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Probable Vascular Dementia in a Tropical Environment: Study of Twenty-Six (26) Observations at the Conakry University Hospital

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ABSTRACT

Introduction: *In a tropical environment, diagnostic certainty of vascular dementia is difficult to establish due to under-medicalization, consultation delays and especially the inadequacy of exploration methods.*

Material and methods: *We analyzed the files of 153 patients hospitalized for dementia syndrome over a period from January 1, 2016 to December 21, 2021 in the Neurology Department of the Hospital Center. University of Conakry. Dementia status was assessed according to the Clinical Rating Scale (CDS), confirmed by cognitive tests of the Mini-Mental State Examination (MMSE <24) or by the Mental State Examination score. Neurobehavioral cognitive state (NCSE).*

Results: *26 (twenty-six) patients met the DSM-IV criteria for vascular dementia, based on the association of dementia and cerebrovascular disease certified by the presence of focal neurological signs of vascular origin and imaging data.*
Conclusion: this study shows a non-stereotyped clinical and etiological profile of the spectrum of vascular dementias in the tropics, in a context of under-medicalization. These results are useful for diagnostic and prognostic discussion.

Keywords
Dementia, Cerebrovascular disease, MRI, Conakry (Guinea).

Introduction
The existence of vascular dementias, especially the etio-clinical profiles, is now a well-established fact since the initial publications of Hachinski et al. [1,2], by Jorm et al. [3], by Roman et al. [4] by Anderson et al. [5] and recentRost NSet al. [6], ofIadecolaet al. [7]. Most of this work reveals the theoretical and practical difficulties which raise the nosology of this entity of vascular pathology and its limits with other conditions in which vascular lesions can be involved in their outbreak: Alzheimer's disease, mixed dementias [8].

In sub-Saharan Africa due to delays in consultation and treatment of vascular pathologies, dementias are gradually establishing

themselves and their etiological dismemberment is not easy [9]. We report a series of 26 patients in whom vascular diseases led to the emergence of dementia certified by psychometric and imaging tests. We studied the clinical, biological and neuroradiological profile of these patients in order to better specify the characteristics in a tropical environment. We therefore report 26 cases of presumed vascular dementia occurring in Conakry between 2016 and 2021 with the aim of a re-evaluation of this pathology from a clinical and etiological point of view. The interest of this work lies in the fact that these observations clearly illustrate vascular dementias and the difficulty in diagnosing them in a tropical environment in the vast group of subcortical dementias.

Material and Methods

This is a prospective study of a descriptive and analytical type lasting 5 years from January 1, 2016 to December 31, 2021 which concerned all patients hospitalized in the neurology department of the university hospital center of Conakry for a dementia syndrome. The inclusion criteria were as follows:

- Patients presenting with a dementia syndrome according to criteria adopted from the DSM-IV [10] decreed by the National Institute of Neurological Disorders and Strokes and those of ICD-10 [11].
- These criteria are underpinned by the association of dementia and cerebrovascular disease certified by a Hachinski score [1]: 0 and 4 points degenerative dementia and 7 and 8 points multiple infarction dementia.

With a view to detecting the vascular cause, the following explorations were also carried out:

- All subjects underwent a series of additional examinations: CBC, ESR, fasting blood sugar, 24-hour proteinuria, ionogram, blood calcium, fasting serum iron, 24-hour proteinuria, ionogram, blood calcium, serum iron, SGPT transaminases and SGOT, CPK, total plasma homocysteine, coagulation test, transaminases, CPK, coagulation test (PTA, TP, fibrinogen), fasting lipid profile: triglycerides, total cholesterol, LDL and HDL), prothrombin level.

A cardiological assessment was carried out according to the etiological orientation: ECG, cardiac ultrasound, Doppler of the vessels for encephalographic destination, and if necessary TEE and ETT.

The search for a vascular risk factor was reported in all patients: arterial hypertension, smoking, obesity, atrial fibrillation, migraine and estrogen-progestin contraception in women.

Brain MRI with coronal sections on the hippocampi.

As part of the differential diagnosis, an immunological assessment was carried out:

- FAN (with anti-DNA and anti-nuclear antibodies) ANCA, FR, ECA
- PCR (LCR), TPHA-VDRL, HSV1 and 2, VZV, EBV, HIV as well as HIV, HSV, VZV serologies.
- TP: prothrombin level; APTT: activated partial thromboplastin time; FAN: antinuclear factors;

ANCA: anti-neutrophil antibodies; RF: rheumatoid factor; ACE: angiotensin converting enzymes.

