Clin Schizophr Relat Psychoses
Volume 15:S5, 2021
Doi: 10.3371/CSRP.FH.010322

Clinical Schizophrenia & Related Psychoses

Case Study

Hybrid Open Access

Comorbidity of Neurocysticercosis, HIV, Cerebellar Atrophy and SARS-CoV-2: Case Report and Systematic Review

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Abstract

Background: On November 24, 2021, the omicron (B.1.1.529) variant of SARS-CoV-2 was first reported to WHO from this country. Whether the association of Omicron (Om) variant of concern (VoC), neurocysticercosis, and Cerebellar Atrophy (CA) in people living with HIV/AIDS (PLWHA) is actual or fictitious, require to be investigated and what is the most probable pathogenesis of SARS-CoV-2 mutation deserved to be analysed.

Method: We performed a comprehensive search of publications on PLWHA/NCC/CA/COVID-19-Om written in English, Spanish, and Portuguese.

Results: Forty-five cases presented COVID-19 and cerebellar manifestations, mainly cerebellar ataxia with a mean age of 54.3 ± 12.3 years, were identified. No patients presenting an associated HIV/AIDS/NCC/Om VoC were found.

Case presentation: A 32-year-old-male HIV-positive patient on treatment with a past medical history of NCC and CA infected by SARS-CoV-2 Om VoC is reported.

Comments: We considered theca in our patient is secondary to prolonged consumption of antiepileptic medication. The current fourth wave of COVID-19 is heralded by the Om variant, which is spreading faster worldwide, suggesting it has a growth advantage. In our opinion, those fully vaccinated infected by Om, after recovery from their "Flu", will remain super immunized, which will probably be the beginning of the end of the current pandemic.

Conclusion: The role of SARS-CoV-2 speedy the mechanism of T cell exhaustion and its capacity to decrease the production of INFγ, IL-2, and TNFα must not be ignored in future medical research. It is the first report on PLWHANCA, Om reported in the medical literature up to date from our knowledge.

Keywords: COVID-19 • HIV • AIDS • Comprehensive review • People living with Human Immunodeficiency Virus • NEURO-COVID-19 • Cerebellar atrophy

Abbreviations: CAV: Coxsackievirus, Rubeola, Varicella; TI/T: Traumatic Insults and Toxins; A/I: Anoxic Brain Injury; M/X-L: Mitochondrial and X-Linked Diseases; CVH: Congenital Vermian Hypoplasia; DWM: Dandy-Walker Malformation; ET: Elevation of Torcula; JSRD: Joubert-Syndrome-related Disorders; IH/AV: Inherited Hypoplasia or Aplasia of The Vermis; MTB: Molar Tooth Brainstem; B/US4V: Batwing or Umbrella-Shaped Fourth Ventricle; PCH: Pontocerebellar Hypoplasia; ARD: Autosomal Recessive Disorder Present at or Before Birth; M: Microcephaly; ISCA: Inherited Spinocerebellar Ataxia, NI-TSE S: Non-Inherited Transmissible Spongiform Encephalopathies; PND: Paraneoplastic Disorders; ND: Nutritional Deficiency.

Introduction

At the time of writing (on December 17, 2021), more than 271,963,258 million confirmed cases of COVID-19 have been reported, with a global death toll approaching 5,331,019 million and a total of 8,337,664,456 vaccine doses have been administered being the United Arab Emirates the country with more people fully vaccinated (99%) [1].

