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Clinical evaluation of the ICD-10 criteria for vascular dementia

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Summary. The clinical feasibility of the ICD-10 criteria for subtypes of vascular dementia were examined in an investigation of 61 demented patients (74.4, SD 8.2 years) showing CT appearances of vascular lesions. Only 15 cases (24.6%) fulfilled the ICD-10 criteria of vascular dementia. Of these cases, 66.6% could adequately be classified in subtypes. The most frequent types of vascular dementia were multi-infarct dementia and subcortical vascular dementia. Our findings suggest that the ICD-10 criteria of vascular dementia are more selective than the classical 'ischaemic scales'.

Key words: ICD-10 – Dementia – Vascular dementia – Multi infarct dementia

Introduction

Vascular diseases are the second most common cause of dementia in Caucasians and seem to be the main cause in Asian countries [35, 58]. The autopsy of about 15–29% of all demented people reveals only pathological changes due to vascular disorders and a further 10–20% have vascular as well as degenerative changes [1, 12, 34, 46, 49, 73, 74, 76]. Nevertheless, the histopathological features of vascular dementia (VD) have not yet been established [12, 34, 72, 76].

Recent reviews of VD have stressed the need to distinguish between the various causes [15, 48, 64]. Nevertheless, all types of VD are often subsumed under the term multi-infarct dementia (MID) as in the widely used DSM-III-R [2]. But the concept of MID [29, 30, 74] postulating multiple small cerebral infarcts with a volume greater than 60 ml [74] causing dementia has been criticized mainly for the three following reasons:

1. The size of the vascular lesions considered to be responsible for dementia is still a matter of debate and seems to be very variable [25, 44, 74]. Sometimes even very small infarcts (< 5 ml [25, 44]) may cause dementia.

2. How multiple infarcts can cause dementia has yet to be determined. Multiple lesions, each impairing a certain cognitive function, might lead to dementia simply on an additive basis. In view of the fact that many patients with lacunae are asymptomatic (up to 81% [75]), a multiplicative mechanism becomes more likely. Nevertheless, the concept of MID, namely that several lesions, each individually innocuous or trivial in its clinical symptoms, cause dementia, still needs validation.

3. MID is not the only type of VD. Dementia can be caused by a variety of vascular processes [64].

Thus, the widely used term MID seems seems inadequate for denoting the whole group of vascular dementias. Some proposals for a classification of VD subtypes have been made [12, 15, 21, 28, 41, 48, 67, 79], but none or only very few clinical criteria are given to differentiate subtypes of VD. Up to now virtually no studies have been undertaken to test these criteria [22, 48].

In the ICD-10 [79] the following general criteria for VD are given (Table 1).

Table 1. ICD-10 criteria for vascular dementia

A.	Evidence of a dementia of specified level of severity, as set out under the general criteria of dementia.
B.	Unequal distribution of deficits in higher cognitive functions, with some affected and others relatively spared. Thus memory may be quite markedly affected while thinking, reasoning and information processing may show only modest impairment.
C.	Evidence for focal brain damage as indicated by one or more of the following: a history of stroke (usually several); unilateral spastic weakness of the limbs; unilaterally increased tendon reflexes, an extensor plantar response, pseudobulbar palsy.
D.	Evidence from the history, examination, or tests of significant cerebrovascular disease which may reasonably be judged to be aetiologically related to the dementia.

The diagnosis is supported by evidence of infarction from special investigations, such as CT, PET or MRI. It is confirmed on neuropathological examination of the brain at autopsy.

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The characteristics of the VD subtypes are:

VD of acute onset (F 01.0)

The dementia develops rapidly (i.e. usually within 1 month, but within no longer than 3 months) after a succession of strokes, or (rarely) after a single massive haemorrhage.

Multi-infarct (predominately cortical) VD (F 01.1)

The onset of the dementia is more gradual (i.e. within 3–6 months) following a number of minor ischaemic episodes which produce an accumulation of lacunae in the cerebral parenchyma. There may be periods of actual clinical improvement.

