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in Inflammation Resolution; 2.3.2. Cellular Effectors; 2.3.2.1. Leukocyte Recruitment; 2.3.2.2. Neutrophils; 2.3.2.2.1. Neutrophil-Mediated Cardiac Injury; 2.3.2.2.1.1. Reactive Oxygen Species; 2.3.2.2.1.2. Granule Toxicity; 2.3.2.3. Mononuclear Cells; 2.3.2.3.1. Monocytes; 2.3.2.3.2. Macrophages; 2.4. Reverse Remodeling
2.5. Clinical Implications: Is There a Causal Link Between Dysequilibrated Inflammation and Remodeling?References; Chapter 3: The Role of Inflammation in Myocardial Infarction; 3.1. Introduction; 3.2. Role of the Inflammatory Response Before MI; 3.2.1. Development of the Atherosclerotic Plaque; 3.2.2. Immune Cells Involved; 3.2.3. Maturation and Rupture of the Atherosclerotic Plaque; 3.3. The Role of the Inflammatory Response in MI; 3.3.1. MI and Wound Healing; 3.3.2. Humoral Immune Response Post-MI; 3.3.2.1. Cytokines; 3.3.2.2. Chemokines; 3.3.3. Cellular Immune Response Post-MI
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3.4.2.1. Current Pharmacotherapy Targeting Inflammation After MI

Sommario/riassunto

Inflammation in Heart Failure, edited by W. Matthijs Blankesteijn and Raffaele Altara, is the first book in a decade to provide an in-depth assessment on the causes, symptoms, progression and treatments of cardiac inflammation and related conditions. This reference uses two decades of research to introduce new methods for identifying inflammatory benchmarks from early onset to chronic heart failure and specifically emphasizes the importance of classifying at-risk subgroups within large populations while determining the patterns of cytokines in such classifications. Further, the book details c