As a matter of excellent transparency, on November 24, 2021, the B.1.1.529 variant of SARS-CoV-2 was first reported to WHO, from this country, as soon as the first patient infected by Omicron (Om) was documented in the Tshwane District, one of the disadvantage is regions of Gauteng Province where Pretoria and Johannesburg are the main cities coinciding with the sharp rise in new infections, heralding the beginning of the fourth wave in South Africa. Three previous distinct peaks of COVID-19 characterize the epidemiological situation in this country; the last one was predominantly by Delta variant like all over the world. Now, this region is the global epicentre of the SARS-CoV-2 Omicron Outbreak, with several cases rising exponentially every week, reaching just over 8569 cases in Epi Week 47 from twenty-one of November to twenty-seven of November and more the 41-921 cases by December 03, 2021, and it has continued increasing exponentially till reach 9929 new cases five days later. Unfortunately, the clinical profile of this presentation was not identified with accurate precision. However, it was identified that most of the admitted patients in the COVID ward were non-oxygen-dependent, nobody complaint about respiratory symptomatology, and SARS-CoV-2 Om confirmation was an incidental finding among patients presenting other surgical, medical, or gynae-obstetric conditions.

On the other hand, only nine patients developed COVID pneumonia, and eight were unvaccinated. Only two patients required ICU attention, and the mortality rate was 6.6% [2]. The Om variant has 36 mutations on spike protein, and some are concerning. The first observation made and delivered information suggests a remarkable risk of reinfection with this variant compared to other Variants Of Concerns (VoCs). Today, December 17, 2021, the number of Omicron cases is increasing countrywide at faster rates than previous surges in infection in the previous wave, but nobody has died up to date. Fortunately, the current SARS-CoV-2 PCR diagnostics test continue to detect this Om variant.

Multiple Myeloma (MM) is classified as a bone marrow malignancy characterized by the fast proliferation of plasma cells that produce monoclonal antibodies, combining the high transmissibility of SARS-CoV-2 in immunocompromised cases, results in a high risk for hospitalization and death [3]. Recently, the first case presenting comorbidity of MM and a heavily mutated variant of SARS-CoV-2 infection ten days after vaccination with mRNA-1273 who developed protective anti-spike antibodies has been reported. The authors found an extensively mutated variant with ten spike protein mutations, including E484Q and N440K and concluded that patients

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Received date: 13 December, 2021; **Accepted date:** 27 December, 2021; **Published date:** 03 January, 2022

able to develop detectable antibody responses to vaccination may have increased risk for breakthrough infections secondary to a rapid decline in antibody levels and suggested a higher level of anti-spike antibodies to avoid a poor outcome in COVID-19 patients in case of viral variants with immune escape mutations such as N440K such as SARS-CoV-2 Om variant. The reported case met the criteria for severe COVID-19, but his oxygen saturation of 92%, hospitalization was not required, and he returned home. Symptomatology persisted for another ten days before recovering fully during the next month. His outcome was much better than his unvaccinated contact, but that event confirmed the limitation of COVID-19 vaccination in patients presenting MM and the benign course of infections caused by heavily mutated variants [4]. Other authors confirmed that N440K is present in the SARS-CoV-2 Om variant and it has been associated with breakthrough infections *in vivo* [5-7].

Sheng-Han Kuo documented that cerebellar ataxia can be a clinical presentation of COVID-19 [8]. On October 15, 2020, the first case presenting a cognitive cerebellar syndrome with vasculopathy, and stroke was reported to the medical literature by Chia and collaborators [9]. At the same time, other authors reported a case of a 30-year-old male patient presenting cerebellar manifestations diagnosed as COVID-19 cerebellitis [10]. It has been well documented those patients presenting cerebellar ataxia are more prompt to poor outcomes if SARS-CoV-2 infects them due to immunosuppressive therapies [11]. Colac and collaborators reported a 44-year-old male presenting bilateral interstitial pneumonia followed by acute necrotizing encephalopathy related to the COVID-19 with extensive cerebellum damage. These investigators concluded that acute necrotizing encephalopathy with significant involvement of the whole cerebellum might be a neurological consequence of COVID-19 [12]. Acute postinfectious cerebellar ataxia post-COVID-19 recovery characterized by mild horizontal vestibular nystagmus, dysarthria, bilateral dysmetria, dysdiadochokinesia, and impaired tandem gait in a child of 13-year-old has been reported as well [13]. Chan et al. reported fifty-two cases with myoclonus or ataxia infected by SARS-CoV-2, concluding that ataxia and myoclonus are very uncommon, treatable para-infectious postinfectious diseases, and immune-mediated disorder associated with COVID-19 [14].

