

The Disorder Within Diagnosis

Diagnostic factors for Neurocognitive Disorders

with emphasis on

Vascular and Alzheimer's Dementia,

affected domains,

and cross-culture

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1. Introduction

Neurocognitive Disorders are conditions that exhibit a marked decline in human functioning in areas such as paying attention, carrying out complex tasks, remembering, recalling, language, perception, movement and social ability, and which are brought on by neurological and/or psychological triggers - adapted from Pillay (2015) and Trivedi (2006).

A neurocognitive disorder (NCD) can manifest itself in a person of any age, depending on causal factors, for example, in a child due to a head injury. Much of the incidence of NCDs, however, occurs in persons during the latter part of their lives as they move towards and into old age (Krause, 2016).

At our current time of scientific and medical advancement, a greater number of people are living to an older age, and thus many more people are experiencing the effects of the inevitable slow down of the body's neurological system with its concomitant negative effects and symptoms (O'Hara et al., 2001). Also, these days, there is an increase in people of all ages suffering from NCDs due to the prevalence of violent crimes and vehicle accidents in many parts, like in South Africa, which bring with them potentially debilitating neurocognitive effects (Adnams, 2010). Such increases in the occurrence of neurocognitive malfunction means that the functioning and lives of many more sufferers are at stake, and this in turn affects the associated family members, the community, and society at large, as each of these need to find ways of effective coping and treatment.

Germane to the entire subject of people experiencing NCDs are the issues of prevention and treatment or cure. In order to properly treat such individuals, we must know what we are treating, so that we may apply the best remedies. We must also ensure that we understand each person's cultural background and social setting. Otherwise, we could find ourselves applying a correct solution to the wrong malady, and the patient not being helped or even being harmed as a result. Properly identifying or diagnosing what a person may be suffering from is therefore crucial.

In fact, with the advent of the new editions of the DSM-V and ICD-10

classification systems, diagnosis seems to have become the central topic in the world of psychiatry (Alarcon, 2009). Hence the importance of the subject of this paper.

We will now examine the diagnostic factors for NCDs with emphasis on Vascular and Alzheimer's Dementia, affected domains, and cross-culture.

2. Diagnostic factors for Neurocognitive Disorders (NCDs)

There are divergent opinions as to how to best assess a person for any mental disorder. Two systems of classification of disorders are used at present (Austin, 2015), being the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) (American Psychiatric Association, 2013) and the *International Classification of Diseases* (ICD-10) (World Health Organisation, 1993). The diagnostic criteria for the Neurocognitive Disorders as contained in these two classifications will now be discussed.

DSM-5 deals with Neurocognitive Disorders (NCDs) under the following main divisions: Delirium, Major NCD, and Mild NCD (APA, 2013). Two of the more prevalent subtypes of Major/Mild NCD are Alzheimer's Disease and Vascular Disease. Several other etiological subtypes of interest are Traumatic Brain Injury, Substance/Medication Use, HIV Infection, and Multiple Etiologies.

We will use the following categories for our discussion below: Delirium, Alzheimer's Disease, Vascular Disease, and HIV Infection.

2.1 Delirium

Delirium is an inattentive confusional state of organic origin occurring over a few hours or days and can usually be cured with the correct treatment. Entities causing delirium typically restrict oxygen or other needed substances from the brain (Zieve et al., 2016).

DSM-5 requires diagnostic specification due to substance intoxication/withdrawal, medication, another medical condition or multiple causes (APA, 2013, pp. 596-597). ICD-10 allows for coding of mild/moderate/severe psychoactive substance abuse/dependence/use disorder with intoxication/withdrawal delirium (AAAASF, 2015, pp. 196-214). It presents certain apparently unsystematic discrepancies,

such as alcohol dependence with withdrawal delirium, but opioid, cannabis, cocaine, other stimulant, hallucinogen, inhalant and nicotine dependence are not offered a possible diagnosis with withdrawal delirium. Acute confusional syndrome due to nicotine withdrawal has however been reported in reviewed literature, and nicotine withdrawal induced delirium has mounting support in research findings (Klein, 2002). Although ICD-10's greater specificity appears to be helpful in regard to more accurate diagnosis, it seems that greater consistency in ICD-10 diagnostic coding is needed with regards to delirium.

