Supplementary Table 1

Sensitivity analyses excluding two records with uncertain etiologic assignments

Topographic Patterns and Etiologic Spectrum of Isolated Pontine Infarcts on Diffusion-Weighted MRI

The record labeled branch atheromatous disease and the record with an isolated acute-phase Protein S abnormality were excluded in a post hoc sensitivity analysis. The original 69-patient cohort and its operational classifications were preserved; no new etiologic category was assigned to either patient.

Among the remaining 67 patients, the principal Holm-adjusted inferences were consistent with the primary analysis. These results assess sensitivity to the two assignments and do not establish the etiologic mechanism in either patient.

A Topography and etiology after excluding the two records

Comparison	Raw p	Holm-adjusted p	Cramer's V
Lesion side	<0.001	<0.001	0.721
Rostrocaudal segment	0.357	0.357	0.267
Recorded morphology/topography	<0.001	<0.001	0.577

B Admission NIHSS comparisons after excluding the two records

Grouping variable	Raw p	Holm-adjusted p	Epsilon-squared
Lesion side	0.025	0.101	0.084
Rostrocaudal segment	0.915	0.915	0.000
Recorded morphology/topography	0.034	0.101	0.116
Etiologic category	0.152	0.304	0.044

C Vascular risk factors and etiology after excluding the two records

Risk factor	Raw p	Holm-adjusted p	Cramer's V
Hypertension	0.732	1.000	0.177
Diabetes mellitus	0.513	1.000	0.228
Hyperlipidemia	0.920	1.000	0.128
Active smoking	0.614	1.000	0.199
Coronary artery disease	0.040	0.201	0.373
Prior cerebrovascular event	0.025	0.149	0.372

Panels A–C include 67 patients. Holm adjustment was applied separately to the three topography tests, four global NIHSS comparisons, and six risk-factor tests. Atrial fibrillation remained descriptive because it contributed directly to cardioembolic classification. NIHSS, National Institutes of Health Stroke Scale.

Supplementary Table 1 continued

D Descriptive comparison with the primary cohort

Recorded category	Primary cohort	Two records excluded
Operational small-vessel/lacunar	36/69 (52.2%)	34/67 (50.7%)
Cryptogenic	13/69 (18.8%)	13/67 (19.4%)
Other determined	7/69 (10.1%)	7/67 (10.4%)
Cardioembolic	7/69 (10.1%)	7/67 (10.4%)
Large-artery atherosclerosis	6/69 (8.7%)	6/67 (9.0%)
Anteromedial morphology	44/69 (63.8%)	42/67 (62.7%)
Middle pontine segment	51/69 (73.9%)	49/67 (73.1%)
Bilateral involvement	6/69 (8.7%)	6/67 (9.0%)

The two excluded records were both classified as right-sided, middle-segment, anteromedial lesions in the retained dataset. Their admission NIHSS scores were 2 and 3. These descriptors do not by themselves establish an intrinsic small-vessel mechanism. The six bilateral cases remained identical to the six large-artery atherosclerosis cases.

E Additional exclusion of the six overlapping bilateral and large artery cases

Comparison	Raw p	Holm-adjusted p	Cramer's V
Lesion side	0.536	0.536	0.200
Recorded morphology/topography	0.038	0.076	0.334

Panel E includes 61 patients after exclusion of both disputed records and the six overlapping bilateral/large-artery atherosclerosis cases. Holm adjustment was applied across these two tests. Neither association remained statistically significant after adjustment; this does not establish the absence of an association.

Statistical methods

Categorical associations were evaluated with two-sided Fisher–Freeman–Halton Monte Carlo tests conditional on the observed margins, using probability-mass ordering, 500,000 resamples, and a NumPy default random generator reset to seed 55179 for each table. Cramer's V was calculated from the uncorrected Pearson chi-square statistic. Global NIHSS comparisons used tie-corrected Kruskal–Wallis tests, with epsilon-squared as the effect estimate. No pairwise comparisons were added. Calculations used Python 3.12.14, SciPy 1.17.0, and NumPy 2.3.5.

Data reconciliation

The locked 69-patient source workbook was linked to the archived patient-level adjudication audit. Baseline category totals, topography-by-etiology tables, and risk-factor counts matched the accepted manuscript. Baseline NIHSS test values matched at the reported precision; Monte Carlo estimates were consistent within simulation variability. The original primary-analysis values were not replaced.