Last year, Fadakar et al. reported the first case of acute cerebellitis and COVID-19 [15]. Other authors reported a child presenting acute fulminant cerebellitis induced by SARS-CoV-2 [16]. Recently, another author delivered a case report on a 63-year-old Caucasian man presenting cerebellar signs like broad bases gait and truncal ataxia. Imagenology confirmed white matter degeneration and oedema of the cerebellar hemispheres and COVID-19 with complete regression after corticosteroids and intravenous remdesivir therapy [17]. However, we have no idea if SARS-CoV-2 can damage the cerebellum remarkable enough to lead to permanent Cerebellar Atrophy (CA). Neurocysticercosis (NCC) parasitic is a zoonotic disorder of the central nervous system secondary to infection by the cysticercus' larval stage (Cysticercus cellulose) of the pig tapeworm *Taenia solium* (Ts), common helminth to cause infection in almost all organs in human beings. The occurrence of acquired Ep or intracranial hypertension in a person living in or visiting a region where Ts is endemic or even in close with persons who are portable of taeniosis suggest a diagnosis of NCC if the cysticercosis is localized in the cerebral parenchymal, brainstem, cerebellum, spinal cord, or optic nerves. This parasite is transmitted among humans and between humans and pigs. Taeniosis is acquired only by humans after eating raw or undercooked pork meat contaminated with cysticerci, the parasite's larval form. The cysticerci migrate to the human intestine when ingested, becoming a mature parasite (Taeniosis). These adult worms shed eggs through human faeces that can infect other humans and pigs through direct ingestion or indirect water and food contamination. In developing countries, pigs are often allowed to roam freely, and they can eat human faeces containing eggs or proglottids of Ts. Ingested eggs result in larvae migrating to different parts of the pig or the developing human cysticercosis. A leading site of migration in humans is the CNS. Then, NCC occurs when the cysts develop in the brain, spinal cord, or optic nerve. ES is the most common clinical manifestation of NCC, affecting 66% to 90% of cases, as reported many times [18-27].

This zoonotic parasitic disease may remain asymptomatic for months or even years, and the diagnosis can be confirmed by imagenology. Symptoms and signs are related to the parasite, showing different biological behaviours from one country to another and the inflammatory-immunological response of the host [20,21]. Nonetheless, most NCC cases respond very well to antiepileptic and antiparasitic therapy [28]. The comorbidity of NCC/COVID-19 is not frequently seen, and only three patients have been reported to the medical literature recently [29,30]. Based on the previous background, we are formulating two questions: How often is the comorbidity of NCC/HIV/AIDS/CA/Om? and what neurological manifestations can be observed? Therefore, the primary aid of this review is to answer those questions.

Materials and Methods

A comprehensive search of publications on HIV/AIDS/NCC/CA/COVID-19-Om written in English, Spanish, and Portuguese was performed to answer the first research question.

Literature search strategy

We included case reports, case series, observational cohort studies, systematic review and meta-analysis, cross-sectional studies, and clinical trials on the comorbidity of NCC/HIV/AIDS/COVID-19. During the initial search, we looked for all articles published between December 01, 2019, and December 01, 2021. We search the following databases: Medline, Scopus online databases, Google Scholar, Science Direct, Scielo, Search of Sciences, BioRxiv, Medrxiv and Cochrane library. All Studies were retrieved by utilizing Medical Subject Headings (MeSH).

All items are about "COVID-19/Omicron" OR "SARS-CoV-2 Omicron variant" OR COVID-19 cerebellitis, COVID-10 cerebellar syndrome, OR COVID-19 CA, NCC/CA. OR COVID-19/HIV/AIDS/cerebellar diseases. Alternatively, SARS-CoV-2/NCC/HIV/CA is the PubMed Central wild card for every possible word beginning or ending. We did not include other clinical manifestations beyond the current work scope.