The term "delirium" is mentioned 250 times over 711 pages of DSM-5 in diagnostic reference to numerous mental and physical disorders (APA, 2013, pp. 93-803). Delirium is referred to 43 times over 1176 pages of the ICD-10 codes (AAAASF, 2015, pp. 194-1369). Even in ICD-10 Online (WHO, 2016), an advanced search on the term "delirium" is required to obtain 28 differentiated results ranging over several codes. Thus if a mental health practitioner wishes to view all instances of "delirium" in the two chief diagnostic tools currently available, she will have to search and consult 1887 pages and an online database physically clicking on 321 links or find-arrows, view each instance in a scattered and haphazard manner, and then she still cannot be sure that she obtained all entries, as any of the search/find criteria she used may or may not have included all occurrences of the term. This is not good for speedy and thorough diagnosis. An exhaustive database which can produce a systematically arranged collation of all entries in DSM-5 and ICD-10 in which each term or keyword appears, needs to be created, so that for every term searched, a continuous text on the subject in logical sequence appears, with its own index. This then would allow the practitioner to review all current official diagnostic information on any specific term in one place. If such a combined mega-database is not currently practicable due, for example, to financial or human resource factors, either such a DSM-5 or ICD-10 database should be undertaken,

after which a similar extrapolation to the other classification system could be performed, and then perhaps a combination of the two thereafter.

In diagnosing delirium, ICD-10 is currently more specific than DSM-5, the former giving specific examples of delirium due to numerous physical conditions under codes F05 and F06, such as brain syndrome, confusional state (nonalcoholic), infective psychosis, organic reaction, and psycho-organic syndrome (AAAASF, 2015, pp. 195), whereas DSM-5 simply states “delirium due to another medical condition” (APA, 2013, p. 597), and makes no mention of these five physiologic conditions which can cause delirium.

In diagnosing delirium according to DSM-5, one is looking to identify a primary disturbance of attention, timing of short duration, a secondary disturbance in cognition, excluding another neurocognitive disorder or etiological evidence of another medical condition or substance. If diagnosis is one of the latter two elements, namely another disorder or condition, then the diagnosis would be that disorder/condition with delirium. Although this would logically seem to be the case, it is not indicated as such in DSM-5, and practitioners should therefore be careful in working exclusively with DSM-5 as it tends not to present a complete diagnostic scenario.

2.2 Alzheimer's Disease (AD)

2.2.1 DSM-5

In a manual as important as DSM-5, it is advisable to rather delineate the applicable diagnostic criteria for AD under item “A”, instead of simply stating “A. The criteria are met for major or mild neurocognitive disorder” (APA, 2013, p. 611). Even a succinct rendition of the applicable criteria would be preferred, such as:

“A. The following criteria for mild or major neurocognitive disorder are met:

1. Evidence of mild or major cognitive decline in one or more cognitive domain (complex attention, executive function, learning and memory, language, perceptual motor, or social cognition) based on the concern of

the individual's concern, an informant, or a clinician, and a mild or major impairment in cognitive performance, documented by standardized neuropsychological testing or another quantified clinical assessment.

2. The cognitive deficits do not interfere with (mild NCD) or do interfere with (major NCD) independence in everyday activities, such as paying bills or managing medications.

3. The cognitive deficits do not occur exclusively in the context of a delirium.”

To have to refer back several pages and mentally sift through duplicative criteria, such as “D”, the condition not being better explained by another physical or physiological disorder, or instructions on behavioural disturbance, and then to bear the specifics of 1, 2 and 3 above in mind whilst reviewing the AD given criteria, is not only unproductive, but could well result in misdiagnosis.

DSM-5's AD criteria go on to state that probable *major* AD is diagnosed if there is evidence of *either* an AD genotype *or* steady decline in memory and learning and at least one other cognitive domain based on history or testing with no mixed etiology; whereas probable *mild* AD is diagnosed if there is evidence of *only* an AD genotype.

Steady decline in memory and learning based on history or testing is not a criterion for probable mild AD. It is submitted that this is an error, either in the thinking of the DSM-5 authors/reviewers or in their logical syntactical representation of the malady.

DSM-5 then goes on to state that possible mild AD is diagnosed if there is no AD genotypical evidence and only steady decline in memory and learning with no history, testing or mixed etiology. This appears to be highly arbitrary, and calls for an exhaustive and thorough review and wording correction of these diagnostic descriptions.

