The Canadian Journal of Neurological Sciences (2026), 1–4
doi:10.1017/cjn.2026.10570

Canadian Journal of Neurological Sciences
Journal Canadien des Sciences Neurologiques

Brief Communication

Concomitant Pulmonary Embolism and Acute Ischemic Stroke Using Undergoing Extended Chest CT Angiography: A Cohort Study

Hilal Al Hasni[1], Jesmeen K. Deo[1], Dominique DaBreo[1], Gurmohan Dhillon[1], Gordon Boyd[1], Albert Jin[2], Isehaq Al Jabri[3], A. Reza Nasirzadeh[1] and Shirin Jalini[1]

[1]Queen's University, Canada, [2]Medicine, Queen's University, Canada and [3]Division of Neurology, Department of Medicine, Queen's University, Canada

ABSTRACT: We conducted a single-center prospective cohort study in Ontario, Canada, to assess concomitant pulmonary embolism (PE) in hyperacute stroke. Stroke CT angiography was modified to start at the diaphragm in order to include the chest. Primary outcome of PE was reported by cardiothoracic/chest radiologists. 12/208 stroke patients had an acute PE (5.8%), all of which were clinically asymptomatic, despite imaging in some showing right-heart strain. None had cancer and only half had a patent foramen ovale. Patients with PE tended to have more severe stroke, large vessel occlusion and worse outcomes. The implications and management of these findings require further exploration.

RÉSUMÉ : Embolie pulmonaire concomitante détectée à l'aide d'un examen d'angiographie thoracique par tomodensitométrie étendue et AVC ischémique aigu : une étude de cohorte. Nous avons mené une étude de cohorte prospective monocentrique en Ontario, au Canada, afin d'évaluer l'incidence de l'embolie pulmonaire (EP) concomitante dans le cas d'un AVC de nature hyper-aigüe. À cet égard, des examens d'angiographie par tomodensitométrie cérébrale ont été modifiés pour débuter au niveau du diaphragme et inclure le thorax. Des cas d'EP ont été rapportés par des radiologues des régions cardiothoraciques et thoraciques. Au total, 12 des 208 patients victimes d'un AVC présentaient une EP aiguë (5,8 %) ; de plus, tous étaient cliniquement asymptomatiques, et ce, malgré des résultats d'IRM montrant chez certains une tension cardiaque située à la droite. À noter qu'aucun d'entre eux ne souffrait de cancer et seulement la moitié présentait un foramen ovale perméable (FOP). Les patients atteints d'EP avaient ainsi tendance à présenter un AVC plus grave, une occlusion des gros vaisseaux sanguins et une évolution de leur état davantage défavorable. Les implications de ces constatations, de même que la prise en charge des patients, doivent être étudiées plus en détail.

Keywords: chest CT angiogram; pulmonary embolism

(Received 17 June 2025; final revisions submitted 26 November 2025; date of acceptance 11 February 2026)

Pulmonary embolism (PE) is a serious condition with an annual incidence rate of 0.50 to 0.69 per 1,000 persons in the general population[1] and is associated with mortality as high as 24%. Patients with ischemic stroke are at risk of PE, both as a concurrent presentation due to shared risk factors of hypercoagulability and causal pathway of paradoxical embolism, and as a subsequent consequence of their immobility.

Previous work has shown PE rates of nearly 0.3–1% in the hospitalized ischemic stroke population.[2] These studies are mostly limited to symptomatic cases during the hospitalization period and show a three-month mortality between 8.6 and 17% in ischemic stroke patients with PE.

In contrast, the literature on concurrent PE at the time of hyperacute stroke is sparse and limited to retrospective case series.[3] In these studies, CT technique only included the proximal pulmonary arteries and upper lobes. Additionally, subspecialty chest radiologist reporting was not implemented. These factors may have possibly led to underreporting of the true rates of concomitant PE in the hyperacute stroke population.