Study and cohort selection

We select prospectively and retrospective cohort studies, case reports, case series, case-control studies, controlled clinical trials, review, and meta-analysis reporting data on COVID-19/HIV/AIDS/NCC/CA.

Study selection: This study aims at the prevalence of PLWHANCA infected by Om and its mortality rate. A total of 225 manuscripts were retrieved from electronic databases up to December 01, 2021. After removing irrelevancy and duplicates, 79 manuscripts were taken for full-text screening, and finally, 44 publications delivering outcomes of interest were included for review [8-17,31-48]. Of these included studies, 2 were peer-reviewed [14,15]. A PRISMA flow chart for the literature searched is shown below Figure 1.

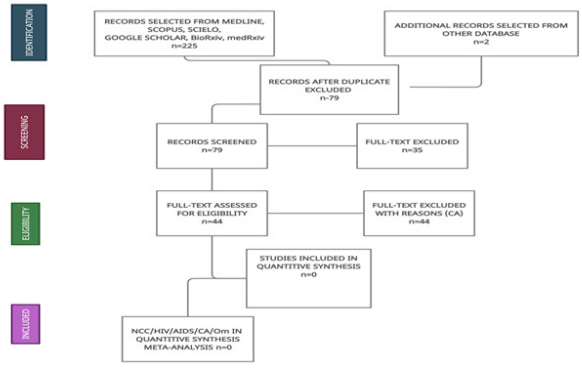

Figure 1. Prisma flow diagram of the included cases.

Results

Forty-five cases presented COVID-19, and cerebellar manifestations, mainly cerebellar ataxia [8-17,31-48] included our patient with a mean age of 54.3 ± 12.3 (SD) years and a median age of 56.2 years (IQR 44.34–73.15). Three cases were free of COVID-19 and presented ataxia and other neurological symptoms [31,42,47]. Other cases either developed cerebellar ataxia concurrently with or after COVID-19 presentation. In the 29 cases with a precise time of onset for ataxia, the median latency between COVID-19 symptoms and ataxia was 15 days (IQR 3–17.3), five cases with concurrent ataxia and COVID-19 symptom onset and a maximum latency of 49 days. No patients presenting associated HIV/AIDS/NCC/Om VoC were found.

Case Presentation

A 32-year-old-male patient is with a past medical history of well-controlled generalized epilepsy secondary to intraparenchymal NCC for the past eleven years. The patient was diagnosed as HIV-positive eight years back and received RV therapy, but his compliance has not been good. He presented in December 2021 to Epilepsy/NCC clinic for routine medical follow up with a 3-day history of general malaise, fever, and mild dry cough (day of assessment is considered as day 1 for confidentiality purposes). At outpatient epilepsy, clinic staff recorded an oxygen saturation of 94% on room air. He had a respiratory rate of 30.

On general examination, no gross abnormalities were found, his heart sounds were normal, and lungs were clear to auscultation; the patient was alert, well oriented and all higher cerebral functions were normal; motor and sensory systems were intact, but he was mild ataxic with mild direction-changing nystagmus horizontally, moderate appendicular ataxia, dysdiadochokinesia, dysmetria with finger-nose testing, and was unable to stand unassisted. He had 5/5 muscle power in the upper and lower extremities with no pronator drift. No meningeal signs on examination. Blood investigations results are shown in Table 1.

Table 1. Parameters measured in patient's blood tests.