2.2.2 AD genotyping

In addition, the over-reliance on AD genotyping as a make-or-break diagnostic criterion for AD in DSM-5 is at best risky at this point in our knowledge and understanding of the malady due to inconsistent

etiological genetic evidence. Some studies have indicated that genotype APOE ϵ 4 carriers can display better cognitive functioning than that of non-carriers, and that APOE ϵ 4 carriers may only be at risk of developing AD if they also have family history of AD, which implies that there may be other more important etiological factors influencing the occurrence of AD than AD genotyping (Tuminello & Han, 2011). Thus, it is submitted that AD genotyping should be a corroborative, rather than decisive criterion together with the other criteria presented by DSM-5, for probable mild or major NCD due to AD diagnosis.

2.2.3 Semantic dementia criterion

AD pathology has been shown to underlie the diagnosis of semantic dementia (SD) (Chow et al., 2010). This study shows that cases of SD can remain clinically diagnosed as such for the full extent of the sufferer's illness, which ranged in this instance from 10 to 13 years, with AD being the actual pathological diagnosis. The neuropathology confirmed elements characteristic of AD such as atrophy patterns, neurofibrillary tangles, and amyloid plaques, but clinical pointers such as APOE ϵ 4 presence, falling MMSE scores, and family history of late onset dementia seemed not to lead to an AD diagnosis. All subjects with AD showed reduced ability to comprehend and repeat verbal testing material which confirms the emphasis on DSM-5 AD diagnostic element: "Clear evidence of decline in memory and learning" (APA, 2013, p. 611). In as much as 90% of the cases in this study who had AD were only clinically diagnosed as having SD, it is submitted that an inclusion of more language-oriented and behavioural (for example aggression and disinhibition) diagnostic elements should be included in the formal diagnostic factors for AD.

2.2.4 Dangers of the NCD mild category

If a person with a minor onset of declining cognitive ability presents herself for diagnosis and she scores 23 on the MMSE test now, 22 six months later, and 21 after another six months, she would still be in the 19-23 mild cognitive impairment range (Wikipedia, 2016) and might still

retain all normal mental functioning except for the slight short-term memory problem she reported when she first sought help. According to DSM-5 this would have her diagnosed as suffering from possible AD. Whilst in the mild range of anything cognitively declining, it is suggested that to start labelling people as having AD is a very dangerous practice, and that all minor or mild versions of NCDs should not carry potentially discriminatory classifications with possible legal and other personally devastating consequences (Ganguli, 2011). Rather the term “Mild Cognitive Impairment” (MCI) should be used, as DSM-5 itself admits that “mild NCD due to Alzheimer's disease is likely to represent a substantial fraction of mild cognitive impairment (MCI)” (APA, 2013. P. 612). Only when there is evidence of seriously incapacitating cognitive processes should specific diagnoses such as AD be given.

2.2.5 Cultural factors

When a Zulu person in South Africa undergoes *ukuthwasa*, she accepts *indlozi* or *amadlozi* (previously lived spirits) to abide within her (Umsamo Institute, 2011) and to guide her through dreams (*amaphupho*). If her communication of such dreams or events is sufficiently euphoric, she could mistakenly be diagnosed as suffering from bipolar disorder (APA, 2013, p. 128). But the same person with evidence of declining memory, learning, and verbal ability, with neuroimaging showing temporal lobe atrophy (Tang-Wai, 2012), should still be diagnosed as having AD. It would seem that the more cognitively debilitating and physically-founded symptoms appear to be, the greater the chance of accurate diagnosis regardless of cultural differences. Hence the need to emphasise physical (neurological and physiological) aspects of NCD diagnosis where possible when dealing cross-culturally. All aspects of clinical diagnosis must nevertheless be done by culturally-sensitive practitioners with the use of culturally-adapted tests.

2.2.6 Neuropsychological testing for South Africa

A country like South Africa has multiple language and cultural groups with the preponderance of the population being in the lower echelons

socially and educationally. This does not bode well for neuropsychological testing to be using raw tests like the Mini-Mental Status Examination (MMSE).

