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CASE REPORT

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Spontaneous Pneumomediastinum in a Patient with COVID-19

Neumomediastino espontáneo en un paciente con COVID-19

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ABSTRACT

Patients infected with SARS-CoV2 show various manifestations consistent with the multiorgan impact of this virus in the system of the human being. However, pulmonary conditions are the most predominant: from slight ground glass infiltrates to severe involvement of pulmonary parenchyma. Pneumomediastinum is a rare expression that only occurs in 1% of patients. We present the case of a critically ill male patient with COVID-19 who develops pneumomediastinum without pneumothorax.

Key words: COVID-19; Pneumomediastinum; Spontaneous pneumomediastinum; Hamman's syndrome

RESUMEN

Los pacientes infectados por SARS-CoV2 presentan manifestaciones variadas consecuentes con el impacto multiorgánico de este virus en la economía del ser humano. Sin embargo, las afecciones pulmonares son las predominantes, dado que abarcan desde sutiles infiltrados en "vidrio esmerilado" hasta un gran compromiso del parénquima pulmonar. El neumomediastino es una expresión rara que se presenta tan solo en un 1% de los pacientes. Presentamos el caso de un paciente varón con COVID-19 crítico que desarrolla neumomediastino sin neumotórax.

Palabras claves: COVID-19, Neumomediastino, Neumomediastino espontáneo, Síndrome de Hamman

INTRODUCTION

The spontaneous pneumomediastinum (SP), also known as "Hamman's syndrome" was first described in 1939. It is defined as the presence of air in the mediastinum generated by alveolar rupture and air exit from the bronchial tree. It may also reach the subcutaneous cellular tissue, the peritoneum or the rachidial canal. The Hamman's sign is the perception of a crackling sound synchronous with the heartbeat in the anterior thorax auscultation. It is a rare disease mostly associated with chronic pulmonary diseases, such as asthma or COPD (chronic obstructive pulmonary disease)[1-7]. One of its multiple triggering factors is the Valsalva maneuver generated by respiratory infectious processes[5-7]. This would justify finding this condition in patients with COVID-19 showing intense, difficult-to-control cough. Other situations that could favor this condition are those related to the use of invasive mechanical ventilation (IMV) and non-invasive mechanical ventilation (NIMV), as for example the high-flow nasal cannula (HFNC), although those situations usually come with a pneumothorax[2, 3, 5].

We present the case of a male patient who developed SP without pneumothorax, in the context of SARS-CoV2 infection and use of HFNC.

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CASE REPORT

59-year-old male patient who had started to have fever up to 39°C six days before consultation. High temperature associated with myalgia was measured and confirmed with a thermometer and was partially lowered with paracetamol. For that reason, and in the context of being close contact of a SARS CoV2 positive patient, a rt-PCR (reverse transcription-polymerase chain reaction) was performed, with positive result. The patient had dyspnea, functional class II-III, 48 hours before hospital admission. He attended the on-call service, where a chest computed tomography (CT) confirmed bilateral ground glass infiltrates, and the physical examination showed desaturation, after which it was decided that he should be hospitalized. It is important to mention essential AHT and obesity degree 1 as relevant patient history. Moreover, the patient had received one dose of the Sputnik V vaccine the day before the onset of symptoms. The physical examination on admission showed the following results: BP (blood pressure): 110/70 mmHg, HR (heart rate): 81 lpm, RR (respiratory rate): 18 rpm, $SatO_2$ 96%-97% with nasal cannula at 4 L/min, T°: 36.9 °C, BMI (body mass index): 35 kg/m^2. The respiratory assessment confirmed good ventilatory mechanics, generalized hypoventilation associated with isolated bilateral crepitant rales. As for the cardiovascular system: S1 and S2 normal heart sounds with clear silent phases, with no signs of pump failure. Supplementary tests: lymphocytopenia, thrombocytopenia, increase in LDH (lactate dehydrogenase), ferritin, PCR and IL-6. The patient also showed acute renal failure. Arterial blood gas: pH 7.43, pCO_2: 31.6 mmHg, pO_2: 66.9 mmHg, HCO_3: 20.8 mmol/L, EB (excess-base): –2.4 mmol/L, SO_2 (AA): 93.7% Table 1 shows laboratory tests taken on admission, and their evolution during hospitalization. Chest CT done on admission: ground glass confluent areas and associated areas of consolidation that compromise both pulmonary fields in a diffuse way, attributed to moderate COVID-19 bilateral pneumonia (according to the criteria of the Chest CT Severity Score). Two-dimensional transthoracic Doppler echocardiography: LV (left ventricular) concentric remodeling with left atrial enlargement and mild enlargement of right cavities; unchanged valves; enlargement of aortic root; preserved global motility; LVEF (left ventricular ejection fraction): 70%; PSP (pulmonary systolic pressure): 44 mmHg; normal diastolic relaxation pattern. The patient was categorized on admission as a moderate case of COVID-19 according to the established criteria of the WHO (World Health Organization), and began treatment with oxygen therapy, dexamethasone in doses of 8 mg/day by endovenous route (which he underwent for 10 days) and pharmacologic thromboprophylaxis. 48 hours after admission, worsening of respiratory failure was confirmed. It was decided that a dose of tocilizumab of 8 mg/kg of weight had to be given to the patient (according to the protocol of the Institution) and he had to use a HFNC. Diphenhydramine and codelase syrup plus inhaled budesonide were indicated because the patient showed persistent dry cough. Due to HFNC weaning failure and progression of respiratory failure, 13 days after admission and 19 days since the onset of symptoms, the patient was transferred to the intensive care unit (ICU). As soon as he was admitted to the ICU, the patient underwent a chest CT angiography that showed signs of pneumomediastinum, and absence of tomographic signs compatible with acute pulmonary thromboembolism (PTE); in the pulmonary parenchyma, patchy and confluent areas of interstitial-alveolar infiltrates with tendency to consolidate, associating overlapping linear images that form a crazy paving pattern compatible with severe tomographic pulmonary involvement (Figure 1). It is worth mentioning that no invasive thorax procedures had been performed before in this patient. The patient stayed 4 days in the ICU; no IMV or vasopressor requirement. Due to the presence of the pneumomediastinum, the patient was evaluated by the chest surgery service, which adopted a watchful waiting approach. The patient returned to clinical medicine, where he weaned from the HFNC after a total of 15 days. 24 days post-hospital admission, the patient was discharged, with indication of home oxygen therapy and outpatient follow-up visits. Follow-up CT done 30 days after discharge confirmed the complete resolution of the pneumomediastinum (Figure 2).

