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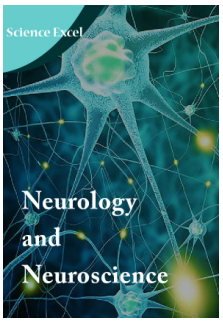
Original Article



Probable Vascular Dementia in the Tropics: A Study of Twenty-Six (26) Observations at Conakry University Hospital

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Keywords

Dementia, Cerebrovascular disease, MRI, Conakry (Guinea)

Abstract

Introduction: In tropical environments, the diagnostic certainty of vascular dementia is difficult to establish due to under-medicalization, delays in consultation and above all the inadequacy of exploration methods.

Material and methods: We analyzed the records of 153 patients hospitalized for dementia syndrome over a period from January 1, 2016 to December 21, 2021 in the Neurology Department of the Centre Hospitalo-. Universitaire de Conakry. Dementia status was assessed according to the Clinical Evaluation Scale (ECD), Confirmed by Mini Mental State Examination (MMSE<24) cognitive tests or Neuro, behavioral Cognitive Status Examination (NCSE) score.

Results: 26 (twenty-six) patients fulfilled 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.

Introduction

The existence of vascular dementias, especially their etio-clinical profiles, is now a well-established fact, since the initial publications of Hachinski et al. [1-2], Jorm et al. [3], Roman et al. [4], Anderson et al. [5] and more recent ones by Rost NS et al. [6] and Iadecola et al. [7].

Most of these works reveal the theoretical and practical difficulties raised by the nosology of this vascular pathology entity and its boundaries with other conditions in which vascular lesions may be involved in their outbreak: Alzheimer's disease, mixed dementias [8].

In sub-Saharan Africa, due to delays in consultation and management of vascular pathologies, dementia sets in gradually, and its etiological dismemberment is not easy [9].

We report a series of 26 patients in whom vascular disease led to the emergence of dementia certified by psychometric and imaging tests. We studied the clinical, biological and neuroradiological profiles of these patients in order to better define their characteristics in tropical environments. Thus, we report 26 cases of presumed vascular dementia occurring in Conakry between 2016 and 2021 with the aim of re-evaluating this pathology from a clinical and etiological point of view. The interest of this work lies in the fact that these observations illustrate vascular dementias and the diagnostic difficulty they entail in tropical environments within the vast group of subcortical dementias.

Material and methods

This is a prospective descriptive and analytical study lasting 5 years, from January 1, 2016 to December 31, 2021, and involving all patients hospitalized in the neurology department of Conakry University Hospital for dementia. Inclusion criteria were as follows:

- Patients presenting a dementia syndrome according to the DSM-IV criteria [10] published by the National Institute of Neurological Disorder and Stroke 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 signs of degenerative dementia, and 7 and 8 points of multiple infarct dementia.

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

- All subjects underwent a series of complementary examinations: CBC, VS, fasting blood glucose, 24-hour proteinuria, ionogram, blood calcium, fasting serum iron, 24-hour proteinuria, ionogram, blood calcium, serum iron, SGPT and SGOT transaminases, CPK, total plasma homocysteine, coagulation test, transaminases, CPK, coagulation test (aPTT, PT, fibrinogen), fasting lipid panel: triglycerides, total cholesterol, LDL and HDL), prothrombin level.

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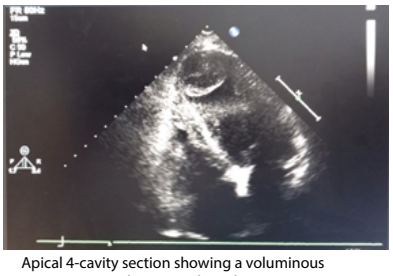
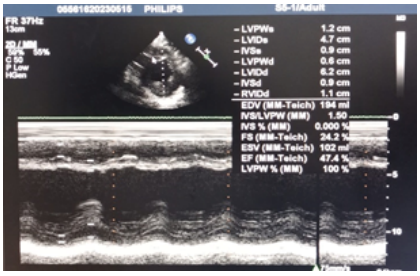
***Table 1**. General characteristics of vascular dementia (n= 32).*

