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Abstract: Stroke is among the major causes of mortality and disabilities in the world. About 80% of all strokes in the anterior circulation are ischemic and up to 20% of all ischemic strokes are caused by extracranial atherosclerotic carotid artery stenosis. The prevalence of a cervical internal carotid artery stenosis increases with age and can be found in 6.9% of the elderly population (>65 years). Atherosclerotic changes of the carotid vessel wall can lead to plaque vulnerability and may result in arterio-arterial embolism, which frequently underlie carotid-related cerebrovascular ischemic events. Carotid atherosclerosis is characterised by inflammation, extensive degradation of extracellular matrix components, neovascularization, and as recently recognised is also affected by epigenetic changes. These factors accelerate the progression of atherosclerosis towards vulnerable plaques and increase the risk of ischemic stroke. In this review, the main pathophysiological mechanisms leading to rupture-prone carotid artery plaques and successive ischemic stroke are considered. It is important to recognise the heterogeneity of atherosclerosis and that various pathophysiological processes dissected in this review are not acting individually, but rather in a complementary manner. The identification and careful integration of all relevant factors will be required for the development of future diagnostic and therapeutic strategies.