



Case Report

The impact of mental health on COVID 19 disease progression: Case report

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ABSTRACT

It has been observed that mental disorder is associated with an aggravation of COVID 19 disease.

A 44-year-old male patient, with no medical history, admitted to the emergency room for dyspnea, the exploration revealed SARS-COV-2 pneumonia.

The patient was stable until he was aware of the death of his sister by COVID 19, he was admitted into the intensive care unit 24hours later in a serious condition after worsening of the inflammatory balance and pulmonary lesions.

COVID 19 requires appropriate mental health management to help improve the prognosis of this disease.

1. Introduction

Mental health can affect the evolution of COVID 19 disease.

In this paper, we represent a case of a 44-year-old male patient with COVID-19 aggravated by grief and stress.

The importance of the case: COVID 19 can be aggravated by mental disorders, especially stress and depression, which requires appropriate mental health management to help improve the prognosis of this disease.

2. Case report

A 44-year-old male patient, without medical history, presented to the emergency room for effort dyspnea.

The history of the disease revealed a febrile cough evolving for 10 days.

On clinical examination, the patient was conscious with a GCS of 15/15, blood pressure of 135/75mmhg, heart rate of 80 beats/m, respiratory rate of 20 breath/m, pulsed oxygen saturation of 93% on ambient air at rest, the temperature of 37.2 °C, and the rest of the examination was normal.

The biological assessment showed white blood cells at 4630/ μ l

(normal between 4000–10.000/ μ l), C-reactive protein at 48 mg/l (normal between 0.00 and 5.00 mg/l), IL-6 at 36 pg/ml (normal less than 7 pg/ml), procalcitonin at 0,05 ng/ml(normal between 0.05 and 0.2 ng/ml) ferritin at 246 μ g/l (normal for adults 20–200 μ g/l), fibrinogen level at 3.3 g/l, the rest without any particularities.

Thoracic CT scan performed was in favor of sars-cov-2 pneumonia with 40% lung damage (see Fig. 1).

PCR test for SARS-COV-2 performed came back positive.

The patient has been put on: Azithromycin, Vit c, Zinc, Methylprednisolone 32mg/d per os, aspirin 160mg/d, and enoxaparin4000UI/d.

After 3 days of hospitalization in the COVID-19 department, during which the patient was stable, and 24 hours after being aware of the death of his sister by COVID-19, the patient was admitted into the intensive care unit in a serious condition after worsening of the clinical condition of the patient, as well as the inflammatory balance and pulmonary lesions, the patient was put on oxygen up to15 l/min on high concentration mask for a pulsed O2 saturation of 90%.

An injected thoracic CT scan was performed, showing lung damage of more than 75% with no pulmonary embolism (see Fig. 2).

The biological reassessment showed ferritin at 3755 μ g/l (normal for adults 20–200 μ g/l), white blood cells at 16180/ μ l (normal between

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Fig. 1. Cross-section of thoracic CT scan, in favor of a sars-cov-2 pneumonia with a 40% lung damage.



Fig. 2. Cross-section of thoracic CT scan, in favor of sars-cov-2 pneumonia with lung damage more than 75%.

4000–10.000/ μ l), C-reactive protein at 231 mg/l (normal between 0.00 and 5.00 mg/l), IL-6 at 1601 pg/ml (normal less than 7 pg/ml), fibrinogen level at 5.4 g/l, procalcitonin less than 0,05 ng/ml, the rest without particularity.

The patient was put in addition to the initial treatment on Ceftriaxon 2g/d, Ciprofloxacin 400mg/12h the first day then 200mg/12h (Antibiotic coverage for administration of Tocilizumab), Tocilizumab 400mg single dose and injectable dexamethasone 6mg/d.

A psychiatrist's opinion has been sought; the patient was put on an antidepressant, a neuroleptic, and an anxiolytic, with psychotherapy sessions.

The evolution was marked by the clinical improvement of the patient, with progressive weaning of oxygen until complete weaning at the end of one week, and biological with a decrease of Ferritinemia to 625 μ g/l, CRP to 3 mg/l, and GB to 7360/ μ l.

The patient was discharged from the intensive care unit 7 days later. This case report followed care guidelines [1].

3. Discussion

The explanation, in this case, is the aggravation of the inflammatory

process related to mental disorders, in this case, grief and stress.

The link between inflammation and mental health has been supported by many studies that have shown that patients with major depressive disorder have elevated levels of inflammatory markers, including pro-inflammatory cytokines, such as interleukin-1 β , interleukin-6, and TNF- α , as well as inflammatory proteins, such as C-reactive protein (CRP), in blood [2,3] and cerebrospinal fluid [4,5].

In addition, studies have shown that immune dysregulation and inflammation are associated with other psychiatric disorders, including stress [6], schizophrenia [7], and bipolar disorder [5,8].

In a study by QuanQiu Wang and al assessing the impact of a mental disorder-including bipolar disorder, depression, and schizophrenia-on COVID-19 mortality rates by analyzing a national electronic medical record database of 61 million adult patients from 360 hospitals and 317,000 providers in 50 US states, patients with both a mental disorder and COVID-19 infection had a higher death rate 8.5% compared to 4.7% in patients without a mental disorder [9].