The objective of this study was to investigate the rate of concomitant PE in hyperacute stroke patients. We performed a retrospective analysis of a previous prospective cohort study, whose methods were previously described[4] and followed the Strengthening the Reporting of Observational Studies in Epidemiology Statement. In brief, suspected hyperacute stroke patients presenting to a Canadian comprehensive stroke center underwent modified Code Stroke imaging with a 64-slice CT scanner, where the longitudinal axis of CT angiography was extended from the carina to the diaphragm as part of neurovascular imaging. This allowed imaging of the full cardiac structures, as well as extended view of the lungs and pulmonary vasculature. The cardiac and chest component of the imaging was reported after acquisition by experienced fellowship-trained chest and cardiothoracic radiologists. The primary outcome in this analysis was rate of concomitant

Corresponding author: Shirin Jalini; Email: shirin.jalini@kingstonhsc.ca

Cite this article: Al Hasni H, Deo JK, DaBreo D, Dhillon G, Boyd G, Jin A, Al Jabri I, Nasirzadeh A Reza, and Jalini S. (2026) Concomitant Pulmonary Embolism and Acute Ischemic Stroke Using Undergoing Extended Chest CT Angiography: A Cohort Study. *The Canadian Journal of Neurological Sciences*, https://doi.org/10.1017/cjn.2026.10570

© The Author(s), 2026. Published by Cambridge University Press on behalf of Canadian Neurological Sciences Federation. This is an Open Access article, distributed under the terms of the Creative Commons Attribution licence (https://creativecommons.org/licenses/by/4.0/), which permits unrestricted re-use, distribution and reproduction, provided the original article is properly cited.

https://doi.org/10.1017/cjn.2026.10570 Published online by Cambridge University Press

Table 1. Stroke patients with and without pulmonary embolism

Characteristic	All patients (*n* = 208)	Without PE (*n* = 196)	With PE (*n* = 12)	*p* value
Age, years, median (IQR*)	75 (65–83)	75 (65–84)	70 (62–77)	0.43
Sex, male *n* (%)	112 (54)	107 (55)	5 (42)	0.38
Risk factors, *n* (%)				
Hypertension	147 (71)	138 (70)	9 (75)	0.74
Dyslipidemia	97 (47)	90 (46)	7 (58)	0.40
Smoking	72 (35)	70 (36)	2 (17)	0.17
Atrial fibrillation	63 (30)	60 (31)	3 (25)	0.68
Coronary artery disease	44 (21)	42 (21)	2 (16)	0.70
Diabetes	56 (27)	53 (28)	3 (25)	0.88
Pre-existing/new cancer	13 (1)	13 (7%)	0 (0)	0.36
Chronic Obstructive Pulmonary Disease (COPD) asthma	25 (12)	24 (12)	1 (8)	0.76
Patient foramen ovale (PFO)	18 (26)**	14 (23)**	4 (50)**	0.04
Baseline mRS, median (IQR)	1 (0–2)	1 (0–2)	1 (0–2)	0.85
Time (min) last seen normal to CT, median (IQR)	246 (108–720)	245 (108–720)	565 (174–668)	0.43
Initial NIHSS score, median (IQR)	7 (3–15)	6 (3–13)	20 (17–22)	<0.001.
Large vessel occlusion	61 (29)	52 (27)	9 (75)	<0.001.
Hyperacute treatment				
Intravenous thrombolysis	41 (20)	40 (20)	1 (8)	0.30
Endovascular thrombectomy	35 (17)	35 (18)	6 (50)	0.006
Stroke etiology				
Small vessel disease, *n* (%)	27 (13)	26 (13)	1 (8)	0.62
Large vessel disease, *n* (%)	43 (21)	42 (21)	1 (8)	0.28
Cardioembolic, *n* (%)	78 (38)	70 (36)	8 (67)	0.03
Other, *n* (%)	13 (6)	13 (7)	0 (0)	0.36
Undetermined, *n* (%)	47 (23)	45 (23)	2 (16)	0.61
90-day mRS, median (IQR)	1 (0–2)	2(1–5)	4 (2.75–3.6)	0.04
90-day mortality, *n* (%)	41 (20)	37 (19)	4 (33)	0.08

*Interquartile range.
**8/12 stroke patient with PE and 62/196 stroke patient without PE had test for PFO.PE = pulmonary embolism.

