A 61-year-old man with a 10-year history of hypertension, who had been generally well previously, noted intermittent non-radiating substernal discomfort with no dyspnea, diaphoresis, orthopnea, paroxysmal nocturnal dyspnea, or coryza-like symptoms. The discomfort was not related to exercise and was self-limited. The patient fell suddenly while talking with friends. He was transferred to the ER after cardiopulmonary cerebral resuscitation because of sudden cardiac arrest and died of acute myocardial infarction (AMI).

An autopsy showed the arterial wall of the left anterior descending coronary artery was completely occluded (Fig. 1A). Histopathology revealed a ruptured atherosclerosis plaque with fresh thrombi formation (Fig. 1B), which was the cause of his AMI.

AMI, commonly known as a heart attack, results from interruption of the blood supply to a part of the heart, causing heart cells to die. This is most commonly due to occlusion (blockage) of a coronary artery following the rupture of the artery with thrombus formation by a vulnerable atherosclerotic plaque, which is an unstable collection of lipids (cholesterol and fatty acids) and white blood cells (especially macrophages) in the wall of an artery. The resulting ischemia (restriction of blood supply) and ensuing oxygen shortage, if left untreated for a sufficient period of time, can cause damage or death (infarction) of the heart muscle tissue (myocardium).

Fig. 1. (A) Grossly, an atheroma totally occludes the left anterior descending coronary artery. (B) Histopathology reveals a ruptured atherosclerosis plaque with fresh thrombi formation (hematoxylin and eosin ×100).
Further reading

