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RESEARCH ARTICLE

TAKOTSUBO CARDIOMYOPATHY (STRESS CARDIOMYOPATHY)INDUCED BY COVIDINFECTION: A CASE REPORT

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Abstract

Stress cardiomyopathy (also called apical ballooning syndrome, takotsubo cardiomyopathy, broken heart syndrome, and stress-induced cardiomyopathy) is a syndrome characterized by transient regional systolic dysfunction, principally, of the left ventricle (LV), mimicking myocardial infarction (MI), but in the absence of angiographic evidence of obstructive coronary artery disease or acute plaque rupture. In most cases of stress cardiomyopathy, the regional wall motion abnormality extends beyond the territory perfused by a single epicardial coronary artery. The term "takotsubo" is taken from the Japanese name for an octopus trap, which has a shape that is similar to the systolic apical ballooning appearance of the LV in the most common and typical form of this disorde; mid and apical segments of the LV are hypokinetic/akinetic, and there is hyperkinesis of the basal walls.

Introduction:-

Case Report:

A 44 years old lady with no chronic medical illness, height 154 cm, weight 46 kg, presented on 01/03/2023 with complain of cough and fever, and recurrent attacks of dizziness and vertigo, Covid 19 reported positive, Chest x ray at the beginning demonstrated bronchitis, and CT brain revealed sinusitis and mastoiditis.

The patient was admitted under medical care, in the isolation room, then suddenly she deteriorated, with signs of tachypnea, desaturation (saturation 72% on room air),tachycardia, and hypotension (cardiogenic shock).Her lab revealed increased troponin to 282.

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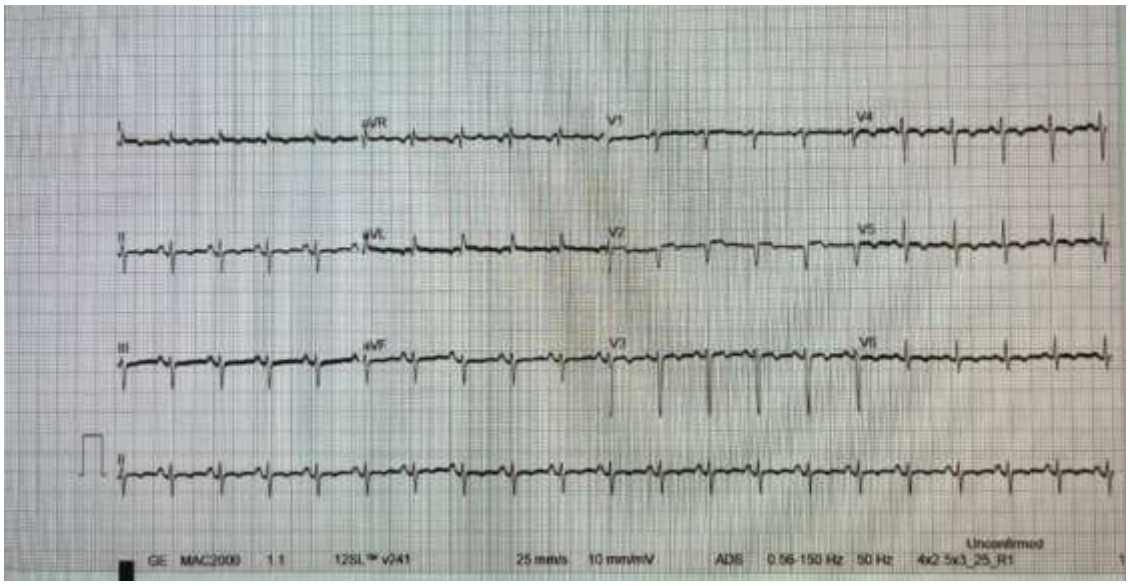


Figure 1:- ECGshowedsinus tachycardia 121/minute, left axis deviation.

