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Myocardial Infarction Without Arterial Obstruction in Patient Post COVID 19 Treatment

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Introduction: Some risks factors associated with COVID 19 infections were established such as coronary artery disease in patients with SARS-CoV-2 infections. The use of highly concentrated medications according to Arndt Shultz Law can induce reduction in the vital energy, leading to stagnation of Blood and propensity to have myocardial infarction even without arterial obstruction.

Purpose: To demonstrate that patient with COVID 19 infection that received highly concentrated medications to treat this infection, have more propensity to develop myocardial infarction some days after the treatment instituted.

Methods: Through one case report of 42 years-old patient with history of acquired COVID 19 on January 2nd 2021, he was admitted in the hospital due to dyspnea symptoms, myalgia, and needs oxygenation. He received the medications, ivermectine, hydroxychloroquine, corticosteroids. After 18 days of the initial manifestations, the patient felt pain in the chest and went to the hospital and they found that his troponin were increasing gradually and after two hours of it increased many times and the physician decided to admit him again in the hospital to make more exams. The patient was submitted to catheterization and the result of this procedure were that all his coronary were in perfect state of health without any obstruction and they treated him as he had myocardial infarction. After three months, the patient went to the author's clinic to evaluate his condition and she did chakras' energy centers measurement.

Results: The results of this condition that revealed that all his chakras' were in the lowest level of energy with exception of the seventh that was normal. The author began his treatment with the use of homeopathic medications according to the theory Constitutional Homeopathy of the Five Elements based on Traditional Chinese Medicine.

Conclusion: The conclusion of this study is that patients when treated with highly concentrated medications to treat SARS-CoV-2 infection can develop myocardial infarction without arterial obstruction due to energy deficiency state that aggravated many times due to the use of highly concentrated medications used to treat this kind of infection nowadays.

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Ventriculomegaly in Costello syndrome

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Costello syndrome is a rare RASopathy resulting from germline mutations of the protooncogene HRAS. Many of these mutations affect SHP2, SOS1, RAS, RAF and MEK proteins. It was discovered by Dr. Jack Costello, a New Zealand paediatrician in 1977. Dr. White says. Costello syndrome is now known to be one of a group of related disorders, caused by abnormal functioning of the Ras-mitogen-activated protein kinase (RAS/MapK) pathway. Ras/MAPK pathway is an essential signaling pathway that controls cell proliferation, differentiation, survival and its dysregulation causes clinically overlapping genetic disorders, called as 'Rasopathies'. In this pathway, Ras, a GTPase, transmits extracellular signaling from receptor tyrosine kinases to two serine/threonine kinases (Raf and MEK) and, finally, to the activation of MAPKs. Costello syndrome is a severe developmental disorder characterised by postnatal growth retardation with delayed skeletal maturation, psychomotor retardation, cutis laxa, and acanthosis nigricans.

Abnormal elastin distribution on tortuous dilated arteries and veins in pulmonary vasculature, causing nonuniform, well thickened, obliterative lesions at arterial branch points leading to early pulmonary hypertensive vascular disease. Structural malformations of the heart present at birth such as valvular pulmonic stenosis and abnormal thickening of the muscular walls of the ventricles (ventriculomegaly). most worrying aspect of the CS phenotype. The features of increased index of suspicion of CS in newborn are fetal atrial tachycardia, increased birth weight and head circumference, neonatal hypoglycemia, severe feeding difficulties and urinalysis for hematuria (embryonal rhabdomyosarcoma) and loose, redundant skin on the hands and feet seen in newborns (key role in clinical suspicion of CS). Ras pathway agents, such as farnesyl transferase inhibitors (tipifarnib and lonafarnib) that prevent posttranslational modification of Ras, are being evaluated and may be of therapeutic use for syndromes in this pathway, especially CS. MEK 162 (Binimetinib), orally available inhibitor of mitogen-activated protein kinase kinase 1 and 2 (MEK1/2), directly target the RAF-MEK-ERK 1/2 cascade and it is the best tool against cardiac hypertrophy. The most established mTOR inhibitors are so-called rapalogs (rapamycin and its analogs) reverse cardiac defects. Simvastatin- interact with Ras isoprenylation, decrease Ras activity and low dose statin with selective inhibition of pathological ERK 1/2 signaling is an exciting possibility in the treatment of CS patients. RAF-1 inhibition by C-type Natriuretic Peptide (CNP) improved bone growth in preclinical animal models.

Gene correction of these germline mutations to restore normal protein functions is anticipated as a new therapeutic option. This can be achieved through disruption of gain-of-function pathogenic mutation, restoration of loss-of-function mutation, addition of a transgene essential for cell function and single nucleotide changes.

Oxidative stress and free radicals determine non-neoplastic clinical features such as elastin anomalies, alteration of skin and appendages, developmental retardation and cardiac defects. PAR therapy (potassium ascorbate with ribose) a reduction in oxidative stress biomarkers in parallel with improvement of clinical features. It combines the antioxidant action of vitamin C with the stabilizing intracellular effects of potassium and causes improvement of skin and appendage lesions, better evolution of psychomotor development, non progression of heart hypertrophy, nor tumor development. It is low cost, no side-effects and orally administered therapy in rasopathies.

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Hypocalcemia-induced Camel-hump T-wave, Tee-Pee sign, and bradycardia in a car-painter of a complexed dilemma; A case report

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Rationale: Electrocardiographic is a fundamental tool for a cardiologist, critical care physician, and emergency medicine specialist. The electrolyte imbalance is a very important entity in clinical medicine management. Camel-hump T-wave and the Tee-Pee sign, recently; Wavy triple and Wavy double signs of hypocalcemia (Yasser's sign) are electrocardiographic findings linked to electrolyte deficiencies.

Patient concerns: A middle-aged male car-painter patient presented to the emergency department with atypical severe twisting chest pain, hypocalcemia, hypokalemia, and hypernatremia.

Diagnosis: Hypocalcemia-induced Camel-hump T-wave, Tee Pee sign, Wavy double sign of hypocalcemia (Yasser's sign), and bradycardia in a car-painter.

Interventions: Electrocardiography, arterial blood gases, oxygenation, and echocardiography. Lessons: The dramatic reversal of Camel-hump T-Wave, Tee-Pee sign, Wavy double sign of hypocalcemia (Yasser's sign) after calcium gluconate injection interpret that these signs were due to hypocalcemia. The twisting chest pain and its limited disappearance immediately after calcium gluconate injection indicate the pain can be named as "chest tetany". Non-atropine bradycardia response is evidence that the management of the cause of bradycardia sometimes is essential e.g. hypocalcemia in the current case.

Outcomes: There was a dramatic response of both clinical and electrocardiography including Camel-hump T-wave, Tee Pee sign, the wavy double sign of hypocalcemia, and bradycardia.

Keywords: Hypocalcemia, Camel-hump T-Wave, Tee Pee sign, Bradycardia, Car-painter, A complexed dilemma

Abbreviations:

ABG: Arterial blood gases

ECG: Electrocardiogram

IHD: Ischemic heart disease

O2: Oxygen

RBBB: Right bundle branch block

VR: Ventricular rate

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