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INSULIN RESISTANCE TO COVID-19 INFECTION: PRESENTATION OF AN ORIGINAL CASE

Diana Ofelia Tutasi Guaquipana	Occupational doctor driving school of the Bolivar drivers' union and progress/Ecuador
Priscilla Elizabeth Machado Unigarro*	Rural doctor at "Centro de Salud Bolívar" /Carchi-Ecuador *Corresponding Author
Jessica Paulina Masapanta Yanchapanta	Rural doctor at "Centro de Salud Tarapoa" /Sucumbíos-Ecuador
Ana Verónica Cruz Andrade	Doctor, coordinator of rotating internship of the medical career of the Higher Polytechnic School of Chimborazo/Ecuador
Luis Gonzalo Chito Ases	Resident doctor, at General Hospital Francisco de Orellana/Ecuador

ABSTRACT

In the city of Wuhan, Hubei province, China, dated December 31, several cases of Atypical Pneumonia of unknown etiology were reported. This was notified to WHO, who subsequently declared it a pandemic due to the increase in cases. The causative agent of the outbreak was identified as a new type of virus called SARS-CoV-2, and its disease COVID-19. When people with type 2 diabetes (DM2) develop a viral infection, it may be more difficult to treat due to fluctuations in blood glucose levels and possibly the presence of complications from diabetes. There seem to be two reasons for this. Firstly, the immune system is compromised, making it difficult to fight the virus and likely leading to a longer recovery period. Second, the virus can thrive in an environment of elevated blood glucose.1,2

We present the case of an octogenarian patient, who has a history of DM2, with good control of it. Go to a compatible clinic for COVID-19. After several extension examinations, lymphopenia, double altered coagulation factors, imaging with various peripheral infiltrates in both lung fields, despite treatment and support measures, the patient died due to various complications.

Objective: To demonstrate how DM2 works in patients infected with COVID-19, rapidly developing various complications and a poor prognosis.

Method: This is a systematic review of insulin resistance and COVID-19 infection, emphasizing its clinical characteristics and short-term complications.

Conclusion: COVID-19 infection is not primarily a metabolic disease, but proper glucose control and lipid levels are key in these patients. This approach is important in addressing the well-established metabolic complications of this comorbidity. Furthermore, effective monitoring of these metabolic parameters would help us prevent and improve the acute effects of this virus, by reducing the local inflammatory response and blocking its entry into cells.

KEYWORDS : COVID-19, Diabetes mellitus type 2, complications.

INTRODUCTION

Coronaviruses are members of the Orthocoronavirinae subfamily within the Coronaviridae family. This subfamily comprises four genera: Alphacoronavirus, Betacoronavirus, Gammacoronavirus and Deltacoronavirus according to their genetic structure. Until the appearance of SARS-CoV-2, six coronaviruses had been described in humans (HCoV-NL63, HCoV-229E, HCoV-OC43, and HKU1) that are responsible for a significant number of mild upper respiratory tract infections, in immunocompetent adults.[3]

The SARS-CoV-2 coronavirus is the seventh isolated and characterized coronavirus capable of causing infections in humans. As with other outbreaks caused by coronaviruses, the most feasible primary source of the SARS-CoV-2 disease is of animal origin.[4]

Bats harbor a great diversity of coronaviruses. For this reason, the currently accepted hypothesis regarding the ancestral origin of SARS-CoV-2 is that a bat virus could have evolved into SARS-CoV-2 through intermediate hosts.[5]

Hyperglycemia and the diagnosis of DM2 are independent predictors of mortality and morbidity in patients with SARS. This finding could be due to the fact that these patients have a state of metabolic inflammation that predisposes them to an increased release of cytokines. For COVID-19, a cytokine storm (i.e., very high levels of inflammatory cytokines) has been implicated with multi-organ failure in patients with severe disease.[6]

Metabolic inflammation will also compromise the immune system, reducing the body's ability to fight infection, impairing the healing process and prolonging recovery.[7]

For all of the above, we present the case of an older adult patient with a history of T2DM, the same one who presented with COVID-19 infection, which led him to different withering scenarios.

METHODOLOGY

This is a systematic review of insulin resistance and COVID-19 infection, emphasizing its clinical characteristics and short-term complications.

The information and images obtained belong to the medical personnel in charge of the case whose reinforcements rest on the statistical package Excel, Word and JPG.

CONCLUSION

COVID-19 infection is not primarily a metabolic disease, but

proper glucose control and lipid levels are key in these patients. This approach is important in addressing the well-established metabolic complications of this comorbidity. Furthermore, effective monitoring of these metabolic parameters would help us prevent and improve the acute effects of this virus, by reducing the local inflammatory response and blocking its entry into cells.