All patients underwent a neurological and psychiatric examination and according to the otolaryngological semiological orientation: by laryngoscope FNL-10KP3, Pentax (France), and ophthalmological (ophthalmoscope IEC LR6, 300 HEINE MINI Germany) and fundus examination. 'eye. All of these explorations make it possible to identify the conditions responsible for cerebral vasculitis: Sussac syndrome, Cogan disease.

All patients were subjected to two electroencephalographic examinations upon entering and leaving the establishment using a Nippon neurofax Japan EEG head box.

Results

The analysis of the results of this study focused on clinical, neuroradiological and electroencephalographic data. All 26 patients met DSM-IV and ICD 10 criterialabeled by the WHO in 1993 and these criteria underpinned by the existence of a dementia syndrome, cerebrovascular damage and the existence of a connection between the two elements. Table 1 summarizes the essential relationships between sociodemographic characteristics and potential risk factors for the appearance or existence of vascular dementia.

Table 1: General characteristics of vascular dementia (n= 32).

Variables	Number P %	Risk factors
1. Sex		
-Women	10(40.6%)	Estrogen-progestin
-Man	16(59.4%)	contraception (4)
2. Age at inclusion		
60-75 years old	8(34.4%)	
≥75 years old	18(66.6%)	
3. History of stroke		
Yes	25(90.6%)	-DALY (21); AVH (4)
No	1(9.4%)	Gaps (1)
4. Previous pathologieshearts		
Yes	9(37.5%)	FA (2). (TOG (2) AV (1)
No	17(62.5%)	FA(2). MI(2)
5. Alcohol consumption		
Yes	10(40.6%)	-Traditional alcohol:
No	16(59.4%)	Bandji; Dolo; Imported
6. Tobacco consumption		bere and alcohol
Smoking	5(25%)	
Non-smoker	21(75%)	
7. Diabetes		1 pack of cigarettes/day(1)
Yes	3(18.8%)	2 packs (4)
No	23(81.2%)	
8. Sedentary lifestyle, obesity		-
Yes	2(15.6%)	
No	24(84.4%)	-

AF: Atrial fibrillation. FA: Atrial Flutter ; TOG: Left ear thrombus. MM: myocardial infarction; AV: Akinesia of the left ventricle.

Table 2: Clinical signs at the onset and stage Clinical signs at onset and advanced stage of the disease**.**

Clinical Signs at the Early Phase	PERCENTAGE
Gradual loan start More or less discreet apathy Attention deficit and inability to acquire knowledge new type of fixation amnesia -Dysphoric disorders: mood, childishness -Periodic memory disorders -Moria frontal syndrome.	**22(84.6)**

Sudden start Memory problem Temporo-spatial disorientation	**4(15.4)**
NEUROPATHOLOGICAL SYNDROME IN THE STATE PHASE -Memory disorders certified by psychometric tests -Motor slowing sometimes associated with severe psychiatric syndromes, dreams, anxiety	**26/100**
NEURO-RADIOLOGICAL SYNDROME. Cortical and subcortical infarcts/hemorrhage sequelae Diffuse white matter damage.	**26/100**

Table 3: Etiological factors.

Form of anatomo-clinical	Number	Percentage
1. Multiple infarction dementia. **(**Vascular accidents >2 documented by clinic and neuroimaging (Multiple localization)	12	42.8
2. Incomplete state (Pure motor hemiparesis, no significant deficit, no cranial nerve damage, epileptic seizures)	**3**	**10.7**
3. Binswanger's disease (Focal neurological signs, gait disorders, dysarthria, dysphoric disorders, very high hypertension >22/ cm Hg)	**2**	**21.4**
4. Functional Infarcts (Predominantly brachiocephalic hemiparesis, subcortical aphasia, right hemisphere, hemi-neglect, epileptic seizures).	**6**	**10.7**
5. CADASIL (Migraine attacks, ischemic strokes, cognitive impairment, responsible type NOT CH3- CH2-19)	2	**5.1**
6. Amyloid Angiopathy (Recurrent lobar hematomas, pseudobulbar syndrome, Moria frontal syndrome.)	**1**	**2.6**

