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The Covid-19 in Young Adults with Non-Obstructive Coronary Artery Disease

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Abstract

Acute myocardial infarction can result from a systemic inflammatory response triggered by a cytokine storm in severe COVID-19 cases, affecting people of all ages and with no known risk factors. We present the case of a 36-year-old man with morbid obesity and well-controlled asthma that developed COVID-19-induced acute respiratory distress syndrome, necessitating mechanical ventilation and, eventually, Extracorporeal Membrane Oxygenation (ECMO), who developed myocardial infarction on day 10 of admission and died on day 15 of admission due to COVID-19 infection, squealed. In the absence of obstructive lesions or plaque ruptures, a significant inflammatory response in young individuals with COVID-19-induced respiratory illness can lead to acute coronary syndrome.

Keywords: Coronary Artery Disease; Extracorporeal Membrane Oxygenation; Coronary Syndrome

Introduction

In 1973, Kemp introduced the concept of Coronary Syndrome X (CSX) into clinical practice to define patients with angina during physical activity and normal coronarography. This term has developed to encompass a broader spectrum of people, including those who suffer from angina regardless of the underlying cause or the absence of significant changes in their coronary veins. Cardiomyopathies, left ventricular hypertrophy, and significant valvular disease are typically excluded from this criteria, although not always. According to numerous authors, angina and micro vascular dysfunction are frequently connected to this illness [1]. Some authors, on the other hand, argue that certain disorders that can contribute to micro vascular dysfunction, such as hypertension or diabetes, should be excluded.

Angina during physical exertion, significant changes in the ST segment during an exercise test, and angiographic ally smooth coronary arteries in the absence of other cardiac or systemic diseases that can lead to vascular dysfunction (e.g., hypertension and diabetes) are the classic definitions of CSX. The fundamental criticism is that it is impossible to include all patients with micro vascular dysfunction in these criteria, making it unsuitable for research and therapeutic use. As a result, the scientific community has recently adopted new, more relevant definitions [2].

Lanza proposes that CSX is defined by chest pain that occurs primarily during physical activity, established ischemia or diminished coronary reserve using noninvasive provocation tests, normal (or nearly normal) coronary arteries at angiography with stenosis less than 20%, and exclusion of other specific diseases like Prinzmetal's angina, cardiomyopathies, and valvular heart disease [3]. As a result, the CSX now includes not only conditions with reduced coronary reserve, which can be determined using modern diagnostic procedures such as ergometer, stress induced myocardial scintigraphy, pharmacological stress tests, or an ECG Holter monitor test, but also diseases like hypertension and diabetes, which are common causes of micro vascular dysfunction.

Cannon and Epstein presented a new concept of micro vascular angina in 1985 as a result of their new understandings. CSX is defined as chest pain associated with normal coronary angiography and increased sensitivity to microcirculation to vasoconstrictive effects or aberrant vasodilation related to endothelial dysfunction [4]. Endothelia (a vasoconstrictor) levels in these patients' plasma are considerably higher. This was an attempt to bring the pathophysiology of the clinical condition together, emphasize the importance of endothelial dysfunction, and create a more homogeneous patient group. However, when it becomes clearer that endothelial dysfunction is only one aspect of the pathophysiological cascade, this approach is insufficient [5]. Kothawade et al. proposed a new name for micro vascular coronary dysfunction in 2011.

The CSX is defined as decreased coronary reserve and/or coronary endothelial dysfunction, and it is clinically manifested by a trio of symptoms: Chronic chest pain, non-obstructive coronary disease (coronary artery stenosis less than 50% on coronarography), and noninvasively confirmed ischemia [6]. Invasive coronary reactivity testing is the gold standard for diagnosis.

Obstructive Coronary Disease (CAD) is defined as a stenosis of a coronary vessel larger than 50% on coronarography, whereas non-obstructive coronary disease (non-CAD) is defined as a stenosis of a coronary artery greater than 50% on coronarography, regardless of definitions and nomenclature [7]. This criterion is shared by all definitions and understandings of this complex clinical condition. However, there are still certain misconceptions about non-CAD that lead to inconsistencies in results and observations. As a result, new definitions should be developed in order to allow for the unique and exact definition of this clinical entity in all of its facets.

Sudden Cardiac Death

Sudden Cardiac Death (SCD) and Coronary Heart Disease (CHD) have a high association. Clinical and post-mortem research, as well as data from death certificates, found that 62%-85% of out-of- hospital SCD patients have prior CHD, 10% have other structural cardiac abnormalities, and 5% have no structural cardiac abnormality. A SCD surveillance study from Ireland found that the majority of episodes occurred at home, and that successful SCD resuscitation was strongly linked to ventricular fibrillation as the presenting rhythm.