Variable	Patient value	Normal range
White cell count	7.10 × 109/L	3.9-12,6 × 109/L
Hb	12.4 g/dL	12-15 g/dl
Platelets	359 × 109/L	186-454/L
Sodium	142 mmol/L	136-145 mmol/L
Potassium	4.4 mmol/L	3.5-5.1 mmol/L
Chloride	102 mmol/L	98-105 mmol/L
Urea	6.8 mmol/L	2.1-7.1 mmol/L
Creatinine	84 µmol/L	48-90 µmol/L
Calcium	2.20 mmol/L,	2.15-2.5 mmol/L
Magnesium	0.84 mmol/L,	0.63-1.05 mmol/L
Phosphate	1.40 mmol/L	0.78-1.42 mmol/L
C-reactive protein	3 mg/L	<10 mg/L
Variable	Patient value	Normal range
Erythrocyte sedimentation rate	12 mm/h	0-10 mm/hr
Total protein	74 g/L	60-78 g/L
Total Bilirubin	<7 µmol/L	5-21 µmol/L
Alkaline phosphatase	90 U/L	42-98 U/L
Aspartate transaminase	29 U/L	13-35 U/L
Alanine transaminase	25 U/L	7-35 U/L
Total cholesterol	3.78 mmol/L	<4.5 mmol/L
HbA1C	0.051	<7%
International normalized ratio	1.01	1
D-dimer	0.3 mg/L	0.00-0.25 mg/L
Rheumatoid factor	11 IU/ml	<20 IU/L
Vitamin B12	236 pmol/L	145-569 pmol/L
Thyroid stimulating hormone	0.98 mIU/L	0.27-4.2 Miu/l
Anticardiolipin antibody	negative	
Protein S	60 IU/dL	55-123 IU/dl
Protein C	110 IU/dl	70-130 IU/dL
Angiotensin converting enzyme	37 IU/L	8-53 IU/L
Interleukin-6	It was no performed	
Anti-streptolysin O titre	98 IU/ml	<200 IU/L
Toxoplasmosis Gondi IgG antibody	Positive	
Cytomegalovirus IgG antibody	Positive	
Rubella IgG antibody	Positive	
Rubella IgM antibody	Negative	
Cytomegalovirus IgM antibody	Negative	
C3	1.1g/L (0.9-1.8g/L)	
C4	0.2g/l (0.1-0.4g/L)	
Antinuclear antibody	Negative	Negative

We also investigated serum antinuclear antibodies, antinuclear antibodies three profile, and autoimmune encephalitis panel, which returned negative. Repeated Reverse Transcriptase (RT)-PCR on a nasopharyngeal swab sample on day five confirmed Om variant. His COVID total antibody (Chemiluminescence immunoassay technique) and serum/CSF levels of interleukin-6 could not be quantified due to our hospital's unavailability of corresponding assays.

Cerebrospinal Fluid Examination (CSF) showed cell count 5/mm^3 (lymphocytes count 100%), protein, and glucose was average and negative for Ziehl-Neelsen, India ink, and Gram stain, *Cryptococcal neoformans*), Toxoplasmosis, NCC and neurosyphilis. Panel for meningitis/encephalitis (*Escherichia coli, Haemophilus influenzae, Neisseria meningitides, Listeria monocytogenes, Streptococcus agalactiae, Streptococcus pneumoniae, Cytomegalovirus, Enterovirus, Herpes Simplex Virus 1 and 2, Human Herpes Virus-6, Varicella-Zoster Virus*, was negative. His mobile chest X-ray did not show abnormalities. Infection with SARS-CoV-2 was confirmed; due to pandemic, he was treated as probable COVID-19 ambulatory since the first day. He did not require admission or supplementary oxygen. CT scan of the brain showed calcified NCC and bilateral and symmetrical atrophy of both cerebellar hemispheres, as we can see in Figure 2.

