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SARS-COV-2 INFECTION CAUSING HYPOPARATHYROIDISM. A RARE CASE.

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ABSTRACT

Infection related hypoparathyroidism is very rare. This is a case report of a 40year old male who presented with history of cough, fever and shortness of breath since one week. He was diagnosed to be covid-19 positive. He was initially managed with non-invasive ventilation therapy, later in view of hypoxia and increased oxygen requirement he was managed with mechanical ventilator support. He was extubated on 5th day. Two days after extubation he developed altered sensorium and unexplained twitching and weakness of right upper limb. His blood tests during that time revealed low serum calcium and low vitamin D levels; Diagnosed as Hypoparathyroidism secondary to Covid – 19 infection. He was managed with tablet calcium, capsule rocaltrol and injection calcium gluconate. Patient's right upper limb weakness and twitching improved gradually later discharged home on 20th day in hemodynamically stable condition.

Key words: Hypoparathyroidism, Hypocalcaemia, Covid-19, Carpopedal spasm.

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INTRODUCTION

Covid-19 infection mainly causes lung injury. Recent studies have reported wide spread organ involvement such as neurological, gastro intestinal, cardiac, renal, cutaneous and ocular [1, 2]. Parathyroid gland involvement is very rare and one case report has been published worldwide [3]. This case report highlights one such case of Hypoparathyroidism secondary to Covid-19 infection.

CASE REPORT:

Our patient was a 40year old male with a past medical history of diabetes mellitus on regular treatment. He presented with history of cough, fever and shortness of breath since one week at outside hospital, where he was diagnosed to be covid-19 positive with RT-PCR nasal swab for SARS-COV-2. He was initially placed on non-invasive ventilation therapy and he received Remdesivir, anticoagulants, steroids and other supportive treatment. Later in view of hypoxia and increased oxygen requirement he was intubated and shifted to our hospital for further management.

In Emergency room examination, patient sedated and paralysed, temperature 98.4˚F, BP-130/70mmhg, RR-20cycles/minute with an oxygen saturation of 92% with 100% FiO_2. Systemic examination was normal except decreased breath sounds on bilateral lung fields with occasional crepts at basal lung auscultation. Lab investigations were significant for elevated LDH-2372, Serum procalcitonin-1.41 IL-6 -77.92, Serum ferritin -2000, D-dimer -1.93, TLC-28,700, Serum creatinine -2.7, blood urea – 102. Other lab findings included Hb-15.6, platelet -3.12, serum sodium-141, serum potassium-5.5 and liver functions were within normal limits, blood and urine

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cultures showed no growth. Radiology investigations were as follows: CT thorax demonstrated multifocal pneumonia with CT severity score of 15/25.

He was continued with broad spectrum antibiotics, steroids, Remdesivir, anticoagulants and other supportive medications. He clinically improved and was weaned off from ventilator and extubated on 5th day. Two days after extubation he developed altered sensorium for which he was further evaluated with CT brain (plain) which was normal. He further developed unexplained twitching and weakness of right upper limb and his blood workup during that time frame revealed the following: elevated phosphorous level (4.9mg/dl) and low levels of serum calcium levels (4.1mg/dl), ionised calcium (0.6mmol/l), vitamin D level (15ng/ml), serum magnesium (1.3) and PTH (2.9pg/ml).

He was treated with tablet shelcal 1000mg three times a day, capsule rocaltrol 0.25mcg three times a day and 10ampules of injection calcium gluconate 10% in 500ml 5% dextrose infusion at rate of 40ml/hour and titrated accordingly for hyperphosphatemia and hypocalcaemia.

Patient's right upper limb weakness and twitching improved gradually later discharged home on 20th day in hemodynamically stable condition.

Summary of patient labs:

	7th day after hospitalisation	8th day	10th day	12th day	14th day
S.calcium	4.1	4.3	7.9	7.7	8.4
Ionic calcium	0.6	1.4	1	1	1.4
magnesium	1.3	1.5	1.6	1.8	2

DISCUSSION:

Hypoparathyroidism (HPT) is considered as an orphan disease. It is characterized by low or normal parathyroid hormone (PTH) with low calcium and high phosphate levels. Parathyroid gland helps in maintaining calcium homeostasis in the human body. It directly helps in absorption of calcium from kidney and calcium resorption from bone. It indirectly helps in calcium absorption from intestine through D1-25OH.

Anterior neck surgery is most common cause of Hypoparathyroidism which constitutes about 75% of all cases of Hypoparathyroidism [4]. Remaining 25% is due to genetic disorders, radiation to parathyroid gland, metastases, granulomatous disease, hemochromatosis and HIV infection [5]. Transient HPT is seen in post-operative period following anterior neck surgery due to parathyroid gland stunning. Permanent HPT is considered when the HPT persists beyond 6 months following anterior neck surgery. Autoimmune HPT is seen in polyglandular syndrome type-1 or antibodies to CaSR (Calcium sensing receptors). The syndromic cause of HPT are polyglandular auto immune syndrome type-1, Di George syndrome, CHARGE syndrome, Kenny-Caffey type-1, Kenny-Caffey type-2, hereditary deafness and renal dysplasia.

Hypocalcaemia causes neuromuscular irritability leading to perioral numbness, paresthesia and extremity twitching. Trousseau's sign seen as carpopedal spasm following inflation of BP cuff in the arm 20mm above systolic blood pressure for 3 minutes. Chvosteck's sign is the contraction of facial muscle following tapping of facial nerve (2 cm anterior to external auditory meatus). Other clinical features include arrhythmias, laryngospasm, seizures, fatigue, brain fog, anxiety and depression [6, 7]. Chronic hypocalcaemia and hyperphosphatemia causes basal ganglia calcification along with nephrocalcinosis and kidney stones. HPT associated with autoimmune disorder can have vitiligo, nail bed fungal infection and candidiasis of mucosa [3]. If associated with genetic syndrome then these patients can have congenital anomalies, hearing loss or learning difficulties [5]. Our patient did not have any of these features hence genetic and autoimmune disorders were ruled out. Laboratory values will have hypocalcaemia, hyperphosphatemia, low or normal PTH.

The acute hypocalcaemia (serum calcium < 7.5mg/dl) is managed with boluses of 10% calcium gluconate 1-2 ampules followed by continuous infusion 0.5-1.5mg/kg/hour over 8-10 hours [8,9]. In addition, oral calcium and activated Vit-D should be added. Chronic hypocalcaemia managed with dietary calcium supplementation. Calcium supplements – calcium carbonate and calcium citrate. Activated Vit-D (calcitriol or alfa calcidol), magnesium, Vit-D2, Vit-D3 (cholecalciferol) and thiazide diuretics [10]. rhPTH (Teriparatide) is used for treatment of osteoporosis. This case report highlights a rare case of Hypoparathyroidism due to covid-19 infection. Early diagnosis and treatment can reduce mortality.

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DECLARATION OF INTEREST:

None declared.

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