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Post - Myocardial infarction heart failure: present scenario and challenges ahead

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Overview

Despite spectacular success in the field of revascularization in coronary artery disease, it still remains the most prevalent cause of atherosclerotic cardiovascular diseases (ASCVD). Myocardial Infarction (MI) is the world's most common presentation of ASCVD and common cause of Heart Failure (HF). Even-though Heart Failure(HF) doesn't have a "Universal definition" like MI, it is generally described as "a clinical syndrome caused by any structural/ functional cardiac dysfunction that impairs the ventricle's capacity to fill/ eject blood". Yet there is one entity that requires considerable attention: Post-myocardial infarction heart failure (PMIHF).

Typically, it falls into 'acute decompensated heart-failure' category. Usually PMIHF is not noticed and treated at the right time, thus setting in progressive heart-disease affecting long-term cardiovascular (CV) outcomes. The treatment and outcome of acute-MIhave been revolutionized by the primary percutaneous coronary intervention (PPCI). PPCI is reperfusion of choice in developing world now. In India, contemporary PPCI is distinguished by improved door-to-balloon time, increasing use of radial-artery access, use of newergeneration drug-eluting stents, and personalized use of newer antiplatelet and antithrombotic agents thus improving CV outcomes.

The newer CV drugs [angiotensin-receptor blockerneprilysin-inhibitors (ARNI), sodium-glucose co-transporter-2 inhibitors (SGLT2i), trimetazidine, cardio-selective beta-blockers, Ivabradine, Ranolazine etc.] andthe artificial circulatory-devices [intra-aortic balloon counter-pulsation (IABP), Impella, left-ventricular assistdevices (LVAD), andextra-corporeal membrane oxygenator (ECMO)] have enhanced the results, although there is still a long way to go. Both physicians and researchers deem chronic heart disease as the most dreaded entity in the management of CAD. There is a lot of scope for the development of new treatment modalities to reduce mortality and morbidity, which requires sustained efforts and resources.

Unmet Challenges

- The overall impact of PPCI on HF is debatable due to varying definitions of HF in different trials.
- Despite the apparent strain of HF, several MI researches centred mainly on thrombosis, bleeding, and composite endpoints (e.g., MACE).
- HF has a high incidence of multi-organ-dysfunction, mortality-rate with recurrent and prolonged-hospitalization.
- PMIHF has been identified as an adverse prognosis determinant for nearly 50 years, in-spite of it being defined vaguely; but attempts to encourage myocardial recovery have failed to convert into therapeutic therapies.

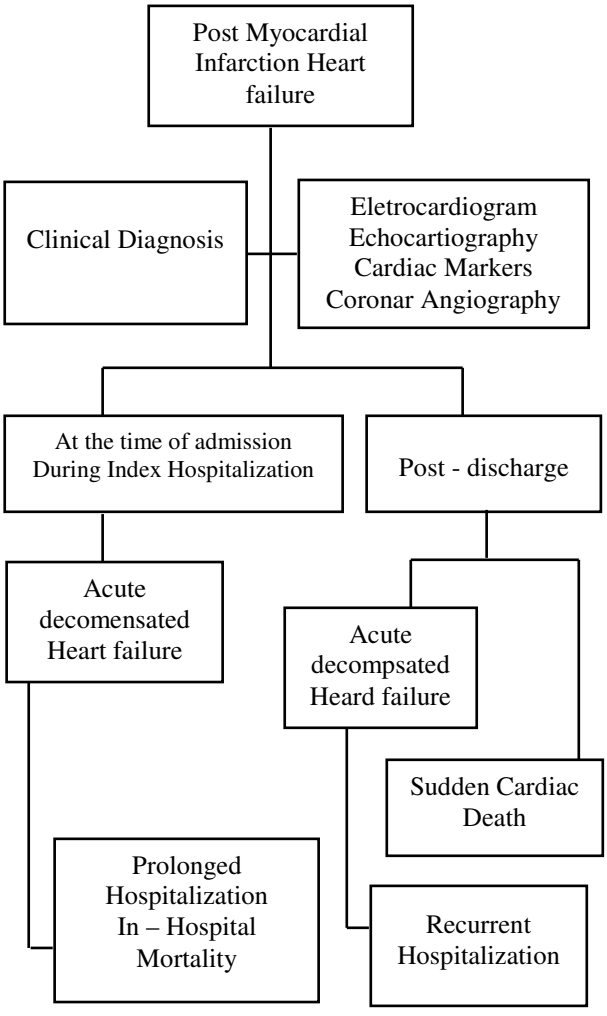
Incidence of PMIHF

PMIHF can either develop at presentation or can develop post-revascularization. Commonly, it indicates loss of large myocardial-territory to the extent of >50%,

thus reducing the cardiac-index (CI), elevating left ventricular end-diastolic pressure (LVEDP), higher pulmonary-capillary wedge pressure (PCWP), and in-turn higher systolic pulmonary-artery pressure (SPAP).