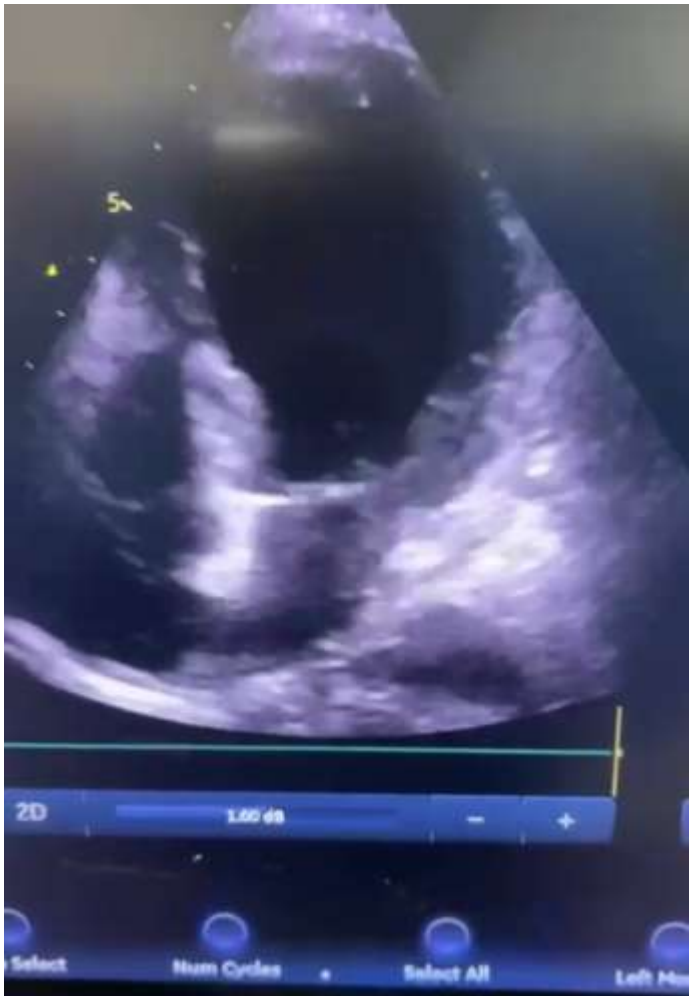


Figure 2:- Bedside transthoracic echocardiography: revealed depressed ejection fraction EF less than 20%, with regional wall motion abnormality mid and apical segments of the LV are akinetic, and there is normal kinesis of the basal walls, with preserved LV dimensions, RV was within normal in dimensions and function.

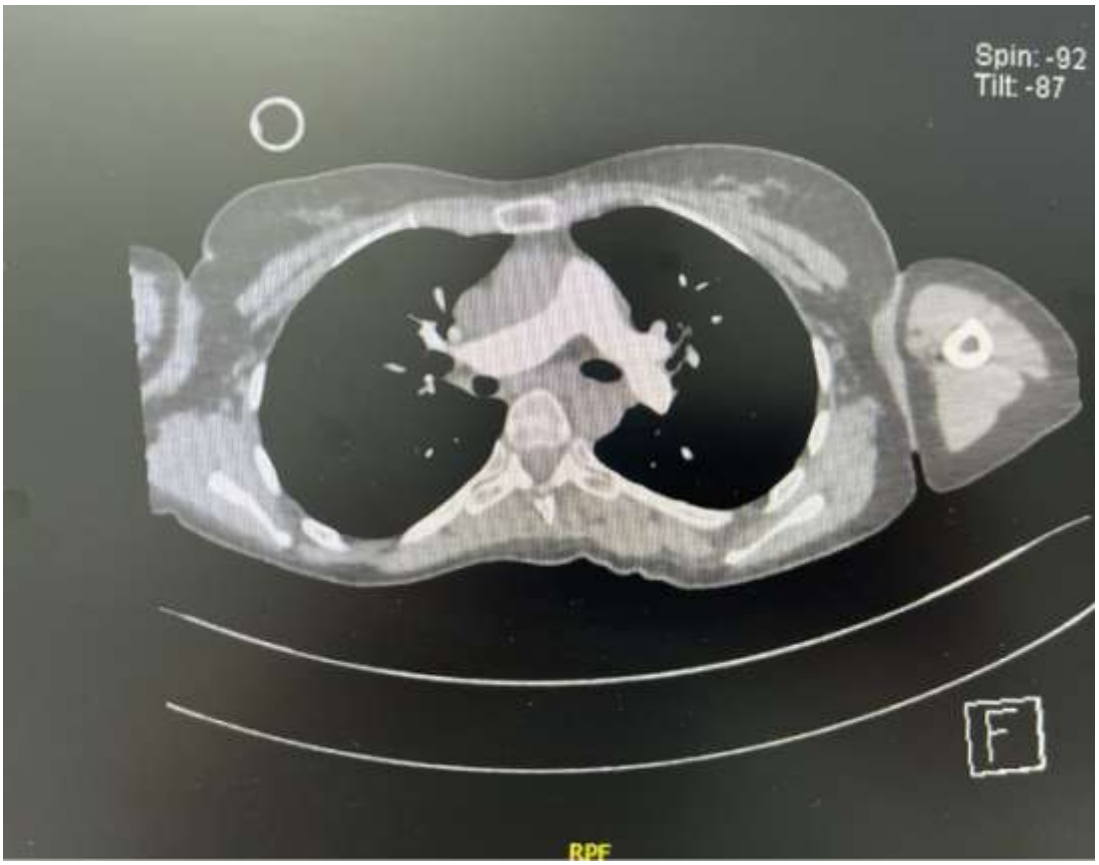
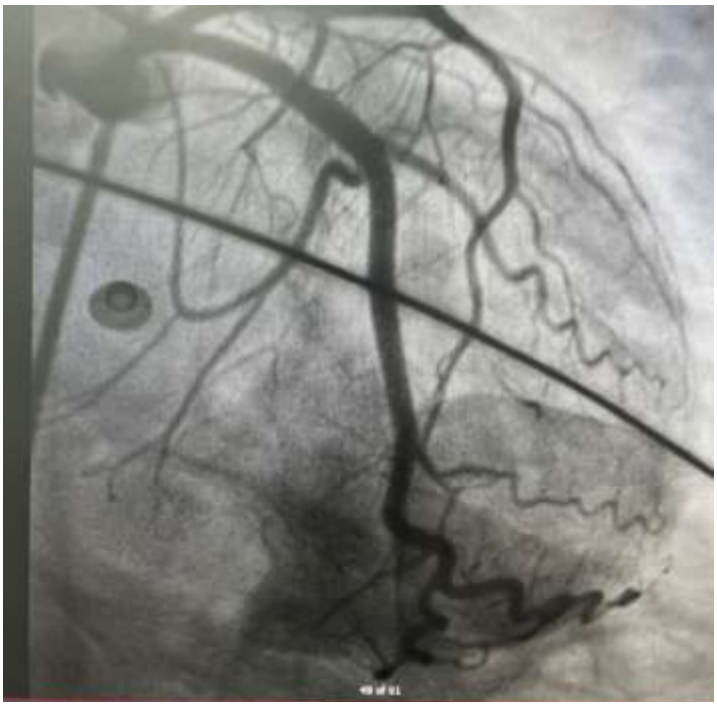


