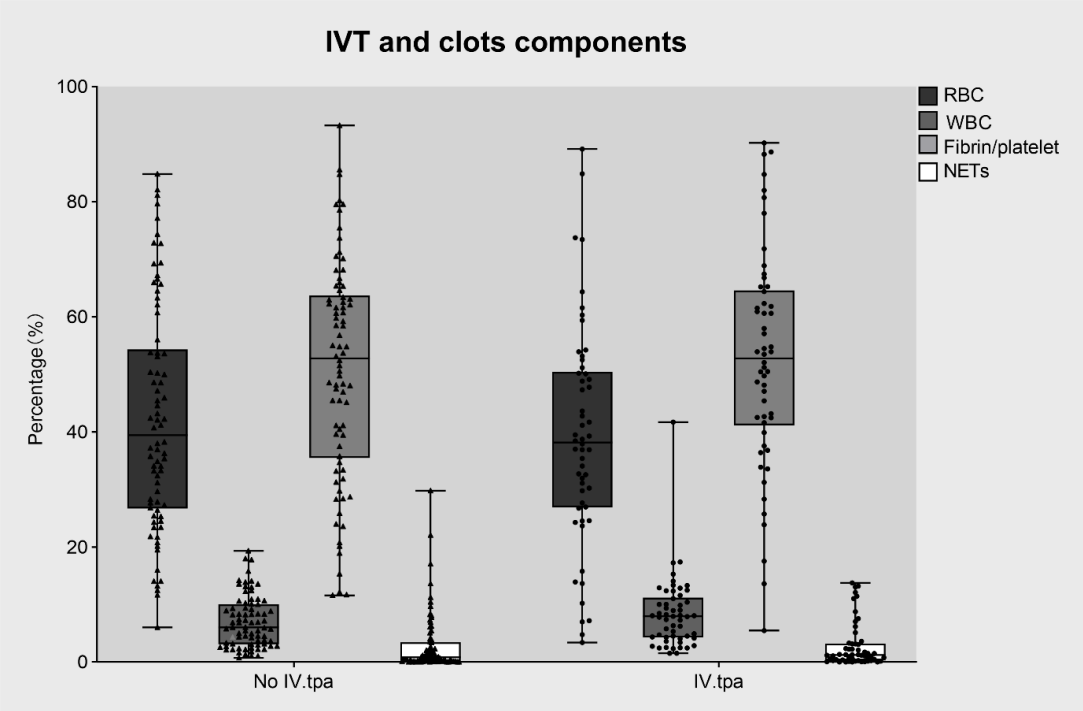
Supplementary Table 1. Comparison of baseline demographic, clinical, and procedural characteristics between patients with and without FPE.

