Comparison of Different Clinical