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Biochemical Markers of Metabolic Syndrome - A Comprehensive Overview

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Abstract: *Metabolic Syndrome (MetS) is a cluster of interrelated biochemical and physiological abnormalities, including obesity, hyperglycemia, dyslipidemia, and hypertension. The intricacy of its presentation and the interconnectedness of its components makes MetS a complex health issue. This review aims to provide a comprehensive overview of the biochemical markers associated with MetS and their implications in disease progression and management. The review also sheds light on the potential of these markers as therapeutic targets, thus paving the way for more effective strategies in the management and prevention of MetS.*

Key Words: *Metabolic Syndrome (MetS), Biochemical Markers, Dyslipidemia*

1. INTRODUCTION:

A constellation of interconnected risk factors of metabolic origin known as the metabolic syndrome (MetS) increases the risk of cardiovascular diseases (CVD) and type 2 diabetic mellitus (T2DM) (Alberti, Zimmet, & Shaw, 2006). Central obesity, dyslipidemia, hypertension, and insulin resistance are all frequently linked to MetS. MetS prevalence is rising globally as a result of sedentary lifestyles and poor eating practises (O'Neill & O'Driscoll, 2015). This review will look at the biochemical indicators of MetS, giving readers a thorough grasp of their functions and potential applications to the therapeutic treatment of this condition.

2. LIPID PROFILE:

High triglycerides (TG), low high-density lipoprotein cholesterol (HDL-C), and increased low-density lipoprotein cholesterol are the three markers of dyslipidemia, a typical MetS characteristic (LDL-C). Cardiovascular risk is linked to high TG levels, and there is a significant negative association between HDL-C levels and cardiovascular disease (Grundy et al., 2004). LDL-C is sometimes referred to as "bad cholesterol," because elevated levels of this lipid are linked to atherosclerosis.

3. GLUCOSE HOMEOSTASIS MARKERS:

MetS is characterised by insulin resistance, and fasting blood glucose, insulin, and glycosylated haemoglobin (HbA1c) are essential markers of glucose homeostasis in its diagnosis (Johnson, Shrewsbury, & Marques, 2017). Impairment in glucose tolerance, which is a precursor to T2DM, is indicated by elevated fasting glucose levels. The long-term glucose control indicator HbA1c, which is frequently elevated in MetS and linked to a higher risk of T2DM and CVD, is also elevated.

4. INFLAMMATORY MARKERS:

A significant factor in the aetiology of MetS is persistent low-grade inflammation. Individuals with MetS frequently have higher levels of the systemic inflammation marker high sensitivity C-reactive protein (hs-CRP) (Ridker, 2003). Furthermore, adipose tissue-produced cytokines such tumour necrosis factor-alpha (TNF-) and interleukin-6 (IL-6) contribute to inflammation and insulin resistance (Hotamisligil, 2006).

5. ADIPOKINES:

Adipokines, which include adiponectin, leptin, and resistin, are a class of bioactive molecules secreted by adipose tissue in addition to storing fat (Ouchi, Parker, Lugus, & Walsh, 2011). The levels of adiponectin, which improve insulin

sensitivity, are frequently inversely linked with MetS. Leptin controls how much food is consumed and how much energy is expended; obesity is associated with leptin resistance. Insulin resistance and inflammation are related to resistin.

6. DYSLIPIDEMIA AND METABOLIC SYNDROME:

A distinctive lipid profile known as atherogenic dyslipidemia, which includes high TG, low HDL-C, and a preponderance of tiny, dense LDL-C particles, characterises MetS. (Grundy et al., 2004). Each of these elements raises the possibility of developing cardiovascular disease. Because they are more susceptible to oxidation and penetrate the artery wall more deeply than bigger, buoyant LDL-C particles, small, dense LDL-C particles are produced when TG levels are elevated (Packard, 2006). Low HDL-C levels also decrease the ability to transport reverse cholesterol, which hinders the clearance of cholesterol from the artery wall (Rader, 2006).

7. GLUCOSE HOMEOSTASIS DISRUPTIONS IN METABOLIC SYNDROME :

One of the main characteristics of MetS and a major factor in its development is insulin resistance (Reaven, 1988). In order to maintain normoglycemia, insulin resistance causes compensatory hyperinsulinemia. This compensatory mechanism eventually breaks down, resulting in reduced glucose tolerance and ultimately T2DM. A higher risk of microvascular and macrovascular problems has been linked to elevated HbA1c levels, a sign of poor long-term glucose control (Selvin et al., 2010). Therefore, the course of MetS and its accompanying consequences are severely affected by the disruption of glucose homeostasis, which is shown by higher fasting glucose, insulin, and HbA1c levels.

8. INFLAMMATION AND METABOLIC SYNDROME :

Inflammation plays a variety of roles in MetS. Elevated levels of hs-CRP are a persistent sign of chronic low-grade inflammation, which is a characteristic of MetS. (Ridker, 2003). Visceral adipose tissue, in particular, is an active endocrine organ that secretes a number of pro-inflammatory cytokines, such as TNF- and IL-6 (Hotamisligil, 2006). The systemic inflammation and insulin resistance caused by these cytokines feed into each other, exacerbating the MetS-related metabolic abnormalities.

9. ADIPOKINES IN METABOLIC SYNDROME:

The interplay between adipose tissue, the liver, the muscles, and the immune system is mediated by adipokines, which are important players in the pathophysiology of MetS. (Ouchi et al., 2011). In people with MetS, adiponectin levels are frequently lowered, and this insulin-sensitizing adipokine is inversely correlated with insulin resistance and the likelihood of T2DM (Yamauchi & Kadowaki, 2008). Obesity frequently has high levels of leptin, an adipokine that suppresses hunger, showing leptin resistance (Considine et al., 1996). Although its function in humans is still debatable, resistin, another adipokine, is connected to inflammation and insulin resistance (Steppan et al., 2001).

10. POTENTIAL THERAPEUTIC TARGETS:

Numerous possible treatment targets are provided by the intricate pathophysiology of MetS. By addressing dyslipidemia with pharmacological treatments like statins and fibrates as well as lifestyle changes, the risk of CVD can be decreased (Grundy et al., 2004). Metformin and thiazolidinediones are examples of insulin sensitizers that can reduce insulin resistance and support glucose homeostasis (DeFronzo & Goodman, 1995). Weight loss and the use of anti-inflammatory medications are anti-inflammatory measures that may help lessen the chronic inflammation linked to MetS. (Hotamisligil, 2006). Last but not least, focusing on adipokines like adiponectin, leptin, and resistin offers fresh treatment possibilities. As an illustration, the creation of adiponectin analogues or substances that boost adiponectin expression may enhance insulin sensitivity and glucose balance (Yamauchi & Kadowaki, 2008). Leptin sensitizers, a strategy to combat leptin resistance, may also aid in regulating appetite and energy balance (Heymsfield et al., 2014).

11. CONCLUSION:

The combination of dyslipidemia, poor glucose homeostasis, inflammation, and aberrant adipokine production characterises MetS, a complicated multifactorial illness. These components' interdependence produces a vicious loop that aggravates the MetS-related metabolic abnormalities. Effective management and prevention of MetS depend on an understanding of the functions and implications of the biochemical markers linked to these abnormalities. Additionally, these indicators provide potential MetS treatment targets, opening the door for the creation of more specialised and successful therapy approaches. Future studies should concentrate on unravelling the intricate interactions between these indicators and their functions in the aetiology of MetS as well as investigating potential new treatment targets for this complicated condition.

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