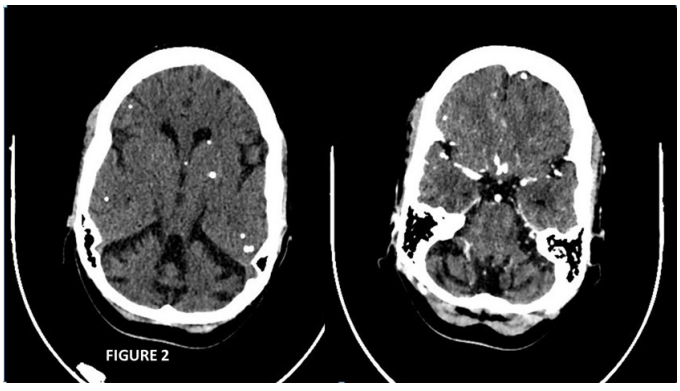

Figure 2. CT Axial view of CT scan of the brain showing multiple calcified NCC and bilateral and asymmetrical atrophy of the cerebellar hemispheres and vermis.

Comments

Due to space limitations, we will not discuss all causes of CA in our patients. Based on our previous experience, we considered the CA present in our case is secondary to chronic epilepsy and prolonged consumption of antiepileptic medication. Other causes of CA are represented in Figure 3.

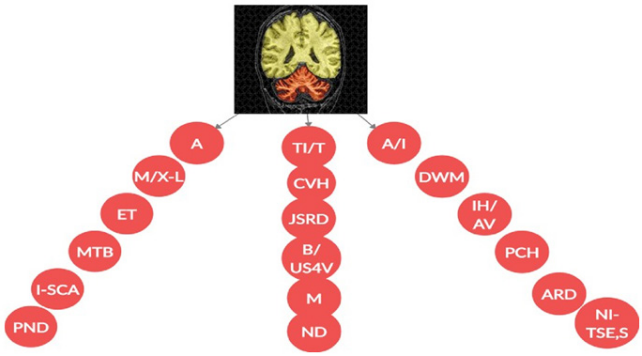

Figure 3. The most prevalent causes of viruses.

Previously, we experienced the source of the SARS-CoV-2 variant in our territory, when the Beta variant (also known as lineage B.1.351) emerged in our province (Eastern Cape) in October 2020 for the first time. This old variant caused more severe illness in PLWHA than non-HIV cases during the second wave in South Africa. However, we recently reported that the clinical manifestations of COVID-19 among PLWHA are like the symptoms and signs seen in non-HIV/COVID-19 cases, if their ARV therapy is not discontinued for more than 2 or 3 months. We also established that the risk of poor outcome and death secondary to COVID-19 in PLWHA with proper ARV therapy is the same with the general population, and other studies should be performed to determine the prevalence and outcome PLWHAC. We also recommended a mandatory plan to vaccinate all PLWHA as a matter of higher priority and not prescribe antiparasitic medication for cysticercosis to any PLWHA at risk to be infected by SARS-CoV-2 and recommended special care to be provided to PLWHAC and other comorbidities like diabetes mellitus, immunocompromised disorders, and lymphopenia. We also hypothesized on the crosstalk of NCC/HIV/AIDS/COVID-19 infections without ARV therapy as a cause of multiple medical consequences and death during the third wave.

The current fourth wave of COVID-19 is heralded by the Om variant, which is spreading faster worldwide, suggesting it has a growth advantage. The Om variant of SARS-CoV-2 has been named a variant of concern (VoC) by WHO based on the evidence that it has more than thirty mutations in the spike protein that impact how it behaves. Fortunately, much research is underway to assess its virulence, risk of reinfection, severity, the outcome of affected cases, and why this fifth VoC has emerged when vaccine immunity is increasing worldwide. While SARS-CoV-2 has more opportunities to spread, it also has more chance to undergo mutations, but it may be beneficial soon according to the hypotheses that we will try to explain below, although some other authors may consider that new variants such as Om are a reminder that the current pandemic is far from over. This variant has some deletions and more than 30 mutations, many of which (e.g., 69–70del, T95I, K417N, T478K, G142D/143–145del, N501Y, N655Y, N679K, and P681H) overlap with those previous variants (alpha, beta, gamma, or delta) VoCs.

