

The Stroke Begins Outside the Brain

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Abstract

An ischemic stroke is a neurological emergency, yet the vessel occlusion may be the end of a process that began years earlier in the intestine, liver, heart, vascular wall or coagulation system. This paper presents a multi-compartment model of stroke causation. It distinguishes lipid retention and plaque development from the immediate thrombotic or embolic event, and asks when impaired sterol handling may contribute to the former without directly accounting for the latter.

The Clinical Question and Its Context

The phrase “fat cannot be processed” is too vague for causal medicine. Dietary triglycerides normally enter circulation through chylomicrons, while intestinal NPC1L1 and ABCG5/ABCG8 transporters govern sterol uptake and exclusion. The liver redistributes cholesterol in lipoproteins and removes sterol through biliary secretion and bile-acid conversion. Each step has different pathological consequences. Atherogenic apoB-containing particles infiltrate susceptible arterial walls; retained lipoproteins may provoke inflammatory responses and plaque formation. None of this implies that undigested fat collects directly within a brain artery.

Proposed Biological or Explanatory Model

A useful model includes five paths: intestinal sterol entry; hepatic lipoprotein production and clearance; endothelial retention of apoB particles; plaque destabilization and platelet-rich thrombosis; and cardioembolism or small-vessel injury arising through separate mechanisms. Rare ABCG5/8 disorders establish that abnormal absorption and biliary excretion can be clinically consequential, but most ischemic strokes are not caused by sitosterolemia. The model therefore treats abnormal clearance as one potential contributor to atherosclerotic stroke, not a unifying explanation for intracranial embolism, dissection or atrial fibrillation.

Mechanism-Specific Interpretation

A sophisticated vascular account must distinguish the source of plaque from the source of the clot. The atheroma develops in a particular arterial environment under sustained exposure to retained lipoproteins, local injury and immune-cell recruitment. A plaque may remain clinically silent until rupture or erosion produces thrombosis, or it may cause critical narrowing without an acute rupture. By contrast, an embolus produced in a fibrillating atrium can occlude a cerebral vessel without needing substantial atherosclerotic plaque in that artery. Bile-acid handling is potentially relevant upstream of lipoprotein biology; it does not by itself explain cardiac rhythm, coagulation or the anatomy of embolization. This nested-cause framework protects both conventional knowledge and original inquiry.

Competing Explanations and Limits of Inference

It would be disproved as a general thesis by stroke subgroups in which plaque biology is irrelevant. Lacunar infarction related to arteriolosclerosis, atrial-fibrillation-associated embolism, and arterial dissection have different proximate mechanisms. Even in atherosclerotic disease, serum cholesterol concentration, apoB burden, hypertension, diabetes, smoking and inherited factors may better explain risk than a presumed bile-flow defect. The distinction between circulating lipoprotein particles and fat malabsorption is indispensable.

Discriminating Observations and Evidence Thresholds

What would distinguish the hypotheses? Evidence for ABCG5/ABCG8 dysfunction or major sterol abnormalities would support a sterol-handling component in an unusual case. Evidence for a left atrial appendage thrombus, atrial fibrillation or a spontaneous cervical artery dissection would favor alternative proximate causes. A convincing explanatory model must identify the stage at which each factor acts, not simply claim that the last event was “downstream.”

Implications for Interpretation and Future Inquiry

The practical research question is not “does bile blockage cause stroke?” but “which measurable transport phenotype predicts which stroke mechanism after standard risk factors are considered?” Useful measures include apoB, lipoprotein phenotype, relevant genetic findings, hepatic disease when clinically indicated, carotid and intracranial imaging, cardiac rhythm evaluation and stroke-territory concordance. No dietary cleanse should be substituted for acute stroke reperfusion assessment or proven secondary prevention.

Selected Evidence and Further Reading

1. [Sitosterolemia: a review and update of pathophysiology, clinical spectrum, diagnosis, and management](#)
2. [Sitosterolemia: Twenty Years of Discovery of ABCG5 ABCG8](#)
3. [Recent Advances in the Digestive, Metabolic and Therapeutic Effects of FXR and FGF19](#)

Source lineage: Prior stroke-pathogenesis investigation; Terrain Medicine Handbook

Editorial note: Conceptual scientific commentary; not an original clinical trial, systematic review, peer-reviewed publication, or medical advice. Hypotheses are not established treatment effects.