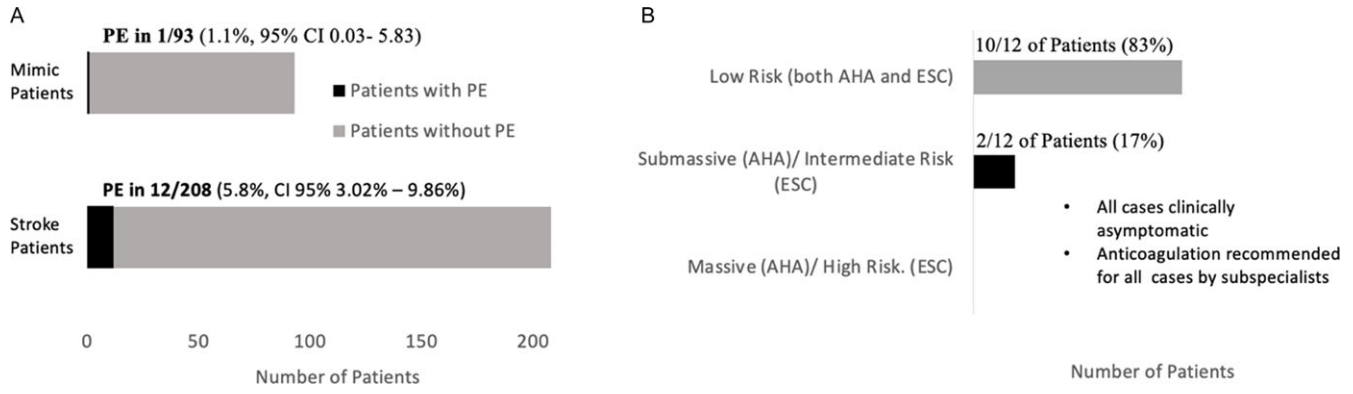


Figure 1. (A) Rates of pulmonary embolism (PE) in patients undergoing extended CT angiography during hyperacute stroke imaging. PE was found in 12/208 (5.8%, 5.8%, CI 95%, 3.02% – 9.86%) of stroke patients and 1/93 (1.1%, 95% CI 0.03– 5.83) of stroke mimics. (B) Most PE (10/12; 83%) found in stroke patients were low risk as per AHA/ESC classification, while 2/12 cases (17%) were of intermediate risk. All PEs were clinically asymptomatic.

https://doi.org/10.1017/cjn.2026.10570 Published online by Cambridge University Press

Table 2. Characteristics of patients with pulmonary embolism

Patient	PE Characteristics	Stroke etiology	Cancer	PFO	Treatment choice	Other
1	Multilobar	Cardioembolic (PE + PFO)	No	Yes	Rivaroxaban-day 9	Lifelong anticoagulation PFO closure
2	Multilobar, Right heart strain	Cardioembolic (Left ventricular thrombus and PE)	No	No	Dalteparin-day 1 Apixaban-day 5	Lifelong anticoagulation
3	Multilobar	Cardioembolic (PE + PFO)	No	Yes	Dalteparin-day 5 Apixaban-day 10	Lifelong anticoagulation
4	Multilobar, Right heart strain	Cardioembolic (PE + PFO)	No	Yes	Apixaban-day 17	Lifelong anticoagulation PFO closure
5	Multilobar	Small vessel disease	No	?	Apixaban-day 3	Lifelong anticoagulation
6	Multilobar	Cardioembolic (AF)	No	No	Heparin-day 4	Died
7	Segmental	Large vessel disease	No	No	Heparin-day 4, Apixaban-day 8	Lifelong anticoagulation
8	Segmental	Cardioembolic (Left atrial appendage thrombus and AF)	No	?	Recommended but none given	Died
9	Multilobar	Cryptogenic	No	No	Apixaban-day 3	Lifelong anticoagulation
10	Segmental	Cryptogenic	No	No	Apixaban-day 21	Lifelong anticoagulation
11	Multilobar	Cardioembolic (AF)	No	No	Dalteparin-day 12, Rivaroxaban-day 20	Died
12	Multisegmental	Cardioembolic (AF)	No	No	Apixaban-day 7	Died

PE = pulmonary embolism; AF = atrial fibrillation.