Subcortical VD (F 01.2)

A. A history of hypertension.
B. Evidence from clinical examination and special investigations of vascular disease located in the deep white matter of the cerebral hemispheres, with preservation of the cerebral cortex.

Mixed cortical and subcortical VD (F 01.3)

No specific criteria are given in the ICD-10 research criteria.

Other VD (F 01.8) and VD, unspecified (F 01.9)

No specific criteria are given in the ICD-10 research criteria. Furthermore, the following findings are also considered to be linked with VD: stepwise deterioration of intellectual impairment or neurological signs [30]; (multiple) focal neurological signs [2, 30]; evidence of other vascular disorders [30]; vascular-caused lesions of the brain, visible on CT or MRI [43]; focal cerebral hypoperfusion, visible in PET or SPECT as well as in measurements of the cerebral blood flow [13, 16, 29, 31, 33]; focal changes on the EEG [14, 23, 50, 57].
In this study, the feasibility of the ICD-10 research criteria for a differentiation of VD subtypes is tested in a clinical investigation. The results are compared with the classification according to 'ischaemic scores' [29, 44, 61].

Patients and methods

Included in this study were 61 patients, admitted to the psychiatric or neurological department of the Medical University of Lübeck, who fulfilled the ICD-10 criteria of dementia and showed vascular lesions on CT. The sample consisted of 38 women and 23 men (mean age: 74.4, SD 8.2 years). All were Caucasians and had had an education of 8 or more years.

The Dementia Scale [8] (slightly modified for use in Germany) and the Mini-Mental-State Examination [26] were applied to assess the severity of dementia (Table 2).

The ICD-10 research criteria [79] were used to diagnose dementia, VD and to specify VD subtypes. The quantification of cognitive function was made according to algorithms adapted from the SIDAM [80]. The maximum score of each cognitive function (Table 3) was set as 100%. An unequal distribution of cognitive deficits was assumed if in one individual the percentage of distinct functions differed by more than 25%. The 'ischaemic scores' were applied according to the original instructions [29, 44, 61].

Table 2. Severity of dementia in relation to age and sex

	n %	Sex	Mean age (years)[a]
Mild dementia (MMSE ≥ 20)	33 54.1%	24 f 9 m	72.4, SD 7.9
Moderate dementia (MMSE 11–19)	13 21.3%	10 f 3 m	75.8, SD 6.5
Severe dementia (MMSE < 10)	15 24.6%	4 f 11 m	77.5, SD 9.5

MMSE, Mini-Mental State Examination; f, female; m, male
[a] *U*-test n.s.

Table 3. Cognitive deficits in 61 patients with probable vascular dementia (according to the SIDAM [80])

	n	%
Orientation	22	36.1
Immediate recall	14	23.0
Short-term memory	42	68.9
Long-term memory	39	63.9
Intellectual abilities	34	55.7
Verbal abilities/calculation	32	52.4
Constructional abilities	40	65.6
Aphasia/apraxia	15	24.6

Deficits of a cognitive function are considered to be present, if the patient attains less than 50% of maximal score

The CT scans were evaluated by the criteria of Ringelstein et al. [56] by a radiologist without information on the clinical findings.

1. Territory infarcts were defined as infarcts sharply demarcated on CT conforming with the supply area of a great artery like the middle cerebral artery (even if incomplete).
2. Watershed or border zone infarct were defined as infarcts in the terminal supply areas.
3. Lacunar infarcts were characterized as small infarcts with a diameter less than 2 cm, mainly located in the deep white matter and the basal ganglia and thalamus.
4. Leuko-araiosis is defined as patchy, diffuse hypodense areas predominantly located bilaterally around the ventral and posterior horns of lateral ventricles on CT. The extent of leuko-araiosis was estimated by the semiquantitative method of Rezek et al. [55].