While 'Cardiogenic-shock' is recognized as the major complication of hemodynamic compromise, less severe form onthe HF spectrum are more common and have associated MACE [1]. The timing of PMIHF is clinically important to differentiate three main periods: HF at the appearance of the index MI, during the index hospitalization, and after discharge. Fig.-1 summarizes the clinical diagnosis, presentation, andoutcomes of PMIHF.

Fig-1: Clinical presentation, diagnosis and outcomes of post-myocardial infarction-heart failure



The frequency of HF in hospitalized patients with an AMI ranges between 14% to 36% among different studies like, GRACE registry (13%HF at admission and 5.6% HF during hospitalisation among 13,707 AMI-patients)[2], Spencer et-al (20.4%and 8.6% among 123,938) [3] and recently Bahit et-al (12% and 4% among 187,803) [4]. Peruvian registry (PERSTEMI) divided post-MI patients into those 'with HF' and those 'without HF'. Certain factors were independently associated with the increased risk of PMIHF like, LVEF<40%, age>75years, anterior-MI, and absence of electrocardiographic signs of reperfusion. The incidence of PMIHF was 48.8% and a higher risk of mortality(20.6%) was seen more commonly in patients with advanced age and those did not demonstrate negative T- wave inversions even after reperfusion [5].

In 2012, a French group as a part of FAST-MI registry, defined PMIHF a shaving at least one criteria i.e., previous history of CHF, CHF features on admission according to the Framingham criteria (Killip-class>2 on admission), Killip-class>2 at any time of hospitalization, and LVEF:40% at any time during hospitalization [6].

Post-MI 'systolic' LV-dysfunction

Approximately 40% of Post-AMIs, are associated with significant LV systolic dysfunction (LVSD). Prevalence of PMIHF is about 25% [7]. Post-MI LVSD faces a greater risk of adverse effects involving cardiovascular rupture, cardiac arrest, stroke, prolonged hospitalizations, ventricular arrhythmias, recurrent-MI, and death (SCD) [1].

Post-MI 'Diastolic' LV-dysfunction

A recent meta-analysis found that in approximately 10% of patients with preserved LVEF, and restrictive mitral filling pattern, the most extreme type of diastolic-dysfunction was reported. Additionally, the restrictive trend was associated with a bad outcome. However, it remains to be elucidated the true nature and importance of diastolic dysfunction after MI [8].

Mechanisms of PMIHF

HF occurs during index MI due to a combination of myocardial stunning, myocyte necrosis, decompensation of pre-existing HF,

or acute mitral regurgitation due to dysfunction of papillary muscle. HF can also be attributed to either of the above during hospitalization, exacerbated by fluid/contrast, renal dysfunction, or complications such as ventricular septal rupture or cardiac-tamponade [6]. The symptoms of cardiomyocyte-death and scar development alongside ventricular remodelling are mirrored in late-HF. Compensatory activation of renin-angiotensin and sympathetic nervous-system, pathological remodelling with wall thinning, ischaemic MR, and further cardiomyocyte failure are observed [9].

Recent advances in the management of PMIHF

Pharmacological advances in PMIHF

Targeted therapies to deter PMIHF, lag behind reperfusion developments, with valsartan/ sacubitril becoming the first experimental pharmacotherapy for HF to enter the mass-market in more than a decade. ARNI is now 'Class-I' recommendation supplementing ACE-i/ ARBs in chronic symptomatic HF patients with decreased LVEF and NYHA type-II/III [10]. The objective of PARADISE-MI trial was to resolve this very issue where MI patients with LVSD were randomizedto either valsartan/ sacubitril titrated to a target dose of 200mg twice-daily or ramipril titrated to a target dose of 5mg twice-daily [11].

Studies proved that non-fatal HF is closely associated with elevated midterm mortality risk in diabetic patients. This highlights the need for better treatment techniques to prevent HF development in diabetic patients with acute-MI[12]. The EMPA-REG OUTCOME analyzed empagliflozin (SGLT-2i) vsplacebo among diabetic patients. The empagliflozin group had a 35%relative risk reduction (RRR) in the HF hospitalization and a 38%RRR in the CV-deaths.

As for MI-related HF, the EMMY study [13] assessed the effect of empagliflozin on HF biomarkers in MI patients with and without type-2 diabetes [14]. From the device perspective, animal-based experiments suggest that unloading the myocardium to reduce myocardial-demand before revascularization may attenuate ischemia-reperfusion injury and reduce the infarction size.