For example, in the MMSE, number 8, for 1 mark is: ‘Have the patient repeat “No ifs, ands or buts.”’ You could be sure that a person not from an educated English background could simply flounder on this point. This means that the majority of South Africans might fail this question for no other reason than that they simply do not recognise the idiomatic expression. It’s like asking a South African from an exclusively Afrikaans background to repeat the Southern Sotho saying: “Bana ba kwale ba bitsana ka molodi”, which directly translated is “The children of the sandgrouse call each other by a tune”, meaning “Birds of a feather flock together”. But having to articulate unfamiliar phonemic combinations does not necessarily indicate degraded cognitive functioning. Rather let the Englishman have the “Ifs, ands or buts” question and the Sotho-speaker the “Bana ba kwale ba bitsana ka molodi”. That way it would appear to test the same comprehension, memory and articulatory ability in two distinct cultures.

The fact that different cultures expect different levels of functioning from older people (Ineichen, 2000) must also be taken into account in the need for intervention timing, and the content of neurocognitive tests designed for the different cultures and culture-combinations which exist in South Africa.

The introduction to item 16 and the question itself in the Cultural Formulation Interview (CFI) offered by the APA on their website (www.psychiatry.org) reads as follows:

“Sometimes doctors and patients misunderstand each other because they come from different backgrounds or have different expectations.

16. Have you been concerned about this and is there anything that we can do to provide you with the care you need?”

This is one of many examples of ill-worded material in cross-cultural testing/interview material currently available to practitioners. It is

precisely this type of approach that retards progress in the field of effective transcultural diagnosis.

A simplifying paraphrase of the above item could be as follows:

“Sometimes doctors and patients do not understand each other very well because they speak different home languages, have different cultures, or grew up in different areas.

16. Are you worried about me and you, that maybe we do not understand each other correctly?

How would you like me to help you with your problem that you have come with today to us here at the clinic?”

Such wording can obviously be modified and adapted further but it simplifies understanding and comprehension for a transcultural person who, for example, may not have English as her primary language. Words like “expectations”, “concerned”, “provide”, and even “care”, may be intrinsically foreign to a patient who only has a rudimentary knowledge of English, and should therefore be avoided in all recommended testing material as well as actual testing situations. An interviewer has to non-condescendingly but consciously “speak down” her English (i.e. make a concerted effort to choose easy to understand basic words) when communicating with an essentially non-English speaker.

Likewise, wording in informant questionnaires for dementia detection, using phrases like “reduction in activity level” (Lenger, 1996, p. 740) is not only intellectually indefensible, but calls into question the motivation of its authors - is it to impress the psychological fraternity or to genuinely seek the understanding and true response from informants from any conceivable educational or cultural background? Why not just say: “Do you see your family member doing less and less things for himself?” and/or “Do you see him doing not so many things like before at home?”

Further to the overall necessity of clinician sensitivity to cultural-linguistic differences in the assessment setting, she must be trained to always be able to put aside her petty differences and impressions which might have the effect of causing the patient to either

be offended or close up emotionally and thus not be as verbally responsive or emotionally accessible as the testing environment requires for normative responses. Many of us know the feeling of being helped by a less than pleasant or emotionally cold or superior person in any number of settings in the real world, and it is hoped that the calibre of clinician or clinician's assistant who interacts with patients will be of such a nature as to preclude such unacceptable behaviour. Thus, "when the examiner and client have different ethnic backgrounds, the examiner may need to make an extra effort to establish a rapport that will result in the person's best performance" (Kring et al., 2007, p. 98).

2.3 Vascular Disease

2.3.1 DSM-5 diagnostics

There are obvious problems with the manual's instructions for diagnosing vascular neurocognitive disorder (VND).

Firstly, no indicators are given for distinguishing mild from major VND (APA, 2013, p. 621). Secondly, possible VND is diagnosed if a significant cognitive decline in complex attention and frontal-executive function is indicated, without neuroimaging evidence or concomitant cerebrovascular events. This is viewed as insufficient factors for any diagnosis, which after all is by its very nature a composite of several key mutually-supporting elements. Thus possible VND diagnosis should include a connective cerebrovascular event such as stroke or infarct, or neuroimaging evidence. DSM-5 comes around in the section following diagnostic criteria and states: "Etiological certainty requires the demonstration of abnormalities on neuroimaging. The lack of neuroimaging can result in significant diagnostic inaccuracy" (APA, 2013, p. 622). But then why not include this as a must under criteria for possible VND? To exclude it in such a prominent section is to sow the seeds of confusion for the budding diagnostician.