According to the results of follow-up studies [19, 59], hypertension (>160 mmHg systolic/95 mmHg diastolic), diabetes mellitus (glucose >100 mg/l, cardiac arrhythmia, a history of myocardial infarction, and hyperlipidaemia [cholesterol >6.55 mmol/l (females) >6.90 mmol/l (males) and or triglyceride >2.00 mmol/l (females) >3.20 mmol/l (males)] are considered to be important cerebrovascular risk factors. To exclude patients with cerebral lesions possibly caused by an inflammatory or immunological process, cases with a blood sedimentation rate more than 20 mm (first hour) underwent further investigations including. TPHA test, and anti-nuclear antibodies etc. In 1 case doubtful an analysis of the cerebrospinal fluid was added.

Results

The CT findings (according to the criteria of Ringelstein et al. [56] are shown in Table 4.

Table 4. CT findings in 61 patients with probable vascular dementia (according to [56])

$N = 61$	n	%
Territory infarcts	7	11.5
Watershed or border zone infarcts	0	0
Cerebral microangiopathy		
– only lacunar infarcts	9	14.8
– only leuko-araiosis	19	31.1
– leuko-araiosis + lacunar infarcts	20	32.8
Haemorrhages	0	0
No classification possible (mixed infarcts)	6	9.8

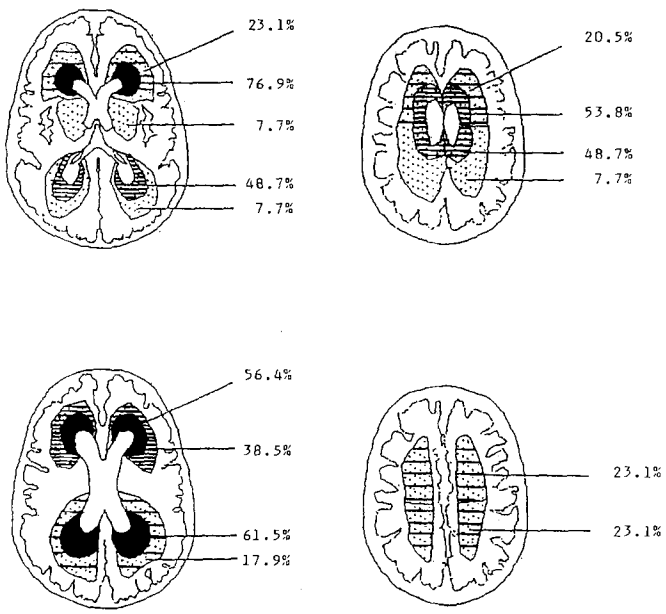

Fig. 1. Extent and location of white-matter lesions in 39 patients with microangiopathy (according to Rezek et al. [55])

Cerebral microangiopathy (leuko-araiosis and lacunar infarcts) is the most frequent type of lesion seen on CT (78.7%) and in territorial infarcts (11.5%). The CT of 6 patients (9.0%) revealed mixed infarcts. The extent and location of leuko-araiosis is given in Fig. 1. The semiquantitatively estimated extension of leuko-araiosis neither correlated with the severity of dementia (Mini-Mental State) ($r = 0.08$) or with the 'ischaemic score' [29] ($r = 0.20$). The territory infarcts were all incomplete and were mostly located in the supply area of the middle cerebral artery. They were located in the dominant left hemisphere in 4 cases, and bilaterally in 3 cases. Of all patients, 70.5% had only white matter lesions. The frequencies of findings considered to be linked with VD are shown in Table 5.

Only 53.1% of the patients showed focal neurological symptoms or had a history of stroke, indicating focal brain damage. Of the cases, 34.4% had an unequal distribution of deficits in higher cognitive functions. Nevertheless, these findings are essential ICD-10 criteria for the diagnosis of VD. Thus, only 24.6% of the sample fulfilled the ICD-10 criteria for VD.