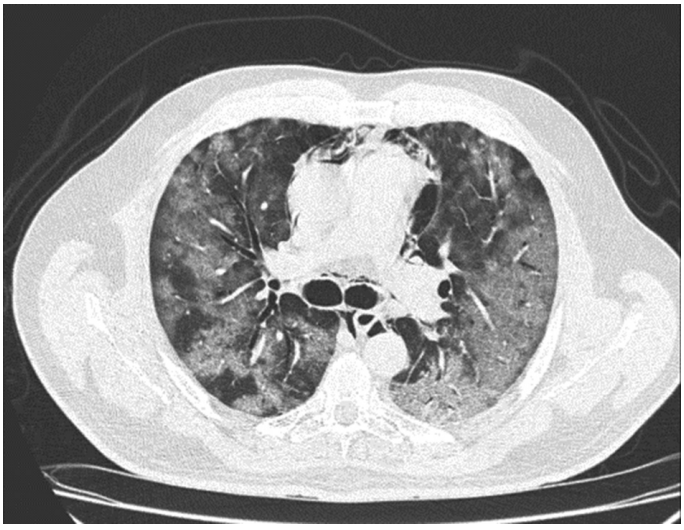

Figure 1. Chest CT angiography

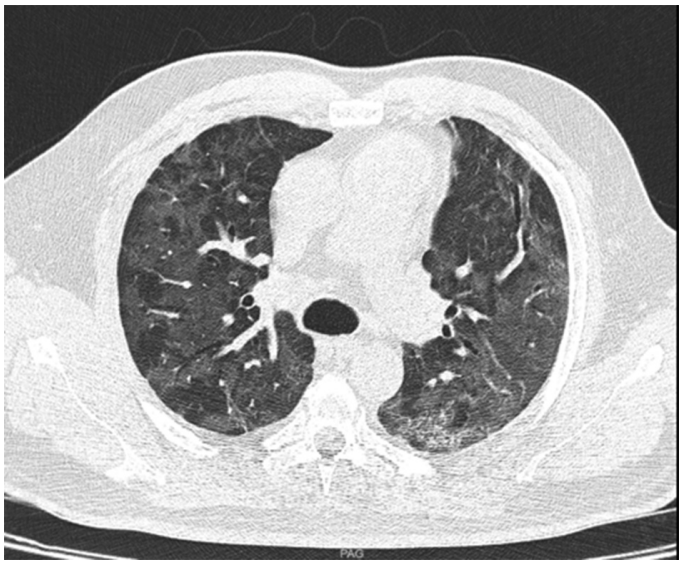

Figure 2. Chest CT after 30 days.