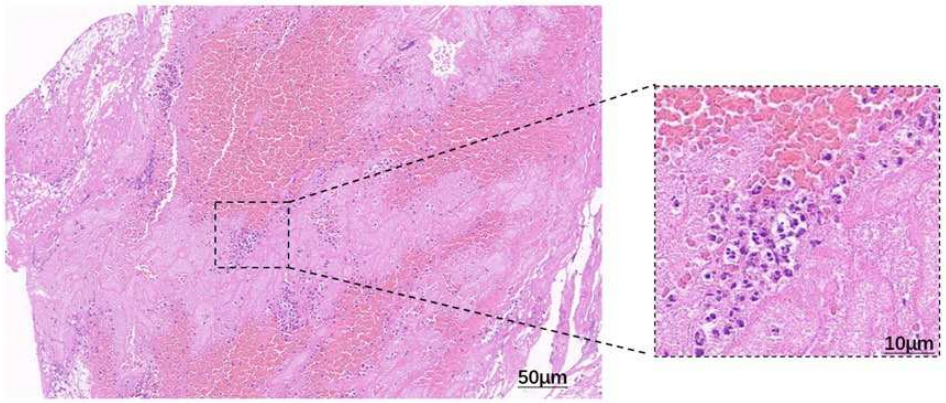
	Non-FPE(n=79)	FPE (n=53)	p-value
Age, years, mean (±SD)	66.4±13.9	63.5±11.8	0.222
Female, n (%)	28(35.4)	17(32.1)	0.689
Vascular risk factors, n(%)			
Atrial fibrillation	37(46.8)	19(35.8)	0.211
Hypertension	45(57.0)	35(66.0)	0.296
Diabetes mellitus	22(27.8)	16(30.2)	0.771
Dyslipidemia	42(53.2)	29(54.7)	0.861
History of stroke or TIA	16(20.3)	10(18.9)	0.844
Smoking	28(35.4)	21(39.6)	0.626
Coronary heart disease	26(32.9)	13(24.5)	0.301
Valvular heart disease	6(7.6)	4(7.5)	1.000
Patent foramen ovale	2(2.5)	2(3.8)	1.000
TOAST classification, n(%)			0.693
LAA	23(29.1)	19(35.8)	
CE	33(41.8)	21(39.6)	
SUE	23(29.1)	24.5(24.5)	
Oral anticoagulation at baseline	22(27.8)	7(13.2)	0.046
Antiplatelet therapy at baseline	14(17.7)	8(15.1)	0.691
IV tPA	34(43.0)	20(37.7)	0.544
NIHSS score on admission, median (IQR)	18.0(13.0,24.0)	17.0(12.0,21.5)	0.367
Occlusion site, ICA/MCA, n(%)	60(75.9)	35(66.0)	0.214
Thrombus components, median			
RBC, mean, %(±SD)	39.3±19.7	43.5±18.9	0.230
F+P, mean, %(±SD)	52.9±19.5	49.3±18.5	0.278
WBC, median, % (IQR)/ %(±SD)	7.1(3.6,10.0)	7.2±4.5	0.811
CD3, median, cell/mm^2(IQR)	19.4(6.4,35.7)	24.7(10.4,48.0)	0.105
NETs, median, % (IQR)	1.3(0.3,3.9)	0.6(0.2,1.8)	0.022
Puncture-to-recanalization time	77.0(50.0,100.0)	47.0(36.0,64.5)	＜0.001
First attempt approach,ADAPT, n(%)	64(81.0)	24(45.3)	＜0.001
Clinical outcome, n(%)			
Any hemorrhage events	34(43.0)	17(32.1)	0.205
Parenchymal hemorrhage	25(31.6)	7(13.2)	0.015
NIHSS score at discharge, median (IQR)	14.0(5.0,33.0)	7.0(3.0,28.0)	0.060
90d mRS, (0-2), n (%)	28(35.4)	27(50.9)	0.077
Mortality	16(20.3)	7(13.2)	0.296

Abbreviations: SD: standard deviation; IQR: interquartile range; Non-FPE: non-first pass effect; FPE: first pass effect; TIA: transient ischemic attack;RBC, red blood cells; F+P, fibrin and plaete; WBC, white blood cells; NETs: neutrophil extracellular traps; CD3: CD3+ Tcells; IV tPA: intravenous tissue plasminogen activator; NIHSS: National Institute of Health Stroke Scale; ICA: internal carotid artery; MCA: middle cerebral artery; mTICI, Thrombolysis in Cerebral Infarction

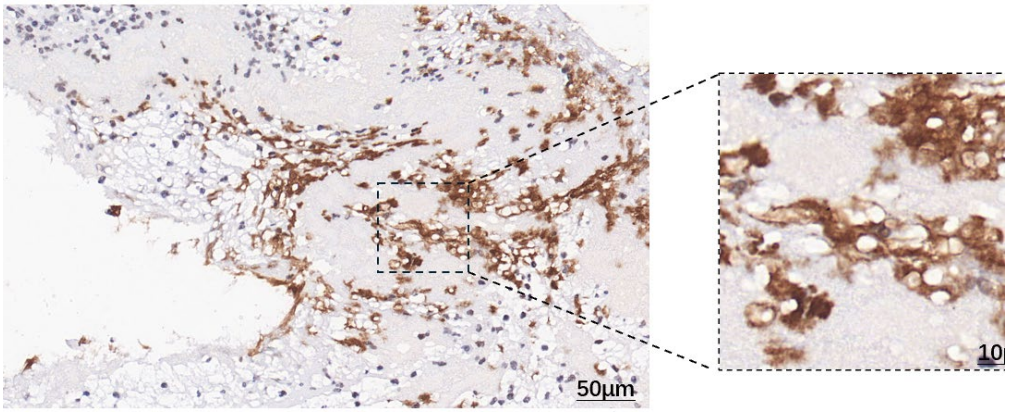
scale; mRS: modified Rankin scale; LAA: large artery atherosclerosis; CE: cardiogenic embolism; SUE: stroke of undetermined etiology; ADAPT: a direct aspiration first-pass technique.