Table 5. Frequency of findings characteristic for vascular dementia

$N = 61$	n	%
Stepwise deterioration of the intellectual impairment or neurological signs	31	50.8
Sudden onset	17	27.9
A history of stroke	21	34.4
(Multiple) focal neurological signs	27	44.2
– Unilateral (spastic) weakness of limbs	11	18.1
– Unilateral increased tendon reflexes	16	26.2
– Extensor plantar response	10	16.4
– Pseudobulbar palsy	3	5.0
(Multiple) focal neuropsychological deficits	29	52.5
– Aphasia	15	24.6
Evidence for cerebrovascular risk factors	50	82.0
– Hypertension	37	60.7
– Hyperlipidaemia	17	27.9
– Diabetes mellitus	17	27.9
– Cardiac arrhythmia	19	31.1
Evidence of other vascular disorders	18	29.5
– Myocardial infarction	7	11.5
Vascular-caused lesions of the brain, visible on CT scans	61	100[a]
Focal changes in EEG	16	26.2

[a] Essential criterion for the sample

Table 6. ICD-10 criteria for vascular dementia

$N = 61$	n	%
– Evidence of dementia	61	100[a]
– Unequal distribution of cognitive functions	21	34.4
– Evidence of focal brain damage	33	54.1
– Evidence of a cerebrovascular disease	52	85.2
General ICD-10 criteria for vascular dementia	15	24.6
– Rapid onset	0	0
Vascular dementia of acute onset [F 01.0]	0	0
– Gradual onset	31	50.8
Multi-infarct dementia [F 01.1]	6	9.8
– History of hypertension	37	60.7
– White matter changes on CT	50	82.0
Subcortical vascular dementia [F 01.2]	3	4.9
– Cortical and subcortical changes on CT[b]	6	9.8
Mixed cortical and subcortical vascular dementia [F 01.3]	2	3.3
Vascular dementia, unspecified [F 01.9]	3	4.9
Dementia of Alzheimer's type	14	23.0
Mixed vascular/Alzheimer's dementia	1	1.6
Cases that could not be specified	31	50.8

[a] Essential criterion for the sample
[b] This criterion is not included in the ICD-10 research criteria

The criteria for MID were fulfilled in 8 cases, those for subcortical VD in 6 cases and that for VD of acute onset in none of the patients. An interesting aspect is that 15 cases (about 25%) of the sample fulfilled the ICD-10 criteria for dementia of the Alzheimer type (DAT). One case (1.6%) met the ICD-10 criteria of VD as well as those of DAT; it was therefore classified as a mixed case. In sum, 66.6% of the 15 cases diagnosed as

Table 7. Rating by 'ischaemic scales'

	VD	DAT	Unclassified
Hachinski Score [29]	9.3, SD 2.4*	1.7, SD 2.1	7.4, SD 4.3**
Loeb Score [44]	5.9, SD 2.5*	3.1, SD 2.5	5.5, SD 3.0**
Rosen Score [61]	6.0, SD 2.8*	0.7, SD 1.4	4.8, SD 3.9**

* $P < 0.001$ vs DAT; ** $P < 0.01$ vs DAT (Mann-Whitney U-test)

Table 8. Cases fulfilling the ICD-10 research criteria for vascular dementia (VD) or dementia of Alzheimer's type (DAT)

CT findings	*n*	VD (%)	DAT (%)
Territory infarcts	7	28.6	14.3
Cerebral microangiopathy			
– only lacunar infarcts	9	22.2	33.3
– only leuko-araiosis	19	21.1	26.3
– leuko-araiosis + lacunar infarcts	20	20.0	15.0
No classification possible (mixed infarcts)	6	33.3	33.3

VD according to the ICD-10 criteria belonged to a specific VD subtype. According to the ICD-10 criteria, only 49.2% of all cases could be classified adequately (Table 6).

The cases that could not be classified by the ICD-10 criteria do not differ from the defined VD cases, while the DAT-like cases differed in some aspects, especially the number of focal neurological signs (unilaterally increased tendon reflexes, etc.). If the cases were rated by the 'ischaemic scales' [29, 44, 61], the DAT-like cases had significantly lower scores (VD vs DAT-like vs unclassified cases, Table 7):

If devided into groups by the CT findings (as shown in Table 8), no significant difference in percentage of classified cases could be found. Only 20–33.3% of the cases in each CT group could be specified as VD. In total, in about 50–65% of the cases, the diagnosis remains obscure if the ICD-10 research criteria are applied.

