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Large-vessel occlusion, large thrombus burden acute stroke in acute pulmonary embolism: a single multi-specialty multi-skill team treatment optimization

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A fully independent 74-year-old man with no significant morbidities was found at 10:55 AM with aphasia and right-sided hemiparesis. He was last seen well at 07:30 AM. Thrombectomy-Capable Stroke Centre emergency admission occurred at 11:37 AM. National Institute of Health Stroke Scale score was 24. There was also a marked respiratory effort with arterial blood

oxygen desaturation to 88%. Arterial blood pressure was 100/70 mm Hg; heart rate 115–120/min. Plain computed tomography (CT; 11:49 AM) showed early signs of left haemispheric anterior circulation infarction in absence of intracerebral bleed (Figure 1A, Alberta Stroke Program Early Computed Tomography Score 7). Hyperdense middle cerebral artery (MCA) sign was present (Figure 1A, inset). Intravenous thrombolysis (stroke protocol) was initiated. CT angiography (11:57 AM) revelaled: (i) left internal carotid artery (LICA) intracranial segment occlusion extending to MCA M1 segment, and (ii) massive, bilateral pulmonary embolism (PE; Figure 1B) with right ventricle enlargement (RV/LV ratio >1; Figure 1C). PE Severity Index was high at 134 (30-day PE death risk 10%–24.5%). Cerebral mechanical thrombectomy (MT) window was maintained [1]. Norepinephrine infusion was started to increase systolic arterial blood pressure to ≈140–160 mm Hg to enhance cerebral collateral supply. Interventional management team (stroke neurologist + anaesthetist + 2 MT-certified cardio-angiologists) had been activated consistent with the institutional standard operating procedure. Management plan included immediate cerebral mechanical reperfusion [2, 3] followed by catheter-based pulmonary thrombus load reduction [4]. At 12:16 PM the patient entered our cerebral intervention-compatible angio suite (intubation 12:20 PM). Optimal-result cerebral thrombectomy was performed (Figure 1E–F; first-pass complete aspiration; angiographic full reperfusion in absence of distal embolism; 12:47 PM).

Troponin I assessment showed marked (>10-fold) elevation. Continued intervention plan involved proceeding to pulmonary thrombus load reduction using aspiration technique (Indigo 8F system [4]; aspiration pump in Figure 1E). However, RV function improvement (RV/LV ratio <1.0; Figure 1E inset) and hemodynamic stabilization were noted; likely following systemic thrombolysis. PE transcatheter intervention was thus deferred, and the patient was transferred to Intensive Therapy Unit. After cerebral control CT excluded intracranial bleed, i.v. unfractionated heparin infusion was started for continued PE pharmaco-management (strict, low-therapeutic-end activated partial thromboplastin time level control;). Twelve days later the patient was transferred to a neuro-rehabilitation centre. He is scheduled for patent foramen ovale (a mechanistic link between PE and acute embolic stroke; Supplementary material, *Figure S1*) percutaneous closure.

Our fundamental considerations have included:

— Paramount role of minimizing the time from stroke onset to reperfusion (see Supplementary material, Figure *S1* for MR PREDICTS https://www.mrclean-trial.org/mr-predicts.html absolute and relative patient benefit calculations in carotid T-occlusion).

— Counteracting cerebral injury aggravation with PE-generated arterial blood desaturation and haemodynamic instability.

— Any potential transportation attempt to the nearest Comprehensive Stroke Centre (Level-1 stroke centre) would have placed this patient, like many others [1, 2, 5], beyond the MT therapeutic window (on top of challenges of heamodynamically unstable patient Comprehensive Stroke Centre acceptance and inter-hospital transfer logistics), depriving the patient from guideline-mandated treatment [1].

Patients and healthcare systems may benefit from developing, and maintaining, multi-area skills of cardio-angiology vascular operators that should, in particular in geographies with unmet needs, include acute stroke interventions within a trained team with local stroke neurology [2, 5].