Variables	Number P % (%)	Risk factors
1. Gender -Female -Male	 10(40,6%) 16(59,4%)	Contraception oestroprogestative (4)
2. Age at inclusion 60- 75 years ≥ 75 years	 8(34,4%) 18(66,6%)	
3. History of stroke Yes No	 25(90,6%) 1(9,4%)	-AVCI (21) ; AVH (4) Gaps (1)
4. History of heart disease Yes No	 9(37,5%) 17(62,5%)	AF (2). TOG (2) AV(1) AF (2). I.M (2)
5. Alcohol consumption Yes No	 10(40,6%) 16(59,,4%)	Traditional alcohol: Bandji; Dolo; Béré and imported alcohol
6. Tobacco consumption Smoker Non-smoker	 5(25%) 21(75%)	1 pack of cigarettes/day (1) 2packs (4) -
7. Diabetes Yes No	 3(18,8%) 23(81,2%)	-
8. Sedentary lifestyle, obesity Yes No	 2(15,6%) 24(84,4%)	

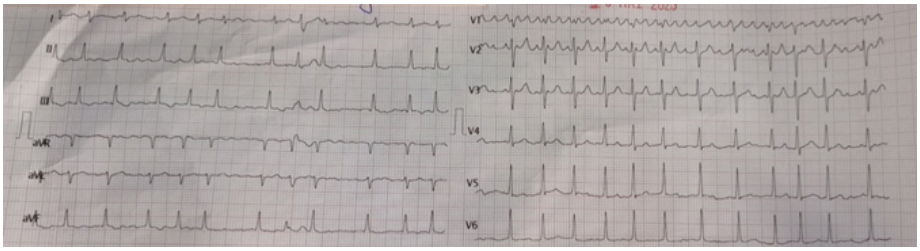
AF: Atrial Fibrillation; AF: Atrial Flutter; TOG: Left ear thrombus; M.M: Myocardial infarction; AV: Left Ventricular Akinesia



Apical 4-cavity section showing a voluminous apical intra-VG thrombus



Two-dimensional long-axis section with TM showing LV dilatation



ECG: recording a complete arrhythmia due to AF

All patients underwent a neurological and psychiatric examination and, depending on their clinical orientation, an otolaryngological examination using the FNL-10KP3 laryngoscope (Pentax, France), an ophthalmological examination using the IEC LR6,300 HEINE MINI ophthalmoscope (Germany) and a fundus examination. All these investigations enabled us to identify the conditions responsible for cerebral vasculitis: Sussac syndrome, Cogan's disease.

All patients underwent two electroencephalographic examinations using a Nippon neurofax Japan head box EEG at the start and end of their stay.

Results

Analysis of the results of this study focused on clinical, neuroradiological and electroencephalographic data. All 26 patients met the DSM-IV and ICD-10 criteria ¬labelled by the WHO in 1993, and these criteria were underpinned by the existence of a dementia syndrome, cerebrovascular involvement and the existence of a link between the two elements. Table I summarizes the main relationships between sociodemographic characteristics and potential risk factors for the onset or existence of vascular dementia.

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

CLINICAL SIGNS IN THE EARLY PHASE	**PROPORTION**
Slow progressive onset	22(84,6)
More or less discreet apathy	
Attention deficit and inability to acquire new knowledge (fixation amnesia)	
-Dysphoric disorders: moodiness, childishness	
-Periodic memory disorders	
-Moria frontal syndrome	
Sudden onset	4(15,4)
Memory impairment	
Temporo-spatial disorientation	
NEUROPATHOLOGICAL SYNDROME IN THE STATE PHASE	26/100
-Memory impairment certified by psychometric tests	
-Motor slowing sometimes associated with severe psychiatric syndromes: fantasies, anxiety	
NEURO-RADIOLOGICAL SYNDROME	26/100
Cortical and subcortical infarcts/sequelae of hemorrhage	
Diffuse white matter involvement.	