Neurorradiological Data

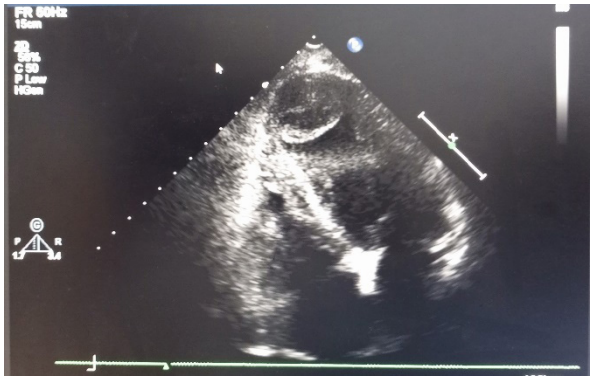
MRI angiography performed in the 12 patients with multiple infarct dementia and functional infarcts with evidence of stenoses (Figure 3). Angio in CADASIL (autosomal dominant cerebral arteriopathy with subcortical infarction and leukoencephalopathy). Note leukoaraiosis with periventricular hyperintensity type infarction with peripheral halo and extension in the SB.

In Binswanger's disease, MRI reveals diffuse but heterogeneous white matter damage in the periventricular regions, discreet images of cortical atrophy. The diagnostic certainty is neuropathological.

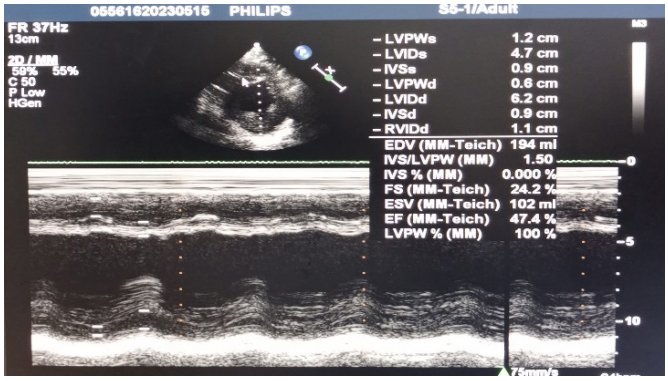
Discussion

This study reports 26 probable cases of vascular dementia determined at the Conakry university hospital center according to the criteria labeled in the DSM-IV [15] and NINDS-AIREN [16]. These are all suspected cases, some of which were diagnosed in the neurology department. Classically, the clinical diagnosis of vascular dementia is based on three elements [10-12] including the existence of a certified dementia syndrome [13], cerebrovascular damage and the presence of a link between the two [14].

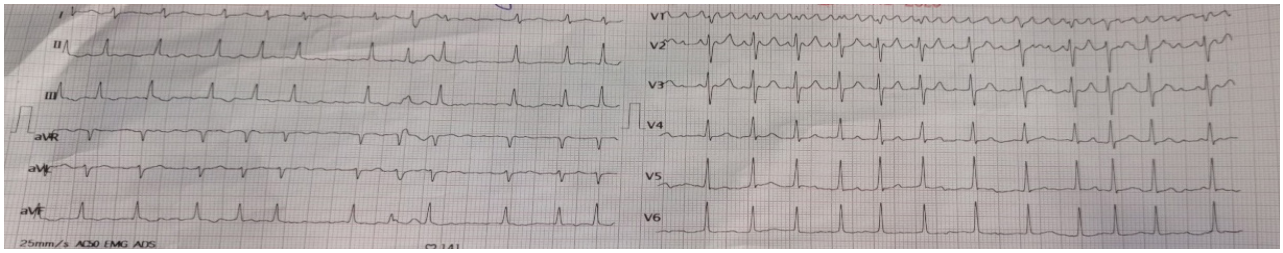
Hachinski et al. [1] in 1974 proposed a partition in favor of a vascular origin of these dementias contrasting with degenerative dementias. The certainty of this link remains questionable and raises several controversies sometimes casting doubt on the existence of vascular dementia [17], with several names: multiple infarctions [18] arteriopathic dementia [19], non-dementia cognitive disorders of origin vascular [20] highlighting the lack of consensus on the definition, pathophysiology and diagnosis [21].



