Introduction

Patients with neurological and psychological conditions have sharply increased in number in recent years. However, due to varied and ambiguous pathogenic pathways, effective therapies for many illnesses and disorders are scarce. Therefore, it is vitally necessary to do additional research into the molecular elements of the illness and to identify fresh targets for the creation of new therapeutic approaches. Systems-level research has suggested that the brain-gut axis and intestinal microbiota may play a role in the development and control of neurological and psychiatric illnesses. It is vital to remember that the host contains some significant sensory and regulatory cells even though the intestinal microbiota is essential for preserving host physiology. The primary controllers of the communication along the brain-gut-microbiota axis are intestinal epithelial enteroendocrine cells (EECs), which are found throughout the epithelium of the whole intestine [1-3]. Over the past few years, the severe coronavirus disease 2019 (COVID-19) outbreak caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) has had a significant impact on healthcare systems. The emergence or persistence of symptoms following the acute phase of SARS-CoV-2 infection, a syndrome dubbed extended COVID or post-COVID-19, is now presenting healthcare professionals with another connected dilemma [4-5]. Multiple systems, including the cardiovascular, neurological, respiratory, and musculoskeletal, are affected by more than 100 symptoms [6]. In fact, multiple meta-analyses have found that up to 50% of COVID-19 survivors have a wide range of persistent symptoms that appear weeks or months or even a year after infection.

Nociceptive pain is described as discomfort brought on by the activation of primary afferent neurons’ peripheral receptive terminals in response to unpleasant mechanical, chemical, or thermal stimuli. When a pain response is proportional to the nociceptive input, it is possible to refer to it clinically as nociceptive pain. According to current beliefs, SARS-CoV-2 cytokine and interleukin storms may cause pain pathways to become more sensitive. As a result, patients with post-COVID pain may display nociceptive pain characteristics [6].

Discussion

This section explains how to distinguish between nociplastic pain and the nociceptive, neuropathic, or mixed phenotypes by using the IASP criteria and clinical reasoning process in people with post-COVID pain. It may be most beneficial to first establish whether nociceptive pain is the primary pain type because one patient can meet the criteria for multiple pain phenotypes. The distinction between neuropathic and nociceptive pain can then be made using additional criteria if a nociceptive pattern is disregarded.

Conclusion

Due to a lack of awareness of the problem, post-COVID pain is still underdiagnosed and possibly undertreated. The research that is now available indicates that some of these people may have nociplastic discomfort. By using the 2021 IASP clinical criteria and grading system to identify distinct pain phenotypes, the worldwide shift toward precision medicine can be used to post-COVID pain to aid in the most efficient treatment planning. The following four factors make it crucial for clinicians to be able to categorise patients with post-COVID pain as having nociceptive, neuropathic, nociplastic, or mixed type: in order to select the most appropriate therapeutic strategies, clinicians must first classify the different forms of pain.

Conflict of Interest

None.

References


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