LETTER TO THE EDITOR

Recombinant von Willebrand Factor (vonicog alfa) reduces platelet inhibition caused by antiplatelet drugs and has potential as an acute haemostatic agent

Authors

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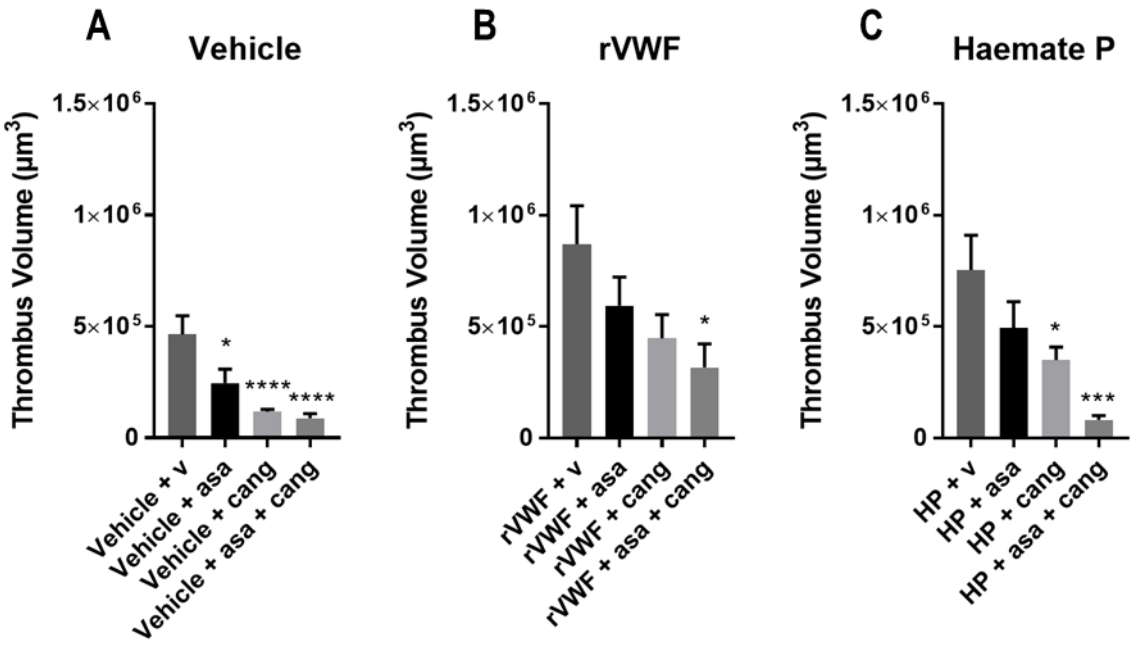
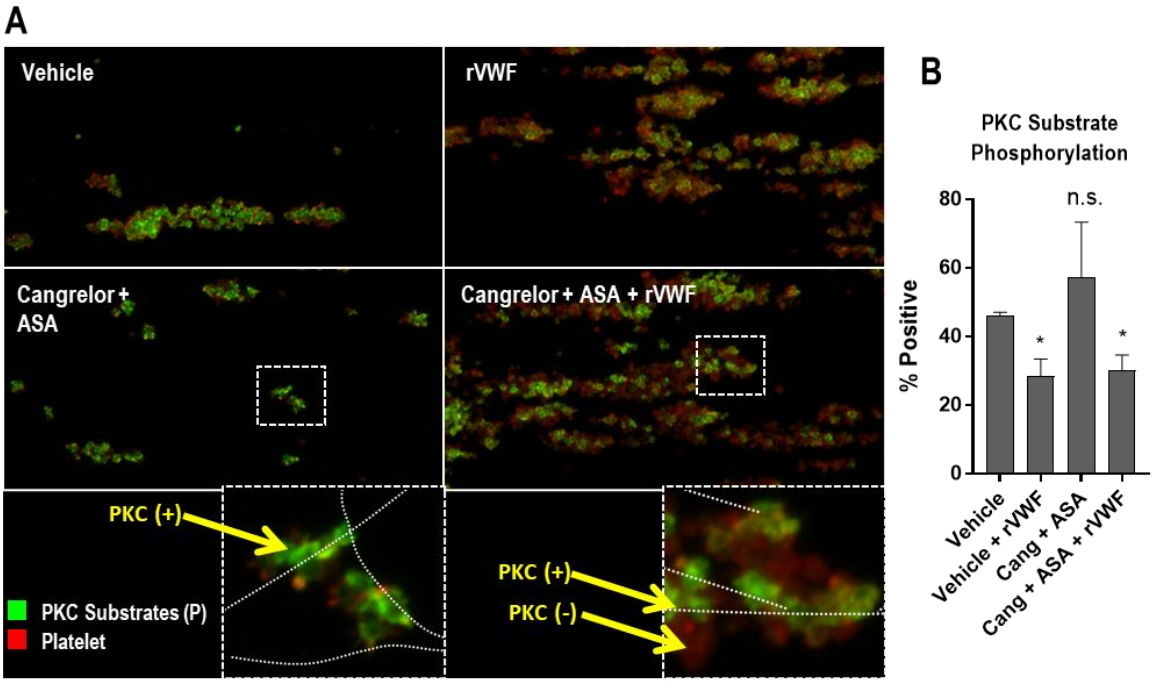
	Patients (n=22)
Age	66 (44 – 81)
Sex (Male)	15 (68%)
ASA only	15 (68%)
P2Y12 antagonist only	4 (18%)
Dual antiplatelet therapy*	3 (14%)
Hypertension	16 (72%)
High Cholesterol	11 (50%)
Diabetes	12 (56%)
Ischemic Heart Disease	19 (86%)
Smoker	6 (27%)
Haemoglobin (g/L), median (range)	142 (106 – 170)
Platelet count (x109/L), median (range)	233 (177 – 322)

* ASA plus clopidogrel, ticagrelor or prasugrel

Supplemental Table 1. Patient characteristics



Supplementary Figure 1. Effects of rVWF and non-recombinant VWF products on thrombus formation following antiplatelet treatment. Thrombus volumes from healthy donor blood pre-treated with cangrelor (1 µM), ASA (100 µM), or both, and treated with vehicle, 1U/ml rVWF or 1U/ml Haemate P. Perfusion was at 1000 s^{-1} over type I collagen for 6 minutes. Bars represent the mean thrombus volume ± s.e.m. *p < 0.05, **p < 0.01, ***p < 0.001, ****p < 0.0001. 2-way ANOVA.



Supplementary Figure 2. rVWF enables platelets with low levels of activatory signalling to join thrombi. A) Confocal fluorescence images of platelets (red) stained with phosphorylated PKC substrate antibody (green) after perfusion over type I collagen for 60 seconds. Yellow arrows indicate platelets positive or negative for PKC substrate phosphorylation; white dashed lines highlight collagen fibres in expanded views (bottom panels). B) Quantification of the percentage of aggregate volume staining positive for PKC substrate phosphorylation. *$p < 0.05$. 1-way ANOVA.