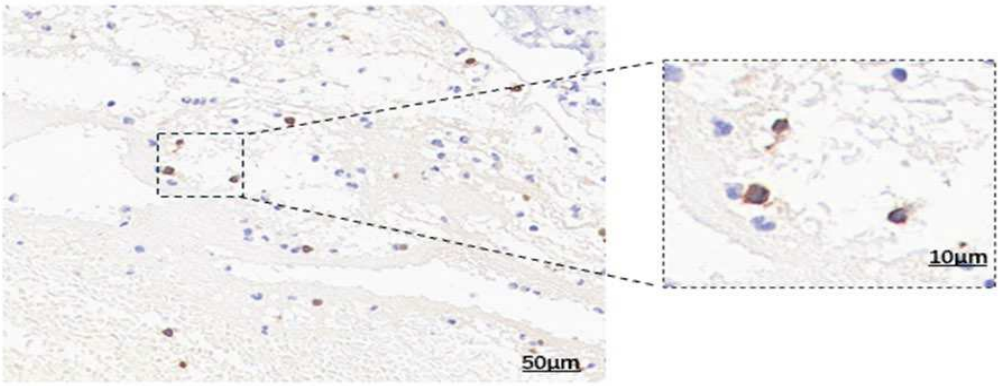
Supplementary Figure 1. Boxplot showing the association between IVT and thrombus components. There were no significant differences in thrombus composition between patients with non-intravenous thrombolysis and intravenous thrombolysis. There was no statistically significant difference in each thrombus component (RBC, WBC, F+P, NETs) between the IVT and Non-IVT groups, with p-values of 0.360, 0.206,0.601, and 0.275, respectively. RBC, red blood cells; F+P, fibrin and platelet; WBC, white blood cells; NETs: neutrophil extracellular traps; IVT, intravenous thrombolysis; Non-IVT, non-intravenous thrombolysis.



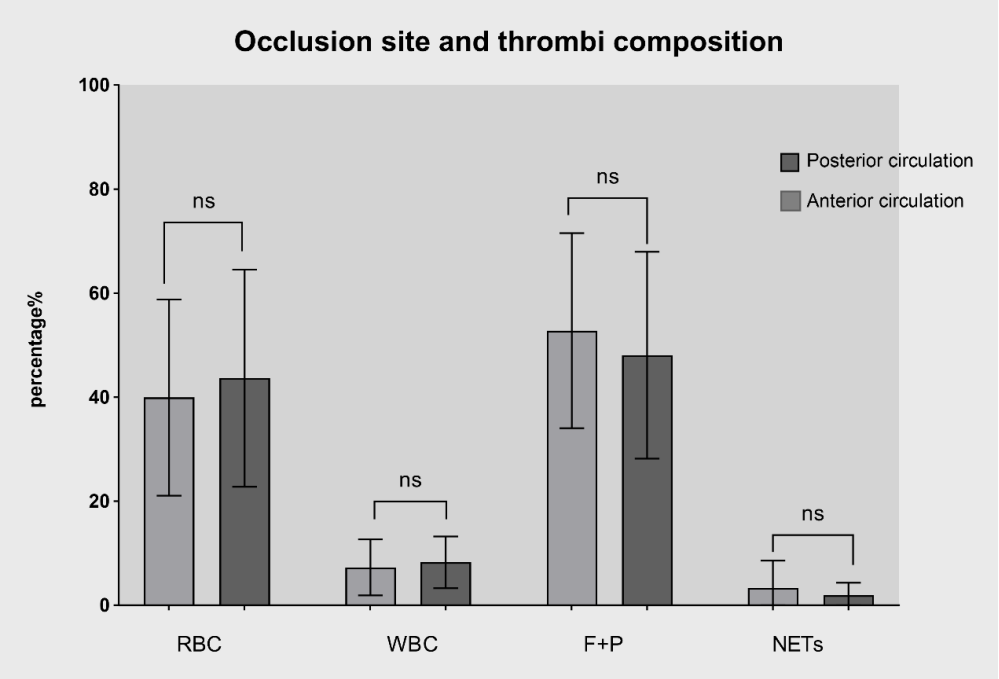
Supplementary Figure 2. Hematoxylin and eosin-stained section. Magnified images are presented on the bottom right (scale bar 10 μm).