The mechanism behind the link between inflammation and mental health is unclear, but probably complex. In this paper, we will discuss the supposed causes of increased inflammation in stress.

It is generally accepted that severe or chronic stress can cause inflammation. This stress-related pro-inflammatory state represents "sterile inflammation," which is inflammation in the absence of pathogenic disease [5].

Sterile inflammation can be triggered by the activation of recognition receptors that sense endogenous ligands called danger (or damage) associated molecular patterns (DAMPs) [5,10,11].

DAMPs are endogenous non-microbial host-derived molecules that are increased in response to physical and psychological stress [5,10,11].

Assuming this, the pro-inflammatory state is considered to occur in the context of heightened stress responses, possibly related to alterations of hypothalamic-pituitary-adrenal (HPA) axis function and autonomic nervous system (ANS) activity [5,12,13].

A putative scenario for how stress-induced responses in these systems lead to increased inflammation is illustrated in the following figure [5] (see Fig. 3):

Stress increases the synthesis and release of corticotropin-releasing hormone (CRH) and arginine vasopressin in the paraventricular nucleus of the hypothalamus. CRH stimulates the autonomic nervous system to produce catecholamines that can induce the production of proinflammatory cytokines, such as IL-1 and IL-6, via nuclear factor- κ B (NF- κ B)-dependent and other mechanisms (although the interaction between the autonomic nervous system and the immune system may be much more complex) [5,14,15].

These cytokines, in turn, stimulate CRH secretion from the hypothalamic paraventricular nucleus [5].

Therefore, COVID 19 requires appropriate mental health management to help improve the prognosis of this disease.

4. Conclusion

COVID 19 can be aggravated by mental disorders, especially stress and depression, requiring appropriate management.

Clinicians need to be aware of this impact to help improve the prognosis of the disease by taking care of mental health.

Ethical approval

The ethical committee approval was not required give the article type (case report). However, the written consent to publish the clinical data of the patients was given and is available to check by the handling editor if needed.

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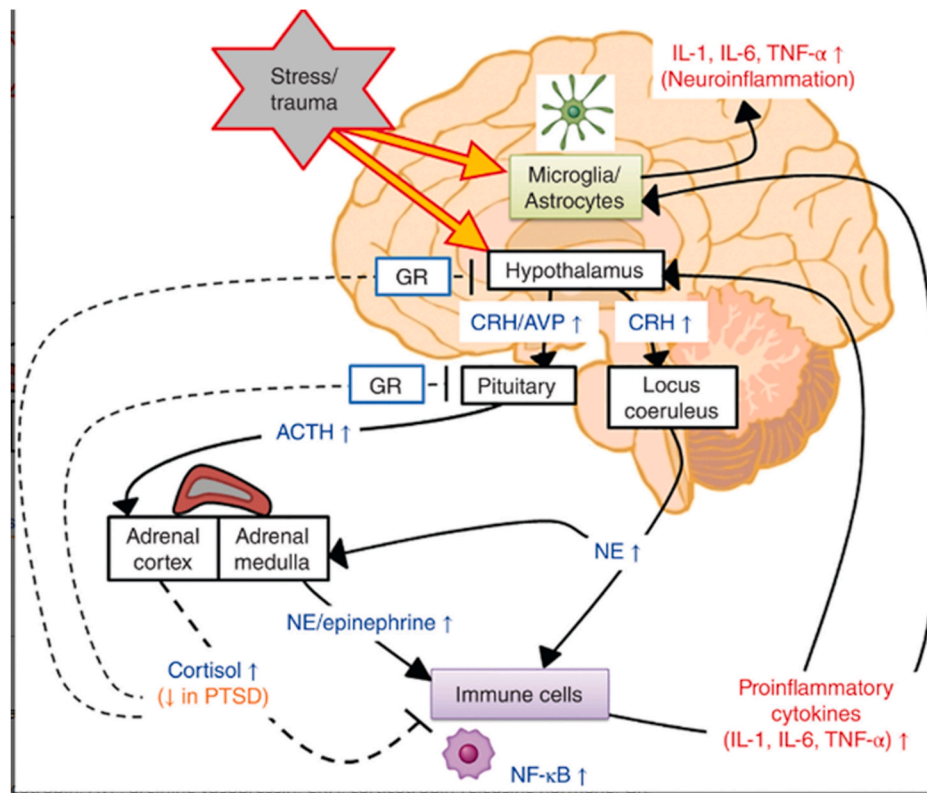


Fig. 3. Mechanism behind the link between inflammation and mental health [5].

Author contribution

BOUABDALLAOUI Amine: Study concept, Data collection, Data analysis, Writing the paper. TAOUIHAR Salma: Study concept, Data collection, Data analysis, Writing the paper. EL AIDOUNI Ghizlane: Study concept, Data analysis, Writing the paper. AABDI Mohammed: Contributor. ALKOUH Rajae: Contributor. MERBOUH Manal: Contributor. Houssam Bkiyar: Supervision and data validation. Brahim Housni: Supervision and data validation.

Trial registry number

This is not an original research project involving human participants in an interventional or an observational study but a case report. This registration was not required.

Guarantor

BOUABDALLAOUI Amine.
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Consent

Obtained.

Declaration of competing interest

None.

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