Mechanical advances in PMIHF

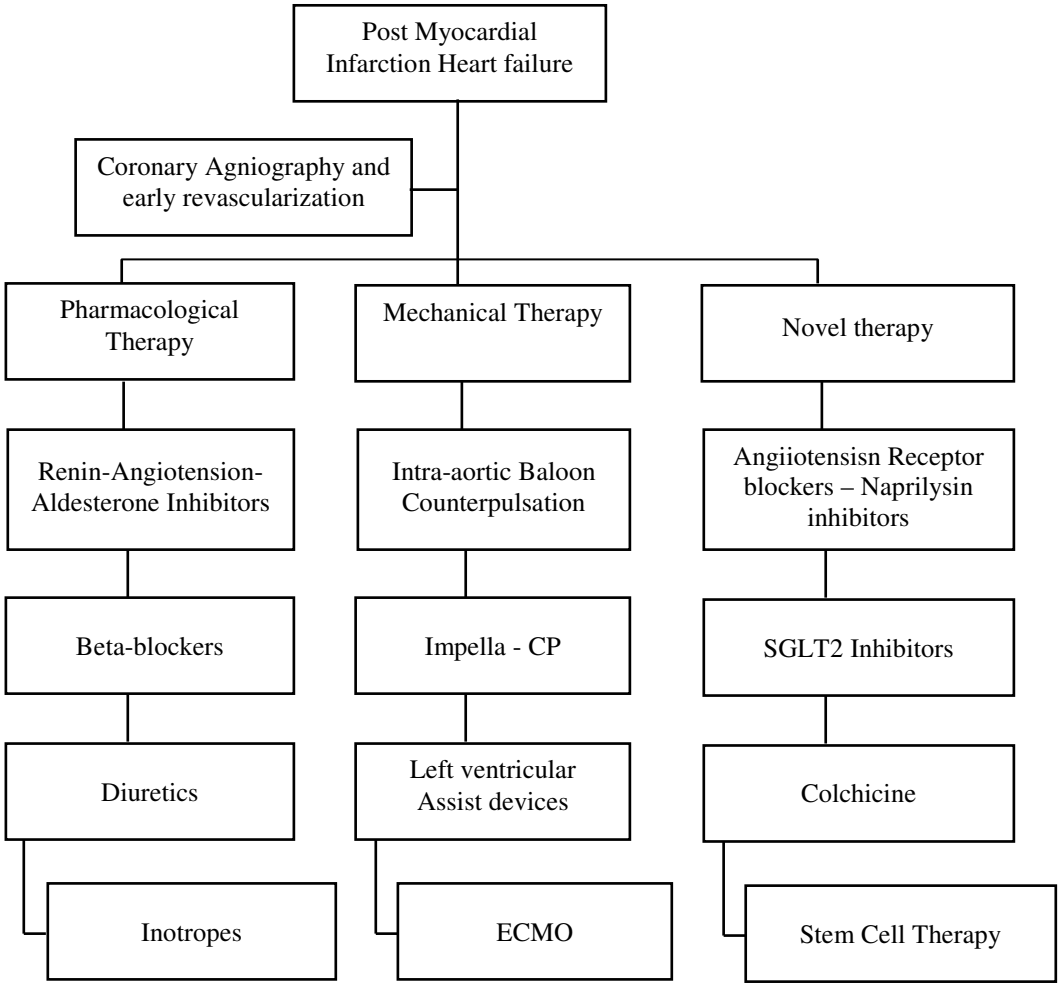
In cases where tissue hypoperfusion happens without cardiogenic shock, inotropic agents may be an alternative. However, some inotropes, such as digitalis and dobutamine, have contradictory effects, and others, such as milrinone, can affect the. Despite hemodynamic improvement there is no survival benefit observed [1].

IABP is recommended in cardiogenic-shocks refractory to pharmacological treatment. IABP decreases systolic-strain, raises diastolic pressure, and thus coronary perfusion and boosts LV activity. The primary limitations of IABP include the lack of active cardiac support, need for accurate synchronization with the cardiac-cycle, and the need for certain level of LV function. Like IABP, Impella-CP system have shown promising results in the DTU(Door to Unloading) study [15].

Usually, LVADs are used as connectors for regeneration and are built for optimum usage of 14 days. In general, there are three types of systems: percutaneous cardiopulmonary bypass, axial-flow pumps, and LA-FA LVADs [16]. The use of ECMO is another possible strategy. Indeed, some research indicates that ECMO may provide additional benefits in improving outcomes in patients with AMI complicated by cardiogenic-shock [17]. Fig-2 outlines the existing and emerging therapies of PMIHF.

In conclusion, understanding the mechanisms responsible for PMIHF is of crucial importance. In addition to identifying these mechanisms, health professionals must try to ascertain the existence of features of HF in the setting of MI. Patients admitted with CHF with AMI have high mortality-risk. Latest guideline-based therapy improves the mortality. Therefore, further attempts should be taken to facilitate the use of prescribed novel emerging therapies in patients with PMIHF.

Fig-2: A summery of guideline-based and novel emerging therapies for the management of post-myocardial infarction heart-failure



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