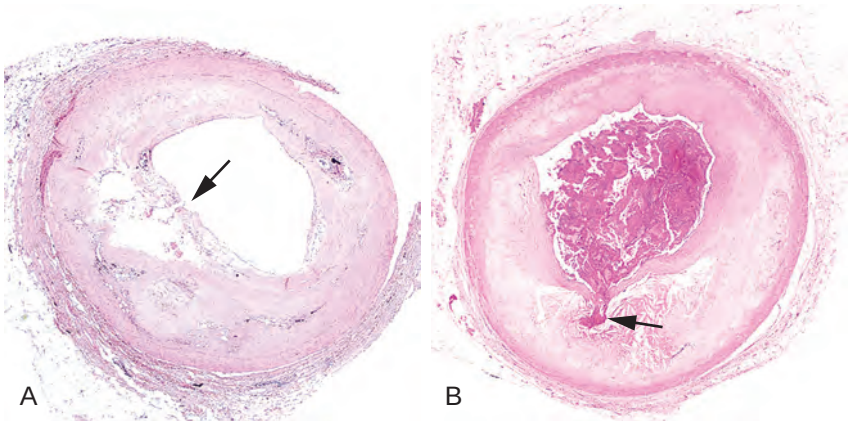

Figure 11-15 Atherosclerotic plaque rupture. **A,** Plaque rupture without superimposed thrombus, in a patient who died suddenly. **B,** Acute coronary thrombosis superimposed on an atherosclerotic plaque with focal disruption of the fibrous cap, triggering fatal myocardial infarction. In both **A** and **B,** an *arrow* points to the site of plaque rupture. (**B,** Reproduced from Schoen FJ: Interventional and Surgical Cardiovascular Pathoresclerosisology: Clinical Correlations and Basic Principles. Philadelphia, WB Saunders, 1989, p 61.)

plaques ("fibrous plaques") are composed almost exclusively of smooth muscle cells and fibrous tissue.

Plaques generally continue to change and progressively enlarge through cell death and degeneration, synthesis and degradation (remodeling) of extracellular matrice, and organization of any superimposed thrombus. Moreover, atheromas often undergo calcification (Fig. 11-14C). Patients with advanced coronary calcification have increased risk for coronary events.

Atherosclerotic plaques are susceptible to the following clinically important pathologic changes:

- **Rupture, ulceration, or erosion** of the surface of atheromatous plaques exposes highly thrombogenic substances and leads to **thrombosis**, which may partially or completely occlude the vessel lumen (Fig. 11-15). If the patient survives, the clot may become organized and incorporated into the growing plaque.
- **Hemorrhage into a plaque.** Rupture of the overlying fibrous cap, or of the thin-walled vessels in the areas of neovascularization, can cause intraplaque hemorrhage; a contained hematoma may expand the plaque or induce plaque rupture.
- **Atheroembolism.** Plaque rupture can discharge atherosclerotic debris into the bloodstream, producing microemboli.
- **Aneurysm formation.** Atherosclerosis-induced pressure or ischemic atrophy of the underlying media, with loss of elastic tissue, causes weakness and potential rupture.

Consequences of Atherosclerotic Disease

Large elastic arteries (e.g., aorta, carotid, and iliac arteries) and large and medium-sized muscular arteries (e.g., coronary and popliteal arteries) are the major targets of atherosclerosis. Symptomatic atherosclerotic disease most often involves the arteries supplying the heart, brain, kidneys, and lower extremities. **Myocardial infarction (heart attack), cerebral infarction (stroke), aortic aneurysms, and peripheral vascular disease (gangrene of the legs) are the major consequences of atherosclerosis.** The natural history, principal morphologic features, and main pathogenic events are schematized in Figure 11-16.

We next describe the features of atherosclerotic lesions that are typically responsible for the clinicopathologic manifestations.

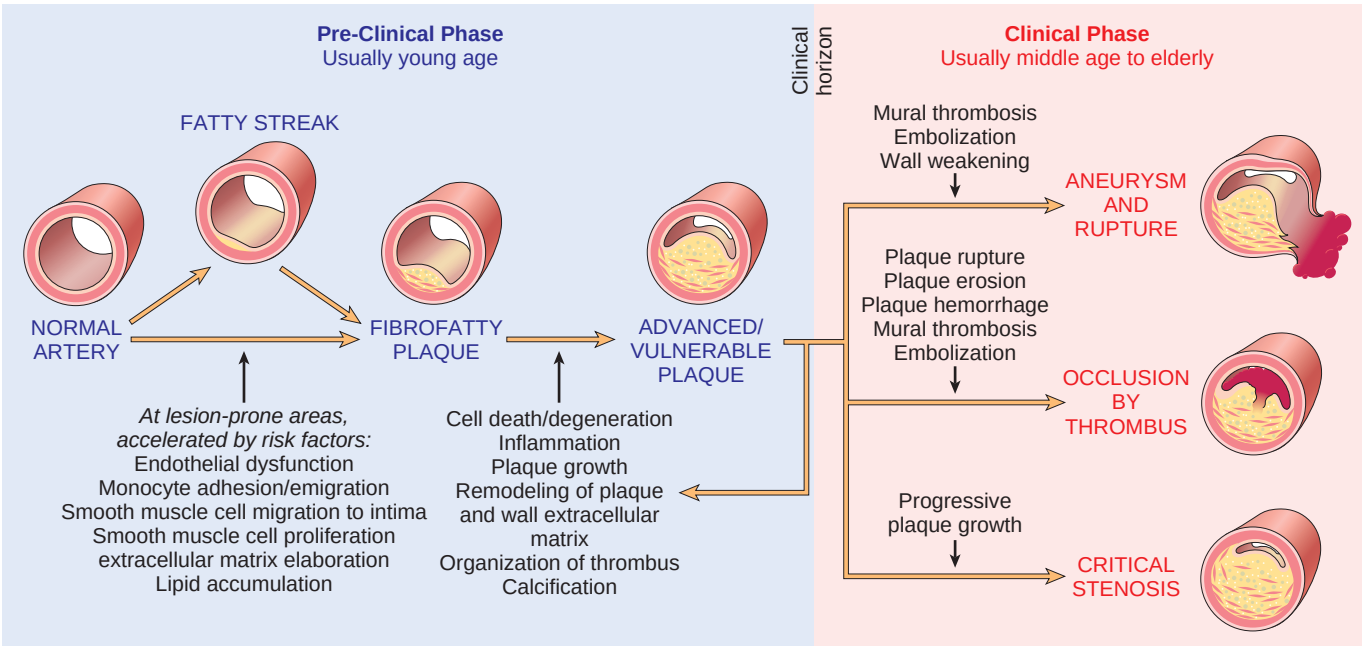

Figure 11-16 The natural history, morphologic features, main pathogenic events, and clinical complications of atherosclerosis.