The comparison of the classification according the ICD-10 criteria and that by the 'ischaemic scales' shows fairly good agreement.

Table 9. Comparison of classification between ICD-10 and 'ischaemic scales'

ICD-10 – Hachinski scale [29]	85.7%	χ^2 27.6	*df* 4	$P < 0.001$
ICD-10 – Loeb scale [44]	39.3%	χ^2 10.8	*df* 4	$P < 0.05$
ICD-10 – Rosen scale [61]	85.7%	χ^2 19.4	*df* 4	$P < 0.001$

Table 10. Differentiation according to 'ischaemic scales'

CT findings	Degen.	MID	?
Hachinski Scale [29]	0–4	7–18	5–6
Territory infarcts	14.3%	71.4%	14.3%
Cerebral microangiopathy			
– only lacunar infarcts	22.2%	66.7%	11.1%
– only leuko-araiosis	47.4%	21.1%	31.6%
– leuko-araiosis + lacunar infarcts	40.0%	45.0%	15.0%
No classification possible (mixed infarcts)	33.3%	50.0%	16.7%
	36.1%	44.3%	19.7%
Loeb Scale [44]	0–2	5–10	3–4
Territory infarcts	14.3%	71.4%	14.3%
Cerebral microangiopathy			
– only lacunar infarcts	11.1%	33.3%	55.6%
– only leuko-araiosis	5.3%	21.1%	73.7%
– leuko-araiosis + lacunar infarcts	10.0%	50.0%	40.0%
No classification possible (mixed infarcts)		50.0%	50.0%
	8.2%	41.0%	50.0%
Rosen Scale [61]	0–2	4–10	3
Territory infarcts	14.3%	85.7%	
Cerebral microangiopathy			
– only lacunar infarcts	33.3%	55.6%	11.1%
– only leuko-araiosis	57.9%	26.3%	15.8%
– leuko-araiosis + lacunar infarcts	47.4%	52.6%	
No classification possible (mixed infarcts)	33.3%	50.0%	16.7%
	43.3%	48.3%	8.3%

degen., Degenerative dementia (i.e. DAT); MID, multi-infarct or vascular dementia respectively; ?, unclassified cases

But the proportion of cases that can be classified is higher if the 'ischaemic scales' [29, 61] are applied: 80.3% [29] and 91.7% [61] vs 49.2% (ICD-10) (see Tables 6 and 10). The use of the scale of Loeb et al. [44] does not lead to a higher number of classified cases. As regards the 'ischaemic' scores there are no significant differences between the CT groups.

Discussion

Up to now only a few studies [22, 25, 48, 67] have been aimed at differentiating VD clinically. Because of the different criteria used in these investigations, a comparison is hampered. To our knowledge this study is the first to evaluate the ICD-10 criteria of VD in a clinical investigation.

Only 24.6% of a sample of 61 patients fulfilling the ICD-10 criteria of dementia and showing vascular lesions on CT met the ICD-10 criteria of vascular dementia. These results can be interpreted in different ways. First, the ICD-10 research criteria show a high selectivity. Second, even when CT reveals vascular lesions, dementia can not always be classified as vascular. Third, dementia is a multi-factorial disorder [64], and vascular changes, if they occur, may become the pacemaker of the dementive decline.

Selectivity of the ICD-10 criteria

Our results suggest a high selectivity of the ICD-10 criteria for VD. With this view in mind the criteria are discussed in detail.

Dementia. Only a proportion of people suffering from a cerebrovascular disease show a dementing decline. Thus, dementia has to be established by clinical examination. With regard to the great heterogeneity of vascular lesions, it is difficult to find equivalent criteria for dementia. The general ICD-10 criteria for dementia are mainly based on memory deficits. But in some cases with microangiopathy (often called Binswanger's disease) the slowness of cognitive processes is the leading symptom [36, 53]. This example shows that the use of the dementia criteria is inefficient when looking for reduced mental capacity in patients with vascular lesions. Thus the question can be raised whether other symptom patterns, such as mental slowness and emotional reduction, should be added to the criteria for dementia.