A recent study found signs of active Coxsackie B virus infections in persons who died of MI when compared to controls, and the authors speculated that disruption of dystrophin in endo myocardial tissue could be one of the underlying mechanisms [8].

In 15% of patients with CHD, SCD is the first clinical coronary episode. Furthermore, SCD is the most common cause of mortality in CHD patients, accounting for 30% to 50% of all deaths. After an acute MI, the risk of SCD is the same for STEMI and NSTEMI, as well as symptomatic and silent MI [9]. Approximately half of patients who have had a MI and are followed for around four years die suddenly in the first year, and one-quarter in the first three months.

Patients with a lower than LV ejection fractions are at a higher risk. Despite the fact that patients with a history of previous resuscitated SCD, MI, or heart failure have the highest risk of SCD, around 80% of SCD episodes occur in asymptomatic patients without such a history. Women with CHD had half the risk of sudden death as men, and the risk of SCD in asymptomatic adults is roughly proportional to the risk of CVD, with the incidence trailing men by more than 10 years. MI doubles the risk of SCD in both sexes when compared to angina. When compared to angina, MI doubles the risk of SCD. The following primary findings were noticed in the Oregon Sudden Unexpected Death Study, which included approximately 1,500 cases of sudden cardiac arrest. To begin with, there was no significant gender difference in obesity, dyslipidemia, LVH, or MI history.

Second, women were less likely than men to have significant LV dysfunction (or 0.51) and CHD (or 0.34). The outcomes of women who experienced an episode of out-of-hospital SCD were studied in a retrospective cohort study of 9,651 men and women. Women were less likely than males to experience ventricular fibrillation as their first rhythm problem (25% *vs.* 43%), but they were more likely to have pulseless electrical activity/asystole (73% *vs.* 55%). After adjusting for the prevalence of atrial fibrillation and other factors, women had a similar rate of survival until hospital discharge (29% *vs.* 28%).

Non-obstructive Coronary Artery Disease

It's critical to stress that non-CAD is identified by exclusion. All non-cardiac causes of angina must be ruled out, including musculoskeletal discomfort, gastrointestinal diseases, pulmonary causes, and numerous psychiatric disorders. Noninvasive diagnostic procedures are used in individuals with suspected angina to confirm ischemia by looking for substantial ST-T alterations (based on defined diagnostic criteria) mostly during exertion. Ergometer or exercise myocardial perfusion scintigraphy is the most basic test of physical activity.

There are currently no established criteria for distinguishing between obstructive and non-obstructive illness in patients who have a positive stress test. Some writers, however, believe that many requirements, such as increased pressure and pulse pressure product during stress, are required to cause ST alterations in non-CAD patients.

Patients who have a positive stress test should have a more invasive diagnostic work-up to confirm or rule out obstructive alterations in the coronary arteries. Although there are other noninvasive procedures like as Multi-Slice Computerized Tomography, direct catheterization of arteries (coronarography) is usually used. Some authors advocate an acetylcholine or ergonovine test (intracoronary or intravenously) in patients with normal coronarography to rule out major artery spasm. Unfortunately, this medication is not included in conventional clinical work-up due to the risk of severe vasospasm and hypotension.

After all non-cardiac causes have been ruled out; CAD is diagnosed based on the clinical presentation of chest discomfort and diagnostic techniques. The first procedure is a noninvasive physical activity test (ergometry, or rarely myocardial stress scintigraphy). Coronagraphy is used in individuals who have a positive stress test to confirm or rule out obstructive stenosis (50%) of the epicardial arteries. The ergonovine or acetylcholine test can diagnose significant artery spasm, but it is not part of normal clinical work-up due to the high risk.

At this stage, the routine clinical examination is completed. Additional diagnostics for a more precise diagnosis of non-CAD are exclusively utilized in research. Some pharmacological tests can be used to confirm altered vasodilation or augmented vasoconstriction in patients with suspected ischemic aetiology (microvascular dysfunction). The cholinergic test or electro stimulation are the most often used tests to determine endothelium dependent dysfunction and adenosine, pyridamole, or palavering tests to determine non-endothelial dependent dysfunction. An ergo ovine test or a cold pressure test can be used to diagnose vascular constriction [10]. Following the completion of one of these tests, induced ischemia in the microvasculature area 500 m is required.

Ischemia is indirectly observed with invasive methods such as the measurement of Coronary Flow Reserve (CFR) or noninvasive methods such as myocardial scintigraphy, MR, or PET because small blood vessels cannot be seen on coronarography. To confirm a non-ischemic foundation of the disease, scintigraphy of sympathetic cardiac innervation can be performed to demonstrate afferent dysfunction and direct heart stimulation with dobutamine or an electro stimulator can be utilized to show efferent dysfunction (altered pain perception). There are also other tests available, such as insufficiently standardized psychological examinations for determining the habitual component of the condition and the lack of tolerance to repetitive pain stimuli.

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