Figure 3:- CT pulmonary angiogram did not show PE.



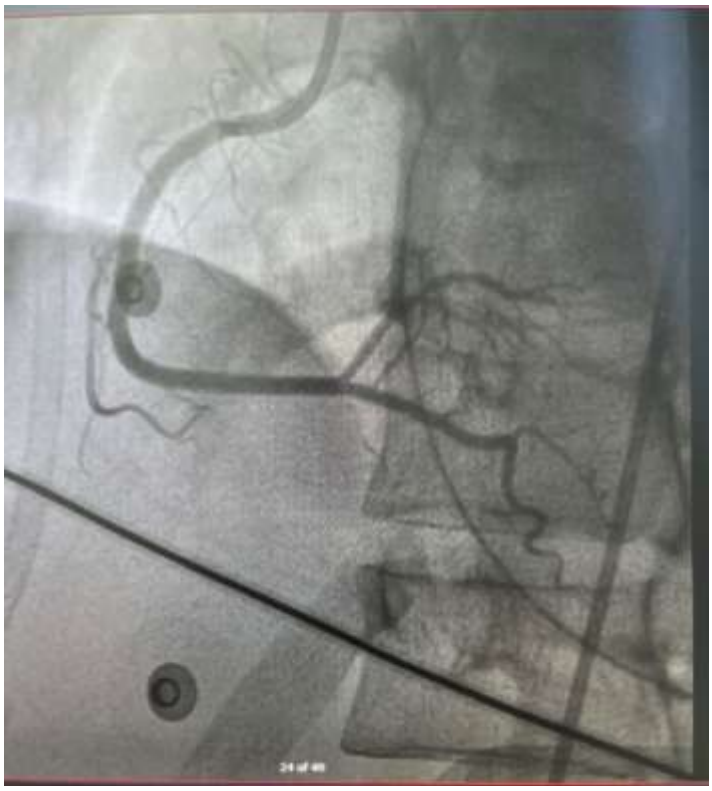


Figure 4:- Photos Coronary angiographywhich wasdone and reported (normal coronary arteries).

Repeated chest x ray revealed pneumonia in the right middle and lower lobe.

She was intubated and ventilated and commenced on inotropes, and gradually she was weaned off inotropes and extubated after 6 days with repeated transthoracic echocardiography revealed improved kinesis on the apical and mild segments.

Discussion:-

Coronavirus disease 2019 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Patients with COVID-19 are typically presenting with symptoms and signs of respiratory tract infection, but cardiac manifestations, including signs of myocardial injury, are common.