It should also be considered that "possible" diagnoses are by definition not diagnoses at all, and should perhaps not be permitted in a discipline such as psychopathology, which requires accuracy and as much

certainty as possible. Tentative diagnoses also carry with them the possibility of incorrectly predisposing clinicians to possible misdiagnoses, as well as having negative self-image implications for the subject. Instead of possible VND, perhaps vascular hypoperfusion syndrome (Román, 2004) (VHS) or vascular cognitive impairment (VCI) (Kim et al., 2012), akin to MCI, might better describe the evidence of a convincing but not conclusive VND diagnosis.

Under diagnostic features of VND a distinction between mild (one stroke) and major VND (two or more strokes) is given, but tucked away four paragraphs down on the next page is not a good place for this information; it should be made an integral part of the main diagnostic criteria section.

2.3.2 Environmental, and other physiological and genetic factors

DSM-5 states in its VND's Risk and Prognostic Factors section (APA, 2013, p. 623) that the outcomes of VND are affected by physical exercise, education and mental activity. By implication the presence or absence of these entities would relatively influence the etiology of VND and should therefore form a part of the assessment of the lifestyle of the subject in the diagnostic phase, but nowhere in the Manual do these entities feature as possible indicators of a probable lifestyle background for NCD diagnosis.

Likewise, the major risk factors for VND are given as including hypertension, smoking, high cholesterol, obesity, and diabetes, but again none of these appear in the main diagnostic criteria section, making that section of increasing dubious comprehensiveness for use in a thorough delineation of VND diagnostic characteristics.

2.3.3 MRI or CT structural neuroimaging

In the interests of clarity and thoroughness, apparently cursory references to "neuroimaging" in the Diagnostic Criteria for VND in DSM-5 should be adequately clarified by replacing the term with "MRI or CT structural neuroimaging" in all instances. Since this exemplification is

made under “Diagnostic Markers” (APA, 2013, p. 623), it could just as well have been stated under the main criteria section.

2.3.4 Neuropathologic examination and exclusionary testing

Besides cognitive tests like MMSE and MRI or CT structural neuroimaging, there are other crucial neuropathological examinations which could be diagnostically undergone by the patient, such as temporal artery evaluation, cardiac auscultation, fundoscopic evaluation, and neurologic assessment including visual field defects, spasticity, pseudobulbar palsy, and hemiparesis (Chawla, 2016). These available diagnostic examinations should be mentioned in the DSM-5 VND main Diagnostic Criteria section.

Likewise tests like thyroid-stimulating hormone (TSH) levels, complete blood count (CBC), and folate, vitamin B-12 and electrolyte levels, to exclude these conditions, should be mentioned in a full scheme of diagnostic criteria for VND (Chawla, 2016).

2.3.5 Affected domains

Braaten et al. (2006) report findings from a study which highlight the important cognitive domains for differential diagnosis of several NCDs.

Their findings can be summarised as follows:

1.)

AD subjects perform worst in semantic fluency;

VND and FTLD subjects perform less worse in semantic fluency.

Mathematically stated:

$AD < \text{semantic fluency} < VND = FTLD$

i.e. Semantic fluency: $AD < VND = FTLD$

Read as: AD subjects are less semantically fluent than VND subjects who are equally semantically fluent as FTLD subjects.

2.)

AD and VND subjects perform worst in immediate memory;

FTLD subjects perform less worse in immediate memory.

Mathematically stated:

$AD = VND < \text{immediate memory} < FTLD$

i.e. Immediate memory: $AD = VND < FTLD$

3.)

FTLD subjects perform worst in executive functioning;

AD subjects perform the same or less worse in executive functioning;

VND subjects perform less worse in executive functioning.

Mathematically stated:

$FTLD = < AD < \text{executive functioning} < VND$

i.e. Executive functioning: $AD \geq FTLD < VND$

or $FTLD \leq AD < VND$

Read as: FTLD subjects are equally or less adept at executive functioning than AD subjects who are less adept at executive functioning than VND subjects.