Supplementary Figure 3. H3cit immunostaining (NETs: black arrowheads). Thrombus NETs were labelled using anti-H3cit antibodies, and the NETs showed a reticulated or striated morphology. H3cit: citrullinated histone H3, NETs: neutrophil extracellular traps.



Supplementary Figure 4. $CD3^+$ cells (brown) corresponding to T cells in thrombus.



Supplementary Figure 5. Relationship between occlusion site and components (RBC, WBC, F+P, and NETs) in intracranial thrombi. There was no statistically significant difference between these thrombus components in anterior circulation and posterior circulation. RBC, red blood cells; F+P, fibrin and platelet; WBC, white blood cells; NETs: neutrophil extracellular traps; ns: non-statistically significant difference.

Supplementary material on diagnostic criteria for etiologic typing

Etiological classification: Using the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification,[1] we employed a comprehensive diagnostic approach incorporating computed tomography, magnetic resonance imaging, digital subtraction angiography, carotid artery color Doppler ultrasonography, extended electrocardiography, and screening for patent foramen ovale to ascertain the most probable stroke etiology. Large-artery atherosclerosis was defined as catheter angiography findings showing >50% stenosis or occlusion of the ipsilateral extracranial or intracranial carotid artery proximal to the occlusion site without evidence of potential sources of cardioembolism in other diagnostic studies. Moderate or severe (>50%) underlying intracranial atherosclerotic stenosis was also regarded as large-artery atherosclerosis. Atherosclerotic features of the large arteries were assessed using anterior circulation high-resolution MRI or cerebral angiography: irregular margins of the arterial lumen, semicircular filling defects, and varying degrees of centripetal and

irregular stenosis and obstruction. Stroke etiology was defined as cardioembolism when at least one cardiac source for an embolus was identified after a complete cardiologic work-up including Holter monitoring (24 h, period of hospitalization after illness inset) and transthoracic echocardiography, which included atrial fibrillation or flutter, left atrial thrombus, a prosthetic valve, severe mitral stenosis, a patent foramen ovale, concomitant acute myocardial infarction, congestive heart failure, infective endocarditis, and sick sinus syndrome in the absence of moderate or severe (>50%) ipsilateral arterial stenosis on imaging studies and in the absence of significant stenosis (50%) of ipsilateral large extracranial arteries or significant atherosclerosis. Stroke of undetermined etiology (SUE) was defined when no reliable etiology or at least two etiologies were found after a complete clinical, laboratory, and imaging work-up (for example atrial fibrillation and permeable foramen ovale). Etiology was classified as TOAST-1 (large artery atherosclerosis [LAA]), TOAST-2 (cardioembolism [CE]), TOAST-5 (stroke of undetermined etiology [SUE]) based on established criteria and consensus guidelines. TOAST-3, which describes small vessel disease, and TOAST-4 (other determined causes) were excluded.

1 Adams HP, Bendixen BH, Kappelle LJ, *et al.* Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. *Stroke*. 1993;24:35–41. doi: 10.1161/01.str.24.1.35