Today it is well known that these deletions and mutations lead to raise transmissibility, also causing higher viral binding affinity and increased antibody escape remarkably. Today (December 17, 2021), Om is present in all provinces of South Africa and indeed is going to displace Delta as the dominant variant in South Africa. Recently, another two elements of relevant concern have been raised by other authors: increased number of younger cases and immune escape and the role of monoclonal antibodies. Notwithstanding, lack of secure information about the immune escape mutations occurring in Om and well-designed investigation results impede the reach of convincing scientific explanations to this equation.

We have hypothesized that in a setting of high expression of pro-inflammatory interleukin, chemokines, and other elements secondary to HIV and NCC infectious (graphically represented in Figure 4), the Delta VoC of SARS-CoV-2 has no other choice than change the composition of its spike protein and become in omicron VoC and to continue spreading faster on all over the world as dominant VoC. This mutation may be one of the final ones.

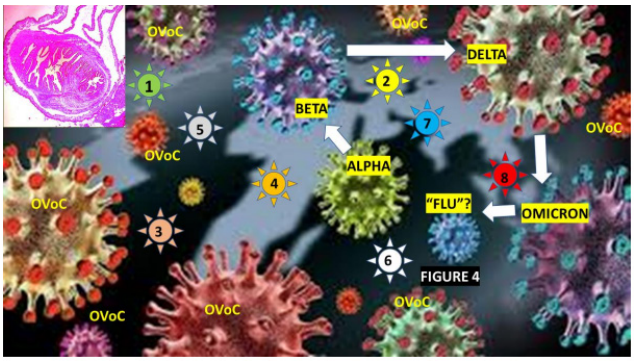

Figure 4. Diagram of the proposed pathway of mutation from alpha VoC to omicron VoC though beta and delta VOCs in a setting of cytokine storm, the presence of HIV and under the influence of colloid stage of NCC.1: Interleukin 1, 2: Necrosing tumour factor-alpha, 3: Interleukin 1 beta, 4: Interferon-Gamma, 5: Interleukin 2, 6: Natural Killer Cell, 7: Cholecystokinin, 8: Interleukin 9: OVoC: Other Variants of Concern.

It is an everyday phenomenon that two SARS-CoV-2 variants surged in Africa? Probably, the answer is not. Figures 3 and 4 show our hypotheses. It has been proved that many zoonotic infections from Africa later spread worldwide. Now, we are affording three of them: NCC (hyenas and African hunting dogs), HIV/AIDS (primates), and SARS-CoV-2 (Bat), leading to alpha VoC. Later in South Africa, beta CoV, SARS-CoV-2 VoC Delta from India where HIV/AIDS/NCC are highly prevalent and back to South Africa again.

We have hypothesized the mechanism of replication of SARS-CoV-2 within a complex immune system affected by other viruses and parasites simultaneously. In this region, the prevalence of cysticercosis and HIV is more elevated than in most countries worldwide, and the prevalence of cysticercosis and HIV is also elevated in India, where the VoC Delta is born, as mentioned before. Therefore, probable, Delta as a dominant VoC in South Africa during its replication process, it should afford the inappropriate immune system due to the presence of HIV and cysticercosis again plus the presence of another epidemic virus (Flu?) and after taking genomic component from that virus favouring the born of Om VoC. If these hypotheses work, Om will be dominant worldwide very soon, causing the only clinical manifestation of "Flu", probably few people will probably pass away like a benign "Flu" pandemic with minor damage to the world economy Figure 5.