Possible causes of myocardial injury in patients with COVID-19 include hypoxic injury, stress (takotsubo) cardiomyopathy, ischemic injury caused by cardiac microvascular dysfunction, small vessel cardiac vasculitis, endotheliitis, or epicardial coronary artery disease (with plaque rupture or demand ischemia); right heart strain (acute cor pulmonale, with causes including pulmonary embolism, adult respiratory distress syndrome, and pneumonia), myocarditis, and systemic inflammatory response syndrome (cytokine storm). However, the contribution of each of these causes to myocardial injury and adverse cardiovascular outcomes in this setting has not been determined.

The diagnostic criteria, all four of which are required for the diagnosis:

- Transient LV systolic dysfunction (hypokinesis, akinesis, or dyskinesis). The wall motion abnormalities are typically regional and extend beyond a single epicardial coronary distribution; rare exceptions are the focal (within one coronary distribution) and the global type.
- Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
- New electrocardiographic abnormalities (either ST-segment elevation and/or T wave inversion) **or** modest elevation in cardiac troponin.
- Absence of pheochromocytoma or myocarditis.

Approach to management; Stress cardiomyopathy is generally a transient disorder that is managed with supportive therapy. Conservative treatment and resolution of the physical or emotional stress usually result in rapid resolution of symptoms, although some patients develop acute complications such as shock and acute heart failure (HF) that require intensive therapy. Appropriate management of shock varies depending upon whether significant left ventricular outflow tract (LVOT) obstruction is present. HF management during acute presentation and following stabilization is generally performed according to standard guidelines for HF with reduced ejection fraction (HFrEF) except that particular care is taken to avoid volume depletion and vasodilator therapy in patients with LVOT obstruction. Recommendations for anticoagulation to prevent thromboembolism in patients with stress cardiomyopathy with LV thrombus or severe LV systolic dysfunction are similar to those for patients post-MI.

Prognosis:

Although most patients with stress cardiomyopathy recover, the risk of severe in-hospital complications is similar to that in patients with acute coronary syndrome. As an example, the risk of a composite of catecholamine use, cardiogenic shock, the use of invasive or noninvasive ventilation, cardiopulmonary resuscitation, and death was 19.1 percent in patients with stress cardiomyopathy in the International Takotsubo Registry study versus 19.3 percent in a control group of patients with acute coronary syndrome. In-hospital mortality rates have ranged from 0 to 8 percent (4.1 percent in the International Takotsubo Registry study).

Conclusion:-

While myocardial injury in COVID-19 may have various clinical presentations, it is overall associated with high rates of complications and mortality and possible long-term cardiac impairments in survivors. Both pathophysiological mechanisms and long-term evolution of survivors still deserve further investigations.

References:-

1. Giustino G, Croft LB, Oates CP, et al. Takotsubo Cardiomyopathy in COVID-19. J Am Coll Cardiol 2020; 76:628.
2. Tsao CW, Strom JB, Chang JD, Manning WJ. COVID-19-Associated Stress (Takotsubo) Cardiomyopathy. Circ Cardiovasc Imaging 2020; 13:e011222.
3. Fox SE, Lameira FS, Rinker EB, Vander Heide RS. Cardiac Endotheliitis and Multisystem Inflammatory Syndrome After COVID-19. Ann Intern Med 2020; 173:1025.
4. Varga Z, Flammer AJ, Steiger P, et al. Endothelial cell infection and endotheliitis in COVID-19. Lancet 2020; 395:1417.
5. Creel-Bulos C, Hockstein M, Amin N, et al. Acute Cor Pulmonale in Critically Ill Patients with Covid-19. N Engl J Med 2020; 382:e70.
6. Wichmann D, Sperhake JP, Lütgehetmann M, et al. Autopsy Findings and Venous Thromboembolism in Patients With COVID-19: A Prospective Cohort Study. Ann Intern Med 2020; 173:268.
7. Danzi GB, Loffi M, Galeazzi G, Gherbesi E. Acute pulmonary embolism and COVID-19 pneumonia: a random association? Eur Heart J 2020; 41:1858.
8. Poissy J, Goutay J, Caplan M, et al. Pulmonary Embolism in Patients With COVID-19: Awareness of an Increased Prevalence. Circulation 2020; 142:184.
9. Klok FA, Kruip MJHA, van der Meer NJM, et al. Incidence of thrombotic complications in critically ill ICU patients with COVID-19. Thromb Res 2020; 191:145.
10. Huette P, Beyls C, Guilbart M, et al. Acute Cor Pulmonale in COVID-19-Related ARDS: Improvement With Almitrine Infusion. JACC Case Rep 2020; 2:1311.