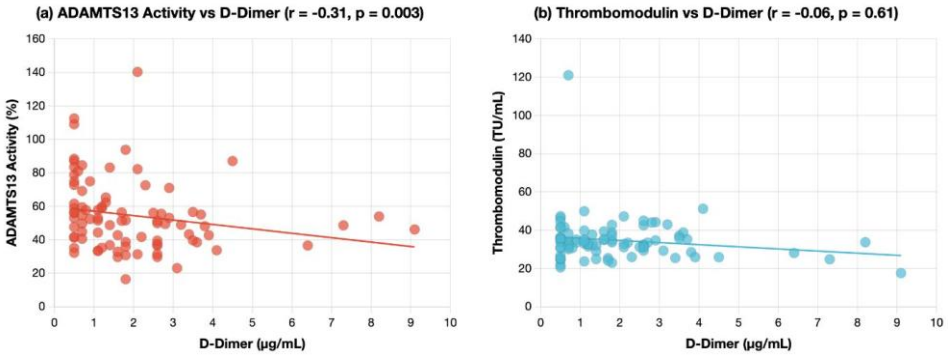

Figure 1. Correlation between Active Thrombogenesis and Endothelial Markers