Cardiovascular disease (CVD) is a major health concern in the United States, with a significant number of individuals affected by various conditions. Hypertension affects 75 million people, while coronary heart disease affects 25.155 million, myocardial infarction 790,000, heart failure 6.5 million, and stroke 7.95 million. These numbers cannot be simply added due to the presence of comorbidities. The underlying pathology of CVD involves the buildup of plaque within arterial walls, a process known as atherosclerosis. This starts with damage to the endothelial cells, triggering an inflammatory response involving phagocytes and monocytes. These monocytes transform into foam cells, ingesting oxidized cholesterol and forming fatty streaks within the vessels. Intracellular microcalcification then occurs, forming deposits within the vascular smooth muscle cells. A protective fibrin layer, or atheroma, forms between the fatty deposits and the artery lining. Atheromas produce enzymes that cause the artery to enlarge over time, compensating for the narrowing caused by the plaque buildup. This process, while seemingly beneficial, can lead to further complications.