Unequal distribution of cognitive deficits. A patchy distribution of cognitive deficits (i.e. involving some functions while sparing others) is judged as characteristic for VD [2, 79]. But the concept of VD has been criticized, because the simultaneous presence of multiple deficits in neuropsychological tests does not automatically allow the diagnosis of dementia [4, 9, 53]. Furthermore, it remains to be clarified how the measurement of an unequal distribution of cognitive deficits should be performed. In this study, an approach is made by using algorithms adapted from the SIDAM [80]. A similar method is suggested in the CAMDEX [62].

Evidence of focal brain damage. In some studies, the factors best discriminating MID from degenerative dementia are focal neurological signs and a history of stroke [25, 43, 61]. However, focal neurological signs or a history of stroke are absent in a remarkable percentage of cases with lacunar infarcts [5, 42, 75] and with leukoaraiosis [18, 38, 65, 66, 81]. Thus a dementia due to vascular changes may develop without sign of focal brain damage.

Evidence of a cerebrovascular disease. Vascular risk factors are commonly judged as indications of cerebrovacular diseases [48, 59], but they are unspecific and rather common in the elderly. Doppler sonography of the extracranial supply vessels reveals pathological lesions in only about 33% of VD cases, so that the diagnostic value is limited [51]. The value of transcranial Doppler sonography in the differentiation of VD and VD subtypes needs to be proved. So, apart from neurological signs, there are no safe indications of a cerebrovascular disease.

Compared with the classical 'ischaemic scales' [29, 44, 61], the ICD-10 criteria are highly selective. But they include items (especially the second and third listed above) that are characteristic of multi-focal vascular disorders, predominantly involving the cortex.

Only 66.6% of the cases that could be classified as VD using the ICD-10 criteria also fulfilled the criteria of a specific VD subtype. These criteria are rather miscellaneous: onset (acute VD), course (MID), location (MID and subcortical VD) and hypertension (subcortical VD). This concept of differentiating VD cases by using distinct kinds of items leads to an overlap in the classification of VD subtypes. Indeed, 33.3% could only be classified as mixed or undefined type using the ICD-10 criteria, suggesting VD is a multi-factorial disorder [64]. The ICD-10 criteria for VD subtypes will be discussed in detail as follows.

Rapid onset of dementia [VD of acute onset (F 01.0)]. In our sample there was no case with a rapid development of dementia within 3 months, but Tatemichi et al. [71] recently reported a high prevalence of dementia in a 3-month follow-up study of stroke survivors. These divergent results are probably due to the different composition of the samples. The patients in the present sample were admitted for the diagnostic evaluation of dementia, mostly not showing any indication of stroke within the last 6 months, while the other investigation [71] included only stroke survivors.

Gradual onset [MID (predominantly cortical) (F 01.1). Cognitive and neurological deficits accumulating in a stepwise manner are considered to be typical for MID [2, 79]. The ICD-10 charactize different VD subtypes on the basis of the onset and course of dementia. A gradual onset of dementia following a number of minor ischaemic episodes due to lacunae is judged to be an essential criterion for MID. Nevertheless, some lacunar cases (about 25% [42], 44.4% in this study) show a slowly progressive deterioration of cognitive impairment of cortical functions and neurological symptoms.

Hypertension. A history of hypertension is rather common in the elderly, especially in those suffering from a cerebrovascular disease. Nevertheless, in the ICD-10, hypertension is an essential criterion only for subcortical VD. But there are some well-documented reports of subcortical VD cases without hypertension [45].

Evidence of subcortical vascular lesions (on CT or MRI). Evidence of vascular lesions from neuroimaging is included as a criterion for the diagnosis of subcortical VD in the ICD-10. Furthermore, CT or MRI is recommended for diagnosing VD. In this study, all patients showed vascular lesions on CT. Nevertheless, only 24.6% fulfil the ICD-10 criteria of VD.