Table 3. *Etiological factors*

Anatomical-clinical forms	**Number**	**Proportion**
1. Multiple infarct dementia. *(Clinically and neuroimaging-documented vascular events >2 (Multiple locations)*	12	42,8
2. Deficient condition *(Pure motor hemiparesis, no sensory deficit, no cranial nerve damage, epileptic seizures)*	3	10,7
3. Binswanger disease *(focal neurological signs, gait disorders, dysarthria, dysphoric disorders, very high blood pressure >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, gender NOT CH3- CH2-19)*	2	5,1

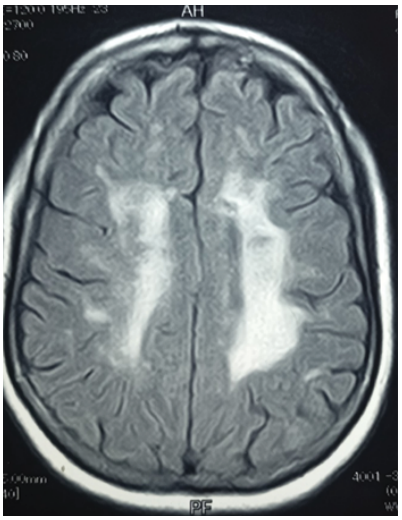


Figure 1. *MRI axial section (T2 Flair sequence): Hypersignals in multiple confluent semi-oval centers (Fazekas score 3).*

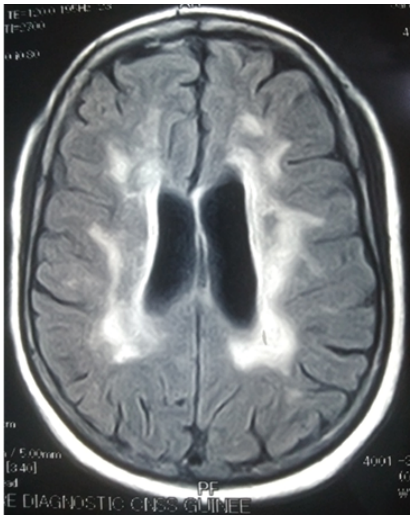


Figure 2. *MRI axial section (T2 Flair sequence): Periventricular hypersignals with extension into multiple, confluent white matter (Fazekas score 3).*

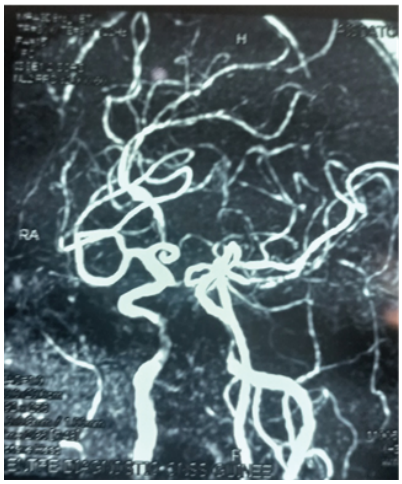


Figure 3. *Multiple infarction with bi-carotid stenosis*

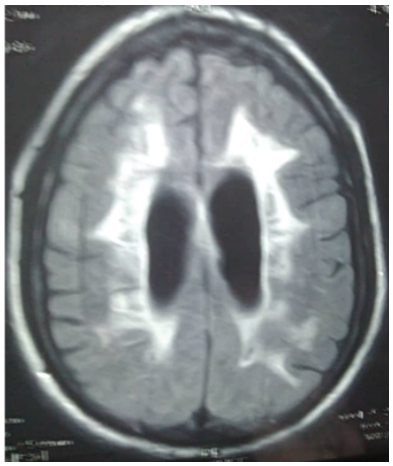


Figure 4. *Diffuse but heterogeneous white matter in periventricular regions, images of cortical atrophy.*

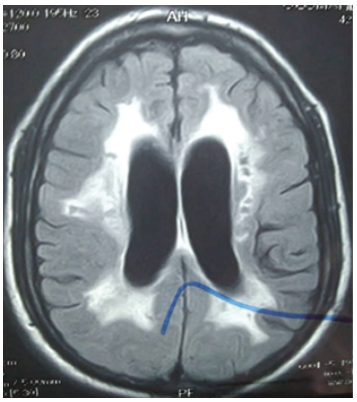


Figure 5. *Leukoariosis with lacunar infarction, periventricular hypersignals with SB involvement*