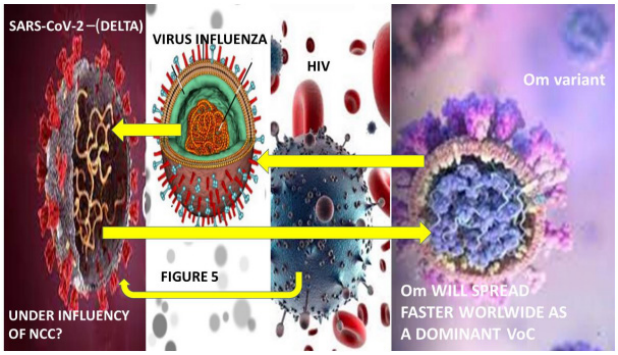

Figure 5. Hypotheses on SARS-CoV-2 delta VoC mutation to SARS-CoV-2 omicron VoC the presence of HIV and influenza after "taking" genomic material from one to another.

In our opinion, those fully vaccinated infected by Om after recovery from their "Flu" are going to remain super immunized, which will probably be the beginning of the end of the current pandemic. Nevertheless, other accurate, confident, and well-designed research will support or reject this hypothesis.

Discussion

After extensive searching of the medical literature, we found no publication on PLWHNACOm up to date. The clinical manifestations of COVID-19 among PLWHA are like the clinical feature seen in non-HIV/COVID-19 cases if their ARV therapy is not discontinued for more than 2 or 3 months. The risk of poor outcome and death secondary to COVID-19 in PLWHA with ARV therapy is like the general population. To determine the prevalence and outcome of PLWHANCAOM is necessary to conduct well-designed statistical research shortly. The comorbidity of NCC/HIV/AIDS has been studied before, and the results were published in the medical literature. Even though SARS-CoV-2 can affect the cerebellum, the atrophy of the cerebellum in our case is not related to COVID-19. Cerebellar atrophy is not commonly seen secondary to NCC [28]. We support the plan to vaccinate all PLWHA as a higher priority. We recommend, we do not prescribe antiparasitic medication for cysticercosis to any PLWHA at risk to be infected by SARS-CoV-2. PLWHAC should follow the international guidelines of social distancing, health education, hygiene, and self-isolation. We have hypothesized on the crosstalk of NCC/HIV/AIDS/COVID-19 infections without ARV therapy as a cause of multiple medical consequences and death. Following the arrows in Figure 2, it is possible to understand the self-explanatory diagram. The role of SARS-CoV-2 speedy the mechanism of T cell exhaustion and its capacity to decrease the production of INFγ, IL-2, and TNFα must not be ignored in future medical research. It is the first report on PLWHANCA Om reported in the medical literature up to date, as far as we know.

Conclusion

Finally, we hypothesized the interactions of NCC, HIV/AIDS, and Omicron-SARS-CoV-2 on patients without ARV therapy, as can be graphically represented. The role of SARS-CoV-2 accelerating the mechanism of T cell exhaustion and its capacity to decrease the production of INFγ, IL-2, and TNFα should not be ignored in future medical research. Of course, more investigation will clarify doubts and establish curative therapy with safe and sustainable accurate prophylaxis.

Ethical Considerations

This review was not considered ethical approval because all the data were extracted from previously published articles.

Ethical approval and consent to participate and publication: This manuscript is based on previously conducted studies and does not include any studies of human participants or animals performed by any authors. Therefore, the Institutional Ethical committee did not consider this study for additional ethical approval.

Competing Interest

The author has not any conflict of interest to disclose. The authors declare that they researched the absence of any commercial or financial relationships construed as a potential conflict of interest.

Funding

Both authors declare that they never received any financial support or personal collaboration that could have influenced the results reported in this paper.

Declaration of Anonymity

The author certifies that he did not reveal the names, surnames, initials, Alternatively, other identity issues of this case in this publication and complete anonymity are guaranteed.

Availability of Data and Material

The data that support the findings of this study are available on reasonable request from the corresponding author.

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How to cite this article: Sibat, Humberto Foyaca. "Comorbidity of Neurocysticercosis, HIV, Cerebellar Atrophy and SARS-CoV-2: Case Report and Systematic Review." *Clin Schizophr Relat Psychoses* 15S (2021). Doi: 10.3371/CSRP.FH.010322