Vascular lesion on CT + dementia = VD?

In our sample the most frequent CT findings are subcortical vascular lesions. But in the elderly leuko-araiosis is a rather common finding, not automatically indicating dementia [27]. Thus the clinical value of leuko-araiosis is still a matter of debate [31, 60, 65, 66, 81]. Another point of issue is the aetiopathology of leuko-araiosis [3, 7, 20, 23, 27, 55, 60, 77, 78, 81].

Moreover, the volume of vascular lesions needed to cause a dementing decline is an ongoing matter of controversy, too [25, 44, 74]. In some studies, the extension of the white matter lesions correlates with the intellectual decline [55, 69], while in others [39] no connection could be demonstrated. In this study, no correlation between the severity of dementia and the (semiquantitatively estimated) extension of the vascular lesion could be found. However, cognitive deficits seem to correlate with cortical and ventricular atrophy rather than with the degree of leuko-araiosis [37]. This leads to the question, whether the intellectual decline is due to the degree of atrophy, to the extension of the vascular lesions, or to their location. However, recent PET [68] and SPECT [54] studies have demonstrated that even small subcortical vascular lesions may cause widespread functional sequelae exceeding the damaged areas visible on CT or MRI.

Furthermore, the evaluation of CT or MRI is not always conclusive. In about 30–65% of all lacunar strokes on lesion responsible for the symptoms is detected on CT or MRI [5, 42]; also, the diagnostic value of small high-signal intensities in the white matter on MRI remains unclear [3, 10, 11, 17, 37, 38, 62], as sometimes no histopathological correlate can be found [47]. Finally, periventricular hyperintensive rims on MRI, very frequently found in elderly patients, are unrelated to clinical events in most of the cases.

In the literature, it has been debated which type of vascular lesion most often leads to dementia [25, 40, 44, 52, 56, 64, 70]. The ICD-10 criteria give only a few aetiological clues. According to the CT criteria of Ringelstein et al. [56], in this study the most common type is microangiopathy, but 6 patients (9.8%) had VD of undefined type. The relatively high proportion of cases (33.3%) that could not be classified as a specific subtype of VD is probably due to the multi-factorial pathogenesis of VD [64]. In another survey [70], a plausible aetiological cause of VD could be found in only 51.7% of the cases.

Multi-factorial aetiopathology of dementia

It is evident that the presence of a stroke or vascular disease does not establish the aetiology of dementia as purely vascular, since Alzheimer-like dementia may coexist. In about 20% of all demented people the autopsy reveals such an overlap [1, 12, 34, 46, 49, 73, 74, 76]. In our study, only one patient (1.6%) met the clinical ICD-10 criteria of VD and DAT. But many cases (23%) which fulfil the DAT criteria present with CT findings showing a vascular lesion. Thus the classification becomes obscure. Moreover, so far there is still no agreement about the neuropathological differentiation of degenerative versus vascular dementia [12, 34, 72, 76]. But even if the brain ages independently of blood circulation, when vascular disease develops it becomes the pacemaker of the aging process of the brain [71].

To sum up, in spite of the present study being limited by the lack of neuropathological verification and by a sample mostly recruited from a psychiatric department without cases showing severe neurological symptomatology, it is considered to show the major problems of a clinical diagnostic approach to VD. However, the cases with a stroke and neurological symptoms do not raise diagnostic difficulties, while the very frequent cases showing leuko-araiosis on CT and revealing a general slowing of cognitive processes do. Although criteria for a subtype 'subcortical VD' are introduced in the ICD-10, a sufficient clinical differentiation of VD subtypes is hampered by the heterogeneous nature of the items. Furthermore, due to the lack of aetiopathological clues these criteria are considered to be of limited value for planning new, more specific therapeutic strategies for VD. Some attempts have been made to develop more detailed criteria for subtypes of VD [6, 21, 48, 67]. The elaboration of such aetiologically based criteria of VD subtypes will be a challenge in future dementia research.

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