PE. Participants presenting on Code Stroke were enrolled through deferral of consent during daytime working hours only, due to requirement for timely reporting of in-house cardiothoracic radiologists.

Microsoft Excel (Version16.61.1, 2019) was used to calculate percentages, means, medians and interquartile ranges. Interrater reliability of PE findings was assessed using kappa score. *P*-values are reported as per *t*-tests for continuous data and chi-squared tests for categorical data.

Ethics approval was obtained from the Queen's University Health Sciences Research Ethics Board (HSREB; #6030510).

Among 301 patients, 208 were diagnosed with stroke. PE was found in 12/208 (5.8%, CI 95% 3.02 – 9.86%) of stroke and 1/93 (1.1%, 95% CI 0.03– 5.83) of mimic patients (see Figure 1A, $p = 0.06$). Interrater reliability kappa score was 1.00 for detection of PE based on two readers (G.D. and M.P.) blindly reviewing imaging. All PEs were clinically asymptomatic at presentation, with no evidence of hypoxia or tachycardia.

Stroke patients with and without PE were matched with respect to age, sex, pre-existing functional status and major risk factors, including cancer or underlying lung disease (see Table 1). PE was associated with severe strokes, presence of large vessel occlusion, cardioembolism and worse functional outcomes (see Table 1). Most cases of PE were low risk, as per American Heart Association (AHA) and European Society of Cardiology (ESC) classification, with the exception of two cases that were submassive (AHA)/ intermediate risk (ESC; see Figure 1B). PEs were often multilobar or segmental (Table 2). Lifelong anticoagulation was recommended by subspecialists for all patients. Initial treatment included heparin and oral anticoagulants starting at day 1 to day 21 and no treatment complications occurred.

Patent foramen ovale (PFO) rate in patients with PE versus without PE was 50% versus 23%, respectively. Although this difference did technically reach statistical significance ($p = 0.04$), it should not be interpreted as true comparative rates between the two groups, as 8/12 (66.7%) of stroke patients with PE versus 62/196 (31.6%) without PE had testing for PFO with a transthoracic echocardiogram with bubble study. Median time from stroke onset to stroke CT imaging was not significantly higher for patients with PE compared to those without.

In summary, in this cohort study of hyperacute stroke patients who underwent extended CT Angiography (CTA), we found 1) concomitant asymptomatic PE is associated with increased stroke severity, Large vessel occlusion (LVO), cardioembolic etiology and worse clinical outcomes, 2) PE findings change clinical management, specifically anticoagulation, as recommended by subspecialists.

No clinical trials or observational studies have evaluated the incidence or prevalence of concomitant stroke and PE and reported data are based on case reports and case series.[5] Our series is the largest to highlight these conditions using extended CTA.

There are no specific guidelines on treatment of incidental PE in non-cancer patients. Management decisions are usually individualized based on emboli size, location, patient factors and institutional protocols. Generally, subsegmental PE may not need treatment[6] but there is general trend in treating asymptomatic PEs that are not subsegmental to prevent chronic thromboembolic pulmonary hypertension or post-PE syndrome. All PEs in this study were segmental or lobar and treated with lifelong anticoagulation.

