Supplementary Fig. S1 Device setup and flow conditions. Blood was loaded into the 8 wells and a syringe pump was used to apply negative pressure to perfuse blood over the surface trigger inside the 8 parallel channels (A). The outlet flow rates were set at 2 µL/min (B) and 20 µL/min (C) to achieve a venous pressure drop ($\Delta P/L = 19 \text{ mm Hg/mm-clot}$) and an arterial pressure drop ($\Delta P/L = 163 \text{ mm Hg/mm-clot}$), respectively.
Supplementary Fig. S2  Fibrinolysis is indispensable for the neutrophil extracellular trap (NET)-promoting effect of tissue plasminogen activator (tPA). The addition of ε-aminocaproic acid (εACA) to tPA-treated blood restored fibrin deposition (A) to the control level. Neutrophil accumulation (B) and total NET generation (C) were elevated when both tPA and εACA were present but not as much compared with clots treated with tPA alone. Normalized NET generation (D) in tPA-treated clots was twice as high as the level in control and combo-treated clots.
Supplementary Fig. S3 Gly-Pro-Arg-Pro (GPRP) and tissue plasminogen activator (tPA) do not affect shear-induced NETosis in the absence of thrombin under arterial pressure drops. After 45-minute arterial perfusion of D-Phe-Pro-Arg-CMK (PPACK) whole blood (WB) over collagen surfaces with or without tPA/GPRP, no differences were observed among the three conditions in either NET or NET/neutrophil signals.