Bringing the formulae together, we can easily view and appreciate the relative importance of these three domains in the diagnosis of the three disorders:

Semantic fluency: $AD < VND = FTLD$

Immediate memory: $AD = VND < FTLD$

Executive functioning: $FTLD \leq AD < VND$

As a result of this study, it was proposed that a battery for neuropsychological assessment of dementia would include “measures of executive functioning, memory, attention, concentration, intelligence, confrontational naming, phonemic fluency, and semantic fluency. Specific tests suggested for use include the Stroop Test, Wechsler Memory Scale—Third Edition, Wechsler Adult Intelligence Scale—Third Edition, Boston Naming Test, Controlled Oral Word Association Test, and Category Fluency (i.e., Animal Naming)” (Braaten et al., 2006, p. 1291). This sounds like a noble venture and definitely good for the type of accuracy that should be aimed at when diagnosing the various NCDs and for VND differential diagnosis in particular.

2.3.6 Comorbidity of VND with AD

The last note in DSM-5 under VND indicates that there is a high chance that those presenting with VND likely also have AD (APA, 2013, p. 624).

It may therefore be helpful to include some kind of reference to this as “additional diagnoses” in the main diagnostic criteria section for VND, and under the AD main criteria, an introductory subsection indicating possible previous or concomitant diagnoses as including VND.

2.4 HIV Infection

2.4.1 Affected domains

NCDs develop in some people who are infected with human immunodeficiency virus type-1 (HIV-1) (APA, 2013, p. 632), also generally referred to as “HIV”. Such NCDs are usually subcortical with the following domains being adversely affected: executive function, processing speed, attentional tasks, and learning new information; recall of learned information is often not problematic; aphasia (inability to understand words or to speak) is not typical, but fluency may slow (APA, 2013, p. 632).

2.4.2 Complex diagnostics and culture

An NCD due to HIV infection can otherwise be termed an “HIV Infection NCD” (HND).

An HND may still be diagnosed when other comorbidities such as syphilis, hepatitis C, drug abuse, neurodevelopmental conditions, or head injury exist, as long as it can be established that the HIV infection has exacerbated a prior NCD (APA, 2013, p. 634).

Studies have shown, however, that upwards of 20% of HND patients have bidirectionally fluctuating neurocognitive symptoms with a return to normality after a year of diagnosis (Antinori et al., 2007). This is indicative of possible misdiagnosis of a HIV-induced condition such as an NCD, when it may in fact not be an NCD, but could be temporarily mimicking one due to some other influence or combination of factors.

It seems therefore that neuropsychological testing could be producing false positives when it comes to HNDs, and that closer attention should be paid to the demographics of populations when it comes to test structure, content and composition. Factors such as age, culture, gender and education should be taken into account (Antinori et al., 2007).

Certain tests like the HIV Dementia Scale include three dimensional cube copying and timed alphabet writing, which may be totally inappropriate for a patient to execute who does not have a western background (Ganasen et al., 2008), thus producing potentially inaccurate diagnoses of HNDs. Such tests need to be re-written into multiple versions for the various linguistic cultures of South Africa.

In assessing urban blacks for HNDs, or for any other NCD for that matter, the clinician will need to have a sensitive understanding and appreciation of the various beliefs which patients might have, such as, 'water in the head', bewitchment, 'dirty organs', spirit possession, or 'something wrong with their blood' (Pillay, 1996). Such beliefs could influence the performance on specific items in neurocognitive tests as well as on such tests in general. They could also negatively impact on the quality of diagnosis if not correctly positioned. For example, an HIV+ patient who believes they are bewitched could be preoccupied by such thoughts leading to slower than usual responsiveness and distraction in a testing situation, which could result in lower scores and a faulty HND diagnosis, whereas the actual condition could be obsessive-compulsive disorder.

2.4.3 Antiretroviral Therapy (ART)

ART is a therapy in which several drugs are combined into a medicine taken by HIV infected (HIV+) people, which slows down the replication of the virus (WebMD, 2106), and thus lessens the severity of the infection's role in the development of possible acquired immunodeficiency syndrome (AIDS).

Neurocognitive impairment (NCI) has been shown to occur whilst undergoing ART post 1996, which poses the issue of the possible toxicity of the more recent ARV (antiretroviral) medications (Heaton et al., 2011). This confounds the diagnostics of HNDs, as the clinician may not know if the NCD is due to the infection or the treatment. Thus, a full history and current-usage description of all ARVs should be documented and analysed when assessing an apparent NCI in a patient.

