Mass list

Calculated m/z window
349.5–375.5
374.5–400.5
399.5–412.5
411.5–424.5
423.5–436.5
435.5–448.5
447.5–460.5
459.5–472.5
471.5–484.5
483.5–496.5
495.5–508.5
507.5–520.5
519.5–532.5
531.5–544.5
543.5–556.5
555.5–568.5
567.5–580.5
579.5–592.5
591.5–604.5
603.5–616.5
615.5–628.5
627.5–640.5
639.5–652.5
651.5–664.5
663.5–676.5
675.5–688.5
687.5–700.5
699.5–720.5
719.5–740.5
739.5–760.5
759.5–780.5
779.5–800.5
799.5–820.5
819.5–840.5
839.5–860.5
859.5–880.5
879.5–900.5
899.5–950.5
949.5–1000.5
999.5–1050.5
1049.5–1100.5
1099.5–1150.5
1149.5–1200.5
1199.5–1650.5

Supplementary Table S1

Protein name	Function, with focus on potential associations with thrombosis
Alpha-1-acid glycoprotein 1	Inflammatory reactant that has been shown to activate platelets directly, which in turn can contribute to thrombogenesis[1]
Alpha-1-acid glycoprotein 2	Inflammatory reactant that has been shown to activate platelets directly, which in turn can contribute to thrombogenesis[1]
Alpha-1-antichymotrypsin	No identified association with thrombosis. Inflammatory reactant[2]
Apolipoprotein A-I	Higher levels have in vitro shown to inhibit clot formation and stability as well as platelet activation, i.e., lower levels equal more thrombosis and vice versa[3]
Apolipoprotein E (Apo-E)	Apolipoproteins, including ApoE, have been linked to arterial thrombosis. Yet, association have also been suggested with venous thromboembolism[4]
CD44 antigen	Adhesion molecule expressed, e.g., in endothelial cells, immune cells and platelets. Platelets express higher levels of CD44 when activated, increasing their "stickiness," which has been suggested to increase risk of thrombosis,[5] potentially by modulating effects of tissue factor[6]
CD59 glycoprotein (1F5 antigen)	A potent inhibitor of the complement membrane attack complex, inhibiting complement-mediated hemolysis. Patients with CD59 deficiency suffer from a disease resulting in hemolytic anemia and thrombosis. When expressed CD59 inhibits platelets from complement mediated attack, "protecting" platelets[7]
Golgi membrane protein 1	No identified association with thrombosis. Has been described as a cellular response protein to viral infection[8]
IgGFc-binding protein	Previously described to be upregulated in microparticles from activated platelets in patients with deep venous thrombosis.[9] No known function associated with thrombosis
Insulinlike growth factor-binding protein 2	Has been linked to platelet activation, which in turn can participate in thrombogenic pathways[10]
Intercellular adhesion molecule 1 (ICAM-1)	Endothelial transmembrane glycoprotein shown to be upregulated in inflammation and thrombosis, with a predilection for pulmonary vasculature. Suggested to be associated with thrombogenic pathways in endothelial injury, COVID-19, in antiphospholipid syndrome and suggested as a drug therapy target to direct antithrombotic therapy[11–13]
Leukocyte Ig-like receptor subfamily A member 3	No identified association with thrombosis. Acts as soluble receptor for class I MHC antigens. Binds with high affinity to the surface of monocytes, leading to abolished LPS-induced TNF-alpha production by monocytes[8]
Low-affinity Ig gamma Fc region receptor III-A	Its importance in heparin-induced thrombocytopenia, a prothrombotic adverse drug effect, has been well documented. In addition, it has been proposed to be involved in severe dengue infections[14,15]
Macrophage mannose receptor 1	Has a role in macrophage endocytosis as well as antigen processing and presentation. Have previously been shown to be associated with uptake, and potentially presentation, of ADAMST13 as well as associated with thrombotic thrombocytopenic purpura[16]
Neutrophil defensin 1	Neutrophil alpha defensins have been shown in vitro to accelerate fibrin polymerization, to increase fiber density and branching, to incorporate into nascent fibrin clots, and to impede fibrinolysis. Once activated by the intrinsic pathway (through exposed endothelial collagen), neutrophils excrete neutrophil extracellular traps containing, for instance, alpha defensins, which have been shown to be a potential link between innate immunity and thrombosis[17–19]
Oncoprotein-induced transcript 3 protein	No identified association with thrombosis. May be involved in hepatocellular function and development[8]
Phosphatidylinositol-glycan-specific phospholipase D	No identified association with thrombosis. Hydrolyzes the inositol–phosphate linkage in proteins anchored by phosphatidylinositol glycans (GPI-anchor), releasing these proteins from the membrane[8]
Plastin-2	Actin-binding protein. Suggested to be a negative regulator of megakaryocyte platelet production, meaning high levels could cause thrombocytopenia[20,21]
Polymeric Ig receptor	No identified association with thrombosis. On its own (free form), it can act as a nonspecific microbial scavenger to prevent pathogen interaction with epithelial cells. When bound mediates selective transcytosis of polymeric IgA and IgM across mucosal epithelial cells. Previously shown to be elevated in released microparticles in patients with deep venous thrombosis[8]
Trans-Golgi network integral membrane protein 2	Involved in regulating membrane traffic to and from trans-Golgi network. Have been identified in activated platelets by mass spectrometry, at low levels, and suggested to be one of several potential "switch" candidates in platelet activation[22]

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