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The "Syndrome of Cardiogenic Insulin Resistance"

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Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

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Opinion Article

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ABSTRACT

Hyperglycaemia in patients admitted in intensive care units (ICU) with acute myocardial infarction is a common phenomena observed. This unique situation of cardiogenic insulin resistance does not spare diabetics or non -diabetics. It is in addition to the inherent insulin resistance that is a part of diabetes mellatus, type 2 (DM2). It is brought about by various cytokines released from the damaged heart muscle. This cardiogenic insulin resistance has cardiac as well as systemic effects. The grave and independent risk role in post myocardial infarction (MI) and the complications, of the cardiac insulin resistance are highlighted. The concerted action of cardiologist and endocrinologist while in hospital is called for, so as to cover the grey areas between the two specialities, which otherwise falls into no man's island!. The systemic insulin resistance, once the patient is back in home setting, would test the patience of the physician, as usual doses of insulin just do not work! The article aims at creating awareness regarding concerted effects of all concerned to deliver holistic treatment to the patients.

Keywords: Cardiac insulin resistance; post MI insulin resistance; stent restenosis.

1. INTRODUCTION

Cardiogenic insulin resistance (IR), manifesting as hyperglycaemia, is a well recognised entity in patients admitted with acute myocardial infarction (MI) in ICU, diabetic or non diabetic. IR, in the setting of acute MI has ominous prognosis [1]. This might sometimes be missed as un-

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controlled or undetected Diabetes mellatus type 2 (DM2). Cardiogenic IR is additional to, IR that is inherent to DM2. The result is that insulin doses that controlled DM2 before, fail to respond in the setting of cardiogenic IR. This cardiogenic IR is true even in cases of nondiabetic patients [2].

The Cardiogenic IR is either Cardiac, with or without systemic IR. Cardiac IR is widely discussed in the literature because of its grave consequences - like high incidence of heart failure (HF) [3,4], incomplete myocardial perfusion and cardiac muscle function (CMF) impairment [5,6]. Size of infarct is believed to be bigger, impairment of myocardial and microvasculature injury after ST elevation myocardial infarction (STEMI) injury [7,8]. The prognostic relevance of hyperinsulinemia in STEMI patients [9,10] and its relationship with coronary flow [11] are less well acknowledged. IR is related to ischemic myocardial injury after elective percutaneous coronary intervention (PCI) Cardiac remodelling and stent re –stenosis [12] is more common with Cardiac IR. Tachyarrhethmias are reported more [13] compared to controls. Data connecting metabolic syndrome to the final infarct size are less clear [14]. However, evidences support direct proatherogenic effects of IR [15] as well as its direct adverse effects on myocardial contractility. IR was related to ischemic myocardial injury after elective PCI [16] and in diabetic ketoacidosis (DKA) with acute MI. [17] the cardiologist can not ignore, the extra risk to patient because of the risks from cardiac IR, in addition to the seriously compromised haemodynamic state of the heart. But the scourge of super speciality is that DM2 is seen as not the concern of a cardiologist, but is the concern of an endocrinologist or a physician. Though DM, by virtue of Cardiac IR has many complications, having bearing on cardiology, the cardiologist passes the buck to the endocrinologist. The latter's concern is solely to control hyperglycaemia and its complications, unmindful of cardiac hazards, because it is not in his domain. Thus, the monitoring, follow up and management, of issues arising out of Cardiac IR are likely to fall in no man's island. Can this be holistic treatment? Hence every case of cardiac IR should open up close communication between the cardiologist and endocrinologist/ physician who are in charge of the DM2 aspect.

Lastly though stress hormones are implicated in the blunted action of insulin in the early setting, the real culprit found to cause the damage to heart is the cytokinines, tumour necrosis factor 1 (TNF) Alfa. Especially in cardiac IR. [18]. Even tumour necrosis factor (TNF) Alfa i inhibitors, like etanercept were effectively used in experimental animals in protecting against the deleterious affects of Cardiac IR on heart. Of special interest is the systemic IR following acute MI. This persists for a week or two, even after the patient is discharged from ICU. Now the patient is in care of a family physician. He has little information or knowledge of persistent IR for some time even after an acute MI. It is certain to baffle him as to why the expected doses of insulin or doses that used to keep patients Blood sugar under control, just do not work any longer!. This leads naturally to additions and deletions of various anti diabetic armamentaria at his disposal. The result is a bigger mess ! So there is a need to take into confidence, the family physician also, regarding issues concerning the cardiogenic IR. Inflammatory cyt kinines like, interleukin 6 (IL -6,) C- reactive protein (CRP) and plasminogen activation inhibitor factor 1 etc. have been implicated in the pathogenesis of cardiogenic systemic IR. The best way to cope up with systemic IR due to MI, Is to buy time till the blocking effects of the cytokines wares off when the regular previous doses of insulin become effective. The purpose of this article is to create awareness about cardiogenic IR, as to its role in damage to ischemic heart it causes and management problems of DM2 due to systemic IR. Cardiogenic insulin resistance (IR), manifesting as hyperglycaemia, is a well recognised entity in patients admitted with acute myocardial infarction(MI) in ICU., diabetic or non diabetic. IR in the setting of acute. MI has ominous prognosis [1]. This might sometimes be missed as un-controlled or undetected DM2. Cardiogenic IR is additional to it, IR that is inherent to DM2. The result is that insulin doses that controlled DM2 before, fail to respond in the setting of Cardiogenic IR. This cardiogenic IR is true even in cases of nondiabetic patients [2]. The Cardiogenic IR is either Cardiac, with or without systemic IR. Cardiac IR is widely discussed in literature because of its grave consequences- like high incidence of HF [3,4], incomplete myocardial perfusion and cardiac muscle function (CMF)impairment [5]. Size of infarct is believed to be bigger. Impairment of myocardial and microvasculature injury after STEMI injury [7,8]. The prognostic relevance of hyperinsulinemia in ST elevation myocardial infarction (STEMI) patients [9,10] and its relationship with coronary flow [11] are less well acknowledged.

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2. CONCLUSION

The purpose of this article is to create awareness of cardiogenic IR, as to its role its role in damage to ischemic heart and management problems of DM2 due to systemic IR.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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