Clinical tests indicate that three of the licenced inhibitors used in current ART (d4T, ddC and ddI) are linked to distal symmetrical polyneuropathy and lipodystrophy, and that these medications can cause acute lactic acidosis and hepatic steatosis (severe liver damage) (White, 2001). This can happen quickly and can lead to death.

The association of ARVs with central nervous system damage and NCI has important implications for diagnosticians. It means that a usual diagnosis of HND may provide a sufferer with whatever possible interventions may be at the clinician's disposal, but the latter might just be missing the one thing that could lead to amelioration of NCI symptoms and possible death, that is, a reappraisal of the patient's current ART regimen and overall reassessment for the necessity of ART itself with the possibility of a substitute therapy.

Although the question of alternative therapies for somatic HIV control, the reduction of AIDS symptoms (Rath et al., 2016), and the likely improvement of HND, is beyond the scope of this paper, this vital, potentially successful therapeutic avenue should receive urgent attention by researchers committed to the altruistic pursuit of aiding humankind.

3. Comorbidity, co-occurring and multiple disorders

In diagnosing NCDs, one needs to be ever aware of the possibility of other factors influencing the presentation, and that there may be other concurrent symptomatic conditions.

Two or more significant disorders may also co-occur within an individual (Psychology Today, 2016), such as post-traumatic stress disorder (PTSD) and HND or AD. The relative importance of the one disorder in relation to the other must be kept in mind when diagnosing cross-culturally, as certain communities may not even view certain conditions as abnormal (Pillay, 2000). Thus, PTSD, for example, may be viewed in a less debilitating light in certain communities.

Then there is the notion that one disorder may aid in mollifying the ill effects of another. It is suggested that PTSD might in some instances keep the brain

sufficiently active, involved, and stimulated, so as to have the effect of lessening the probability or exacerbation of arterial decay or neurological damage in NCD sufferers.

4. Conclusion

Mental or physical disorder diagnosis is not an exact exercise. Determining the disorder from which a patient is suffering is a complicated endeavour. Like all branches of medicine and psychology, NCD diagnosis shares in these difficulties.

Delirium, Alzheimer's, Vascular, and HIV NCDs in their current diagnostic delineations show areas of symptomatic overlap and similarity, as well as elements that uniquely distinguish them from each other.

Delirium is, generally, a temporary confusional state brought on by a medical condition, medication or exogenous substance.

Alzheimer's NCD (AD) is an impairment of several faculties of everyday living (notably semantic fluency, immediate memory and executive functioning) brought on by specific brain-area atrophy confirmed by neuroimaging and other methods such as cerebrospinal fluid analysis (NIH, 2016).

Vascular NCD (VND) is also such an impairment of a lesser degree than AD for the mentioned domains with its own distinctive pattern of cortical pathology.

HIV NCD (HND) differs from AD and VND which are cortically-located pathologies, in that it is usually subcortical, and affects the following domains: executive function, processing speed, attentional tasks, and learning of new information; recall of learned information is mostly unproblematic, unlike AD and VND.

Current instruments for diagnostic guidance, namely DSM-5 and ICD-10, should be made to present diagnostic criteria in a more consistent, thorough and comprehensive manner. Some of their current diagnostic criteria appear to have missing or incomplete elements and also require more descriptive detail in their listings.

The current presentation of six cognitive domains for assessment of NCDs seems also to be somewhat restrictive when one considers the possibilities of a broader range of identifiable domains, which might include, for example, executive function, attention, sensorimotor, learning, memory, language, intellect, visuospatial, emotions, and social (adapted from Neuropsychology Sketches, no date).

Neurocognitive tests need to be simplified, culturally adapted, and translated into the home languages of the various people of a geographic region. South Africa, for example, needs to have neuropsychological tests adapted for a number of linguistic cultures, including the following: Zulu, Xhosa, Afrikaans, English, Pedi, Tswana, Sotho, Tsonga, Swazi, Venda, Ndebele, Sign language, and the predominant languages of Zimbabwe, Nigeria, India, China, Pakistan, Bangladesh, the DRC, and Angola, as people from these countries make up a relatively large proportion of South Africa's residents (Statistics South Africa, 2013).

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