Patients with PE had more severe stroke, higher frequency of LVO and stroke secondary to cardioembolism. Potential mechanisms could include paradoxical embolism via PFO and atrial fibrillation (AF), both of which were present in some of our patients. The Tromsø study[7] concluded that AF was associated with increased risk of PE. This relationship is thought to be bidirectional[8], such that 1) increased pulmonary vascular resistance, right ventricular afterload and cytokine release can lead to elevated right atrial pressure and strain, leading to AF, and 2) AF which in turn could lead to PE through right atrial thrombosis.[9] Therefore,

https://doi.org/10.1017/cjn.2026.10570 Published online by Cambridge University Press

anticoagulation for stroke prevention in AF may be beneficial for PE prophylaxis, while anticoagulation to prevent venous thromboembolic disease (VTE) recurrence can provide protection for AF.[10]

Our study has notable limitations. Given that our enrollment was non-consecutive, findings do not represent incidence or prevalence of PE in hyperacute stroke. Additionally, no multivariate analysis was performed. These results should therefore be viewed as possible suggestion of a signal that require further evaluation with properly designed studies.

Acknowledgements. This study was supported by the Translational Institute of Medicine at Queen's University, Kingston, Ontario.

Author contribution. **HA** assisted in the data collection and drafted the initial copy of the manuscript. **JD and IA** collected demographic data. **DD, GD, and ARN** assisted in the interpretation of the imaging. **AJ** and **JGB** assisted with the idea development and interpretation. **SJ** suggested the initial idea, coordinated the project, and completed the final draft of the manuscript.

Funding statement. Shirin Jalini received funding from the Translational Institute of Medicine at Queen's University for this work.

Competing interests. Authors have no other disclosures relevant to this work.

References

1. Skaf E, Stein PD, Beemath A, Sanchez J, Olson RE. Fatal pulmonary embolism and stroke. *Am J Cardiol*. 2006;97(12):1776–7,
2. Pongmoragot J, Rabinstein AA, Nilanont Y, Swartz RH, Zhou L, Saposnik G. Investigators of Registry of Canadian Stroke Network (RCSN) and University of Toronto Stroke Program for Stroke Outcomes Research Canada (SORCan [www.sorcan.ca]) Working Group. Pulmonary embolism in ischemic stroke: clinical presentation, risk factors, and outcome. *J Am Heart Assoc*. 2013;2(6):e000372,
3. Chen D, Yang Y, Bhaskar SMM. Pulmonary embolism after acute ischaemic stroke (PEARL-AIS): global prevalence, risk factors, outcomes, and evidence grading from a meta-analysis. *Neurol Int*. 2025;17(10):168.
4. Lee P, Dhillon G, Pourafkari M, et al. Non-ECG-gated cardiac CT angiography in acute stroke is feasible and detects sources of embolism. *Int J Stroke*. 2024;19(2):189–98,
5. Saleh Velez FG, Ortiz Garcia JG. Management dilemmas in acute ischemic stroke and concomitant acute pulmonary embolism: Case series and literature review. *eNeurologicalSci*. 2021;23:100341.
6. Kearon C, Akl EA, Ornelas J, et al. Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report. *Chest*. 2016;149(2):315–52.
7. Enga KF, Rye-Holmboe I, Hald EM, et al. Atrial fibrillation and future risk of venous thromboembolism: the Tromsø study. *J Thromb Haemost*. 2015;13(1):10–6.
8. Lutsey PL, Norby FL, Alonso A, et al. Atrial fibrillation and venous thromboembolism: evidence of bidirectionality in the Atherosclerosis Risk in Communities Study. *J Thromb Haemost*. 2018;16(4):670–9.
9. Watson T, Shantsila E, Lip GY. Mechanisms of thrombogenesis in atrial fibrillation: virchows triad revisited. *Lancet*. 2009;373(9658):155–66.
10. Miao L, Shi J, Yu H, et al. Studies on atrial fibrillation and venous thromboembolism in the past 20 years: a bibliometric analysis via citeSpace and VOSviewer. *J Am Heart Assoc*. 2023;12(17):e029810.

https://doi.org/10.1017/cjn.2026.10570 Published online by Cambridge University Press