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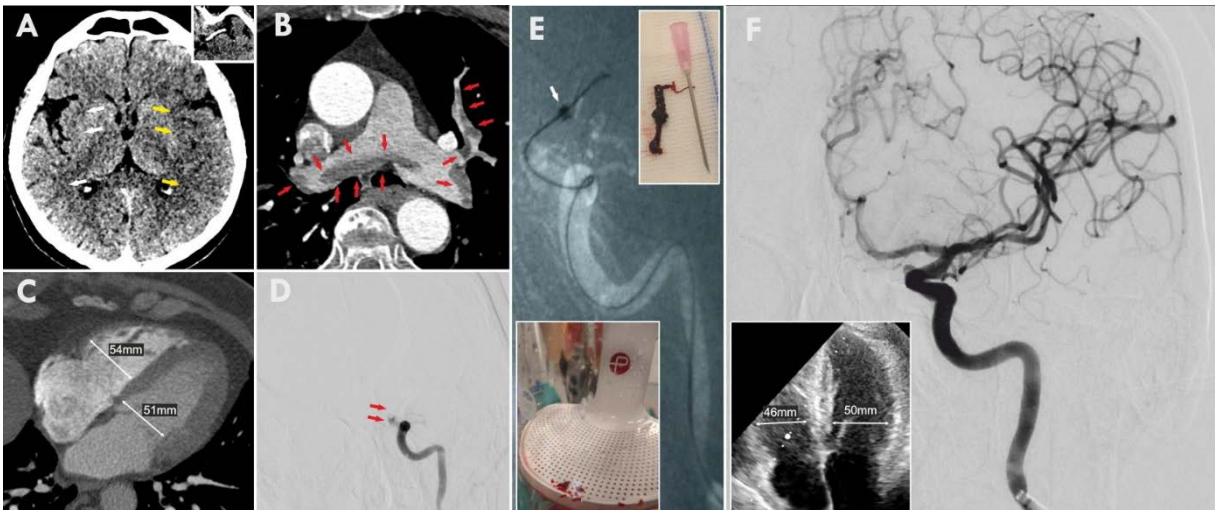

Figure 1. Non-invasive and invasive imaging in a 74-year-old man presenting with acute, major left (dominant) haemispheric stroke and increased respiratory effort with reduced arterial blood saturation (SaO_2 88%).

Emergent cerebral plain CT (**A**, 12 min after arrival) showed early signs of left haemispheric anterior circulation infarction with effacement of the sulci and mild cortical hypodensity (compare the right vs left haemisphere cortex for asymmetry, white/yellow arrows) in absence of intracerebral bleeding; Alberta Stroke Program Early Computed Tomography, (ASPECTS) score was 7, indicating moderate cerebral tissue loss. Hyperdense middle cerebral artery sign was present, suggestive of its thromboltic occlusion (inset, arrow); i.v. thrombolysis (stroke protocol) was initiated instantaneously. CT angiography (2 min after thrombolysis onset) demonstrated radiologically massive PE (**B**, red arrows) with enlarged right ventricle, indidicating RV struggle (**C**). Distal LICA occlusion was present, with moderate-only collateral supply from the right to the left haemispheric vessels. The right ventricle enlargement (**C**) was accompanied by clinical signs of intermediate-high risk PE. Norepinephrine iv infusion was started to increase arterial blood pressure to ≈140–160 mm Hg systolic to enhance collateral supply as the cerebral autoregulation spontaneous hypertensive response was likely offset by the PE effect. Cerebral angiography (**D**, NeuronMax88 8F guiding catheter, Penumbra, white arrow marks the catheter tip and some mild vasospasm) confirmed LICA T-occlusion (red double-arrow). Large-bore aspiration catheter (Jet 7, Penumbra, catheter tip marked with white

arrow **E**) was advanced up to the thrombus head with a cerebral guidewire (Synchro 0.014", Stryker) and microcatheter (3Max, Penumbra) support. Vacuum pump-assissted (**E**, bottom-left inset) mechanical aspiration was performed for 90 sec (backflow absent). JET-7 was removed under active aspiration, followed by the NeuronMax guiding catheter (backflow absent) active manual aspiration removal. A large embolus was retrieved en-block (**E**, top-right inset) with several additional small emboli visible in the aspiration pump filter (**E**, botom-left). Angiography (**F**) demonstrated a "first-pass" optimal cerebral reprfusion effect (TICI-3, absence of any residual embolism, absence of embolism-to-new-territory) achieved swiftly after the diagnosis of large vessel occlusion stroke. The Troponin I level lab evaluation, available at the cerebral embolectomy completion point was 0.146 ng/ml (normal level ≤0.014 ng/ml), consistent with the RV myocardial strain-related injury with the PE load (renal function normal). Right femoral vein puncture was considered for transition to PE catheter embolectomy (clot separator — assissted aspiration, Indigo system, Penumbra), using the same aspiration pump (**E**). Control echocardiography, however, showed RV dilation reduction (**F**, bottom-left inset) likely resulting from the on-going iv. thrombolysis effect on PE magnitude. This was accompanied by the patient haemodynamic stabilization (spontaneous blood pressure 110/75 mm Hg, heart rate 95/min), and a team decision was made to put the transcatheter pulmonary embolectomy on hold.

See text for further details; for transoesophageal echocardiography performed 4 days later see Supplementary material, *Figure S2*.

Abbreviations: CT, computed tomography; LICA, left internal carotid artery; PE, pulmonary embolism; RV, right ventricle