DISCUSSION

The main target of the SARS-CoV2 infection is the respiratory system, and it can affect it in various ways. In hospitalized patients, it is common to observe large involvement of the pulmonary parenchyma by ground glass infiltrates confirmed by CT, but this infection also affects pulmonary vascularization with manifestations such as PTE in a not insignificant percentage[8]. The possibility of developing a pneumothorax is around 1% and may rise to 6% in critical cases[9]. The development of the pneumomediastinum is not very common (1%) and has been observed more frequently in young male adults. In severe pneumonia of viral etiology, the alveolus tends to break due to the condition of the alveolar membrane caused by the infectious involvement of type I and II pneumocytes[7]. The factors that could trigger the SP could be due to Valsalva maneuvers produced by excessive coughing in salvos generating alveolar damage and air leak (Macklin effect)[1, 5-9].

The literature also mentions other conditioning factors associated with the development of nonspontaneous pneumomediastinum, such as the use of IMV or HFNC[1-4, 6-10].

In the case of the HFNC, there are some studies about pneumomediastinum, but it is generally accompanied by pneumothorax[2, 3, 9]. In our patient, there wasn't a SP-associated pneumothorax, so we guided our etiologic suspicion towards the Valsalva maneuvers produced by uncontrolled coughing.

The SP that occurs in patients with a SARS-CoV2 infection usually shares some clinical characteristics according to scientific reports, as for example suffering a severe or critical disease with large involvement of pulmonary parenchyma and the presence of cough as leading symptom[2, 7, 9].

Once the SP is established, the most common clinical manifestations are usually intense thoracic pain and dyspnea. The physical examination confirms a crackling sound in the subcutaneous cellular tissue when emphysema is added[3-5, 7-9]. This wasn't the case of our patient, in whom it was incidentally detected through a chest CT angiography, encouraged by his difficulty in weaning from the HFNC, which has been described in other clinical cases[5].

As supplementary methods, chest X-ray is the most available worldwide, but the SP may go unnoticed if lateral view is not requested. This method is difficult in patients with IMV, due to the technique itself and the need to transfer the patient to other hospital areas. Currently, the CT has become relevant and is the most reliable study for diagnostic confirmation[7].

It is very important to suggest differential diagnoses, such as the pneumothorax, acute myocardial infarction, PTE, neuromuscular diseases or Boerhaave syndrome (spontaneous esophageal

perforation), and those should be considered as possible complications in SARS CoV2 infection[4, 6, 8].

With regard to the prognosis, it is a low-mortality disease, except for the cases where it is associated with pneumothorax, a situation in which it rises to 33%. There aren't any confirmatory studies, but this condition could be associated with a higher mortality rate in patients with severe COVID-19, thus the existence of this condition must warn the physicians about the potential severity of the symptoms.[1, 6, 8, 9] The common treatment of choice is observation and follow-up of the patient, pain control and oxygen therapy, not requiring surgical intervention in most reported cases[1, 4, 6-8].

There is limited literature on this condition. Most publications are clinical cases or case reports, so we think it is of fundamental importance to add suitable studies to determine associated factors and prognosis[1, 2, 6, 7, 9, 10].

CONCLUSION

In short, the SP is a low-frequency entity in SARS-CoV2 infection, with benign behavior in most cases; nevertheless, it should be discarded in patients with progression of hypoxemia or refractory hypoxemia. High clinical suspicion related to imaging confirmation will allow us to choose the correct management of the disease. Improving its diagnosis will let us know its real incidence and optimize treatments.

Conflict of interest

The authors declare that there is no conflict of interest.

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ANNEX

TABLE 1. Laboratory test during hospitalization

Group	Admission	2nd day of hospitalization	Admission to the ICU	Hospital discharge
HCT (hematocrit) (%)	44	43	42	38
Hb (hemoglobin) (g/dL)	15.2	14.8	14.4	12.7
Leukocytes (/mm^3)	4390	9790	10400	4390
Lymphocytes	970	1080	820	1270
Neutrophils	3120	8320	8460	2020
Platelets (x 103/uL)	131	310	350	239
Uraemia (mg/dL)	44	70	39	40
Creatinine (mg/dL)	1.46	1.04	0.9	1.1
Sodium (mEq/L)	135	136	140	139
Potassium (mEq/L)	3.9	4	3.2	3.8
GOT (glutamic-oxaloacetic transaminase) (U/L)	42	89	54	30
GPT (glutamic-pyruvic transaminase) (U/L)	33	121	79	87
LDH (lactate dehydrogenase) (UI/L)	328	502	571	202
PCR (mg/L)	80.24	41.01	27.9	–
IL6 (pg/mL)	–	67.2	419.2	–
D-dimer (ug/mL)	0.35	–	0.74	–
Ferritin (ng/mL)	2983	6533	3560	–
Procalcitonin (ng/mL)	–	0.22	–	–
pH	7.43	7.41	7.42	7.39
pO_2	66.9	69.1	51.8	51
pCO_2	31.6	34.8	32.4	41.6
HCO_{3-}	20.8	21.7	18.5	24.7
Sat O_2	93.7	93.8	86.6	85
FIO_2	0.21	0.35	0.7	0.21
PAO2/FIO_2	320	197	72	242