Apical 4-chamber section showing a large intra-LV apical thrombus



Two-dimensional long major axis section coupled with a TM showing fu VG dilation



ECG: showing a complete arrhythmia by AF

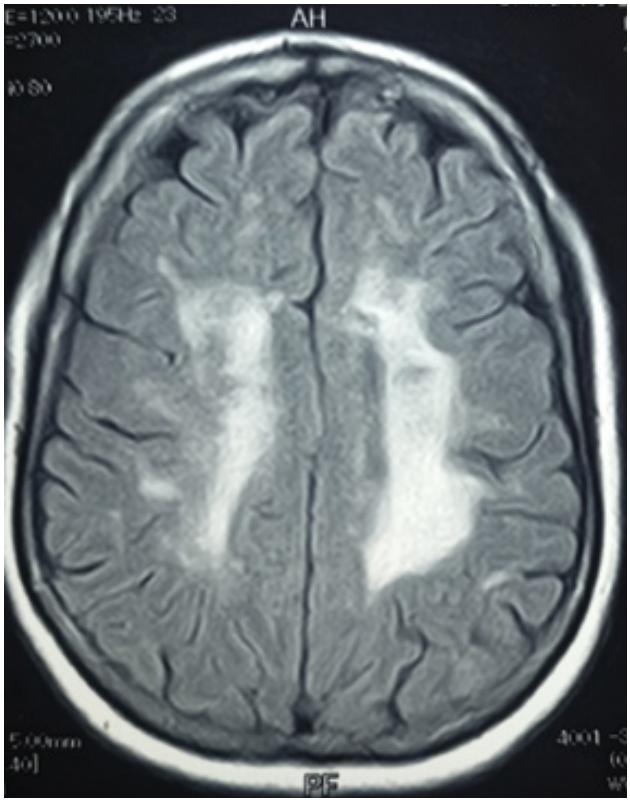


Figure 1: MRI in axial section (T2 Flair sequence): Hypersignals in the multiple and confluent semi-oval centers (Fazekas score 3).

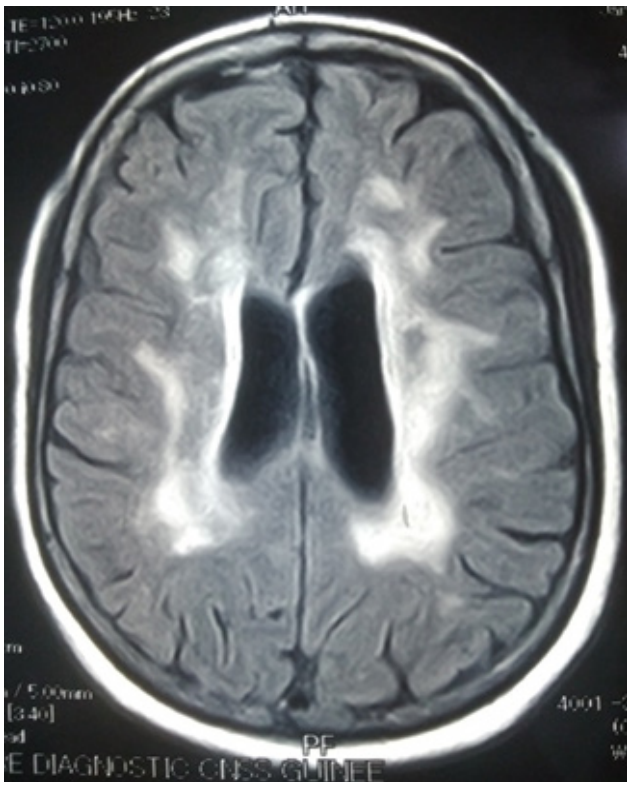


Figure 2: MRI in axial section (T2 Flair sequence): Periventricular hyperintensities with extension into multiple and confluent white substances (Fazekas score 3).

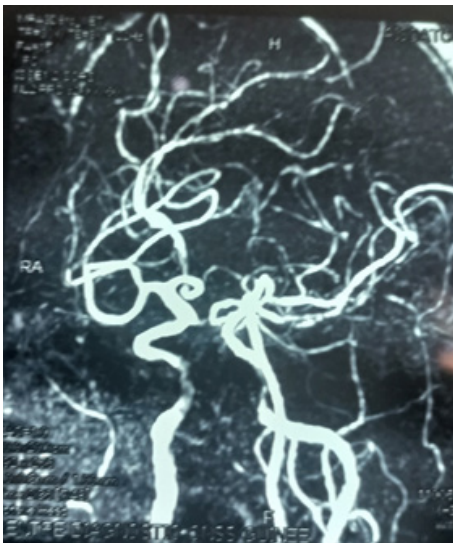


Figure 3: Multiple infarction with bi-carotid stenosis.