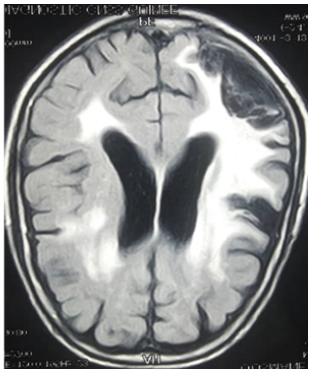


Figure 6. *Leukoariosis with periventricular hypersignal infarcts with peripheral halo and extension into the SBs*

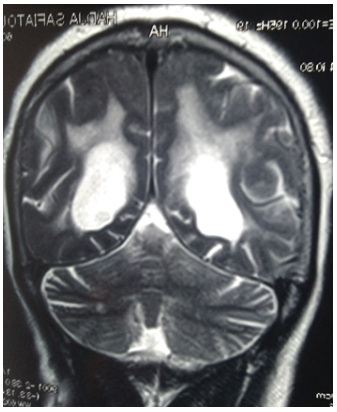


Figure 7. *Coronal slice MRI T2 sequence: confluent periventricular hypersignals extending into the SB: leucoaraiosis*

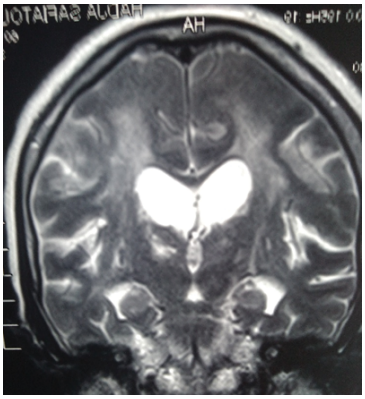


Figure 8. *Coronal slice MRI T2 sequence: Confluent periventricular hypersignals extending into the SBs*

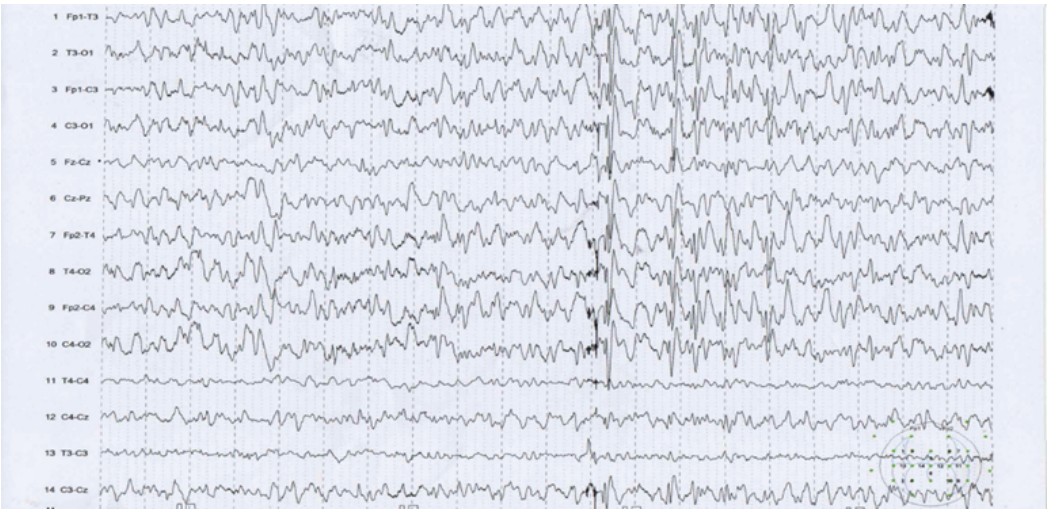


Figure 9. *Type III EEG trace showing abnormal theta and delta rhythms*

Neuroradiological Data

Angio-MRI in 12 patients with dementia caused by multiple infarcts and functional infarcts revealed stenoses (Fig 3). Angio In CADASIL (cerebral Autosomal dominant arteriopathy with subcortical infarct and leukoencephalopathy). Leukoaraiosis with periventricular hypersignal infarcts with peripheral halo and extension into the SBs.

In Binswanger disease, MRI reveals diffuse but heterogeneous white matter involvement in the periventricular regions, and discrete images of cortical atrophy.

Diagnostic certainty is neuropathological.