CASE PRESENTATION

This is an 82-year-old male patient, resident in Pichincha, Ecuador, by profession a retired Civil Engineer, with a history of Type 2 Diabetes Mellitus under treatment with NPH insulin 13 years ago with good control. Surgical antecedent appendectomy 4 years ago. Who, after contact with a positive patient for COVID-19, presented an 11-day clinical evolution of cough without expectoration, accompanied by an unquantified thermal rise, myalgia, arthralgia, for which paracetamol was self-medicated for 3 days, without improvement, he went to the hospital of greater complexity.

Upon arrival at the Emergency service, a simple chest tomography is performed (photo 1)

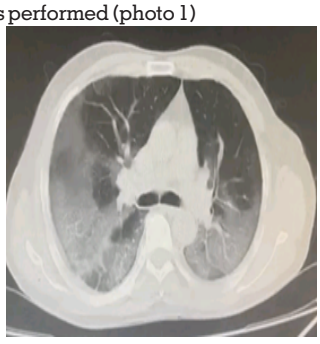

Foto 1. Simple Chest CT: ground glass opacities in both lung fields, predominantly right

In the extension tests, the target was: Leukocytes: 17,000, Neutrophils: 89.5%, Lymphocytes: 780, Glucose 253 mg / dl, Hemoglobin: 12g / dl Hematocrit: 38.5%, Platelets: 335000, D-Dimer: 699, Sodium: 131, Potassium: 4.0, Chlorine: 96, Nasopharyngeal swab: PCR Positive SARS-VOC-2.

Due to nonspecific respiratory symptoms associated with lymphopenia and bilateral interstitial infiltrates, positive SARS-COV-2 PCR diagnosed COVID-19 pneumonia, treatment was prescribed with (ceftriaxone, azithromycin, hydroxychloroquine 5/5), (lopinavir / ritonavir 10/10). In addition to strict glucose control and use of crystalline insulin, to maintain appropriate glucose levels.

During his hospitalization I presented signs of type I respiratory failure, for which a new Simple chest tomography was requested, evidencing a ground glass pattern with a tendency to consolidation (photo 2).

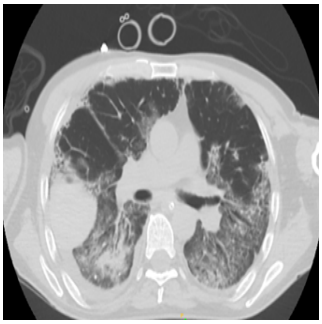

Photo 2: Chest x-ray: Peripheral and subpleural frosted glass pattern, in multiple lobes with bilateral basal predominance with a tendency to basal consolidation plus perihilar and bibasal bronchiectasis

Findings suggestive of late-stage covid-19 plus added pneumonia, together with hyperglycemia (glucose at 170 mg / dl) despite the use of rapid insulin.

Subsequently, the patient presented diaphoresis, tachypnea, with arterial blood gas respiratory acidosis, suffered cardiorespiratory arrest, without favorable response to resuscitation, and died 72 hours after admission.

DISCUSSION

COVID-19 disease was declared a public health emergency of international importance. In Ecuador, the first case was identified on February 29, 2020 and its spread grows rapidly.[8]

People with pre-existing medical conditions, such as diabetes, appear to be more vulnerable to becoming seriously ill with COVID-19. The Centers for Disease Control and Prevention (CDC) report that patients with T2DM and metabolic syndrome may have up to a tenfold increased risk of death when contracting COVID-19. Although DM2 and metabolic syndrome increase the risk of more severe symptoms and mortality in many infectious diseases, there are some additional specific mechanistic aspects of coronavirus infections that require separate consideration, which will have clinical consequences for better patient management. severely affected.[9]

The presence of diabetes mellitus has been described in various studies carried out during the COVID-19 epidemic as one of the most frequent comorbidities present in those patients who developed severe pneumonia or died from the disease. [10]

The reason why diabetes is a risk factor for developing severe COVID-19 disease is not well established, but it is also suggested that ACE2 overexpression in diabetic patients may be involved in the process. These data support the hypothesis that the combination of coronavirus infection and T2DM triggers a dysregulated immune response, resulting in a more prolonged and aggravated lung pathology.[11]

CONCLUSIONS

COVID-19 infection is not fundamentally a metabolic disease, but correct glucose control and lipid levels are key in these patients. This approach is important in addressing the well-established metabolic complications of this comorbidity. Furthermore, effective surveillance of these metabolic parameters would help us prevent and improve the acute effects of this virus, by reducing the local inflammatory response.

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