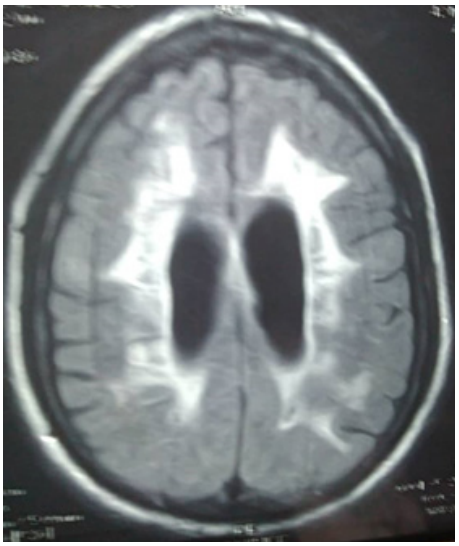


Figure 4: Diffuse but heterogeneous white matter in periventricular regions, images of cortical atrophy.

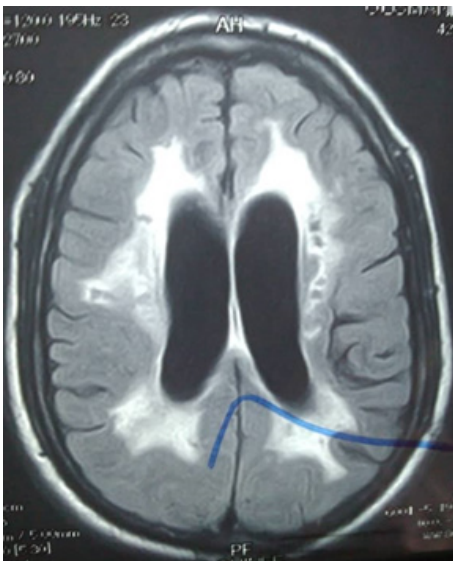


Figure 5: leukoariasis with lacunar type infarction, periventricular hypersignals with SB involvement

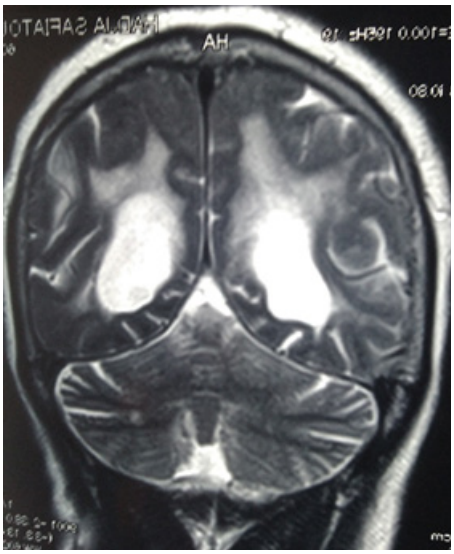


Figure 6: leukoariasis with periventricular hyperintensity type infarction with peripheral halo and extension into the SB

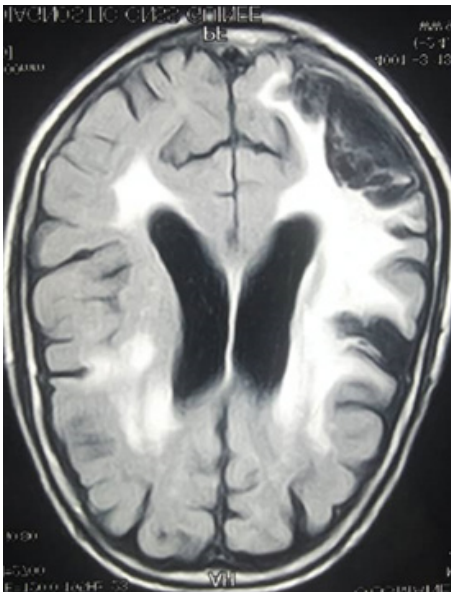


Figure 7: Coronal MRI T2 sequence: Confluent periventricular hyperintensities with extension into the SB: leukoaraiosis

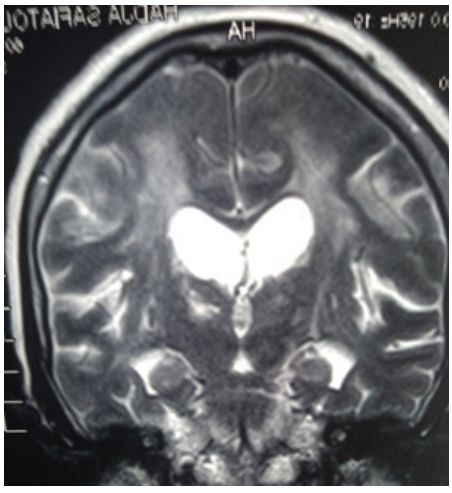


Figure 8: Coronal MRI T2 sequence: Confluent periventricular hyperintensities with extension into the SB.

These various controversies are underpinned by the non-existence of reliable pathological criteria making it possible to differentiate vascular dementias from mixed dementias that are both vascular and degenerative [22,15].

In sub-Saharan Africa, the high frequency of curable dementias neurosyphilis [23-25], HIV-associated dementias [25], metabolic and endocrine dementias, B12 deficiency, dysthyroidism, Wilson's disease [26], and degenerative diseases Huttington [27] and those associated with the different Parkinsonian syndromes make the delineation of vascular dementia difficult in a context of high frequency of subcortical dementias.

On the other hand the certainty of differentiation of vascular dementias from Alzheimer's disease clinico-biological entity characterized by neuropathological lesions biologically characterized by biomarkers of amyloid beta pathology (low level of AB42 in the cerebrospinal fluid or increased ratio AB40-AB42 in the CSF: accumulated retention of the PET tracer amyloid) and biomarkers of Tau pathology (increase in phosphorylated TAU in the CSF: increased retention of the PET tracer tau) remains complicated in tropical environments, in the absence of systematized biological explorations. These difficulties are also increased by the association of vascular lesions in Alzheimer's disease and Lewis bodies. In a documented series on CSF protein biomarkers in dementia, Gabelle et al. drew up a comparative table of the expected variations depending on the types of dementia [32].

Despite the high frequency of studies on vascular accidents in sub-Saharan Africa [9-15] and the diagnostic difficulties of vascular dementia, epidemiological data are non-existent in tropical environments and variable in the data. international according to the criteria used [16,17]. Estimate that vascular dementia is the leading cause of dementia in developing countries in Africa and Asia due essentially to insufficient prevention and management of cardiovascular diseases [18,19] note that the prevalence of vascular dementia is of the order of 8 to 13% contrasting with that of mixed dementia of 12 to 25% [20] the incidence is estimated at 2.52 per 1000 inhabitants.

In sub-Saharan Africa, the data are patchy and poorly documented [27] and this study is the first to our knowledge concerning the description of probable cases of vascular dementia. Generally speaking, the clinical presentations of vascular dementia present in this study do not essentially differ from those described in the literature [21,22], with two dominant entities: multiple infarctions and Binswanger's disease.

The vascular dementias observed in this study respond to the known clinical and neuroradiological characteristics of this condition: presence of lesions of vascular origin on brain imaging, reported existence of vascular accidents with focal signs more or less associated with a suprabulbar syndrome. A temporal relationship between stroke and dementia cognitive disorder [29]. Furthermore, we noted frequent and recurrent auditory hallucinations and illusions, probable expressions of an epileptic discharge in the T1 and T2 convolutions and Amon's horn, and in a second patient, repetitive complex visual hallucinations associated with language disorganization due to to an epileptic discharge in the dominant hemisphere following gaps in the temporal convexity and affecting the anterior part of the temporal lobe. These epileptiform syndromes have been observed in patients with multiple lacunae and thus match the semiology of vascular epilepsies.

In Africa, undermedication is an additional explanation for the severity of the cognitive disorders observed which are moreover most often observed late. In a tropical environment, dementias are also predominantly multifactorial, hence the importance of an extensive assessment of brain imaging in coronal sections on the hippocampi CBC, CRP, liver ionograms, vitamin B12 plasma folate TSH-US, VDRL-TPHA, HIV serology, PCR search for Tropheryma Whippeli etc.

Our study also shows the importance of MRI in vascular dementia. Although the lacunar state of Pierre Marie, Binswanger's disease and leukoaraiosis are associated anatomo-radiological entities. It shows in axial flair and sagittal sections hyperintense hypersignals, sometimes very marked, in the supratentorial white matter at the level of the oval and supracortical and periventricular centers as demonstrated by our study and many others [30]. Some authors note the existence of hyperintense signals in the insular regions and in the gray nuclei [31].

Conclusion

This study shows the presence and persistence of vascular dementia in Africa and the difficulty in diagnosing that it behaves in a tropical environment due to the high frequency of strokes due to insufficient prevention and treatment. burden of reported risk factors.

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