Electroencephalographic Data

A non-specific alteration of the EEG tracings (type II) was detected in 24 patients (85.7%) with no specific abnormalities, in contrast to 4 patients (14.2%) with epileptic seizures (type III) in our classification. (Fig 9).

Discussion

This study reports 26 probable cases of vascular dementia diagnosed at the University Hospital of Conakry according to DSM-IV [15] and NINDS-AIREN [16] criteria. These are all presumed cases, some of which were diagnosed in the neurology department. Classically, the clinical diagnosis of vascular dementia is based on three elements [10,11, 12], including the existence of a certified dementia syndrome [13], cerebrovascular involvement and the presence of a link between the two [14].

In 1974, Hachinski et al [1] proposed a score in favour of a vascular origin for these dementias, in contrast to degenerative dementias. The certainty of this link remains debatable, and has given rise to a number of controversies, some of which cast doubt on the existence of vascular dementia [17], with several different designations: multiple infarcts [18] arteriopathic dementia [19], non-dementia cognitive disorders of vascular origin [20], highlighting the lack of consensus on definition, pathophysiology and diagnosis [21]. Underlying these various controversies is the lack of reliable pathological criteria for differentiating vascular dementia from mixed vascular and degenerative dementia [22,15].

In sub-Saharan Africa, the high frequency of curable dementias neurosyphilis [23, 24,25], HIV-associated dementias [25], metabolic and endocrine dementias, B12 deficiency,

dysthyroidism, Wilson's disease [26], and degenerative Huttington's diseases [27] and those associated with the various 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 differentiating vascular dementia from Alzheimer's disease, a clinico-biological entity characterized by neuropathological lesions biologically characterized by biomarkers of beta amyloid pathology (low AB42 levels in cerebrospinal fluid or increased AB40-AB42 ratio in CSF : increased retention of the amyloid PET tracer) and biomarkers of tau pathology (increased phosphorylated TAU in CSF: increased retention of the tau PET tracer) remains complicated in tropical environments, in the absence of systematized biological explorations. These difficulties are further compounded 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 have drawn up a comparative table of expected variations according to dementia type [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 international data depending on the criteria used. Nevertheless [16,17] estimate that vascular dementia is the leading cause of dementia in developing countries in Africa and Asia, essentially due to inadequate prevention and management of cardiovascular disease [18,19] note that the prevalence of vascular dementia is in the order of 8 to 13%, in contrast to mixed dementias at 12 to 25% [20], with an estimated incidence of 2.52 per 1000 inhabitants.

In sub-Saharan Africa, data are patchy and poorly documented [27], and this study is the first to our knowledge to describe probable cases of vascular dementia.

Generally speaking, the clinical pictures of vascular dementia observed in this study do not differ fundamentally from those described in the literature [21,22], with two entities dominated by multiple infarcts and Binswanger's disease.

The vascular dementias observed in this study correspond to the known clinical and neuroradiological characteristics of this condition: presence of lesions of vascular origin on cerebral imaging, reported existence of vascular accidents with focal signs more or less associated with a supra bulbar syndrome, and a temporal relationship between the stroke and cognitive dementia [29]. In addition, we noted frequent and recurrent auditory hallucinations and illusions, likely expressions of 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 epileptic discharge in the dominant hemisphere following lacunae in the temporal convexity and involving the anterior part of the temporal lobe. These epileptiform syndromes have been observed in patients with multiple lacunae, and are thus consistent with the semiology of vascular epilepsy.

In Africa, under-medication is a further explanation for the severity of the cognitive disorders observed, which are more often than not diagnosed late. In tropical environments, dementia is also mostly multifactorial, hence the importance of an extensive work-up: coronal cross-sectional brain imaging of the hippocampi, CBC, CRP, liver function tests, ionograms, vitamin B12, plasma folate, TSH-US, VDRL-TPHA, HIV serology, PCR for Tropheryma Whippeli, etc.

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

Conclusion

This study shows the presence and persistence of vascular dementia in Africa, and the diagnostic difficulty it entails in tropical environments due to the high frequency of strokes caused by inadequate prevention and management of notified risk factors.

Declaration of interest

The authors declare that they have no conflicts of interest.

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