Rheumatoid Arthritis and Acute Coronary Syndrome: Changing Clinical Presentation

Abstract

Rheumatoid arthritis is a chronic systemic inflammatory disease. Rheumatoid arthritis can affect the pericardium, myocardium, and endocardium [1]. Moreover, RA increases cardiovascular morbidity and mortality by accelerated atherosclerosis. Patients with RA are at a twofold increased risk for myocardial infarction and stroke [2]. The increased

Case Profile

A 40-year-old male was admitted with complaints of neck pain, back pain and gum bleeding sudden in onset. His ECG was showing ST segment elevation myocardial infarction (Figure 1. AWMI). Routine blood investigations were normal, other investigations showed elevated acute phase reactants, elevated RA factor and troponins. He had history of RA for ten years and was on corticosteroids and Non-Steroidal Anti Inflammatory Drugs (NSAID’s). The Patient had no other associated risk factors and no family history of coronary artery disease. Echocardiography revealed regional wall motion abnormality Ejection fraction (EF) 30%. The Patient was taken for cardiac catheterization, Coronary angiography revealed proximal LAD has total occlusion. Successful PCI was done to LAD using drug eluting stent (DES) 3X24mm deployed at 12 ATM (Figure 2A,2B showing angiographic finding in LAD before and after PCI), patient showed prompt recovery and remained asymptomatic on follow up.

Discussion

Rheumatoid arthritis is a chronic systemic inflammatory disease RA can affect the pericardium, myocardium, and endocardium [1]. Moreover, RA increases cardiovascular morbidity and mortality by accelerated atherosclerosis. Cardiovascular mortality accounts for 40–50% of all deaths in RA. Patients with RA are at a twofold increased risk of myocardial infarction and stroke [2]. The increased
Cardiovascular disease risk in RA patients is independent of cardiovascular risk factors. Pathogenic mechanisms include pro-oxidative dyslipidemia, insulin resistance, prothrombotic state, hyperhomocysteinemia, and immune mechanisms such as T-cell activation that subsequently leads to endothelial dysfunction and arterial stiffness, which are the congeners of accelerated atherosclerosis observed in RA patients [3]. Markers of systemic inflammation (eg, interleukin [IL]–6, IL–17, tumor necrosis factor [TNF]) have been correlated with increased the risk of cardiovascular death in patients with rheumatoid arthritis [4]. The high burden of cardiovascular diseases in patients with autoimmune diseases has drawn attention to whether autoimmune diseases might worsen the prognosis for Coronary Artery Diseases. Outcomes after acute coronary syndrome have been compared in patients with autoimmune diseases, such as rheumatoid arthritis and Systemic lupus erythematosus [5], although some patients undergo coronary revascularization with percutaneous coronary intervention. In the modern era of widespread use of intervention therapy, the outcomes of PCI in patients with autoimmune diseases remain largely underdetermined. The impact of autoimmune diseases on patients undergoing PCI requires further elucidation [6].

Conclusion

Patients with Rheumatoid arthritis are at increased risk for the development of Coronary Artery Disease and associated morbidity and mortality. Cardiovascular disease often goes unrecognized in patients with Rheumatoid arthritis therefore it is prudent to carefully assess all patients with Rheumatoid arthritis. Substantial evidence suggests that chronic systemic inflammation contributes significantly to excess cardiovascular disease in Rheumatoid arthritis and that effective suppression of Rheumatoid arthritis -associated inflammation appears to reduce mortality. Classical cardiovascular risk factors play a role and may, in some cases, be exacerbated by the medications used to treat Rheumatoid arthritis. Prevention of cardiovascular disease in Rheumatoid arthritis requires a combined approach incorporating cardiovascular risk-factor screening, management and Coronary Interventions and most importantly effective and sustained control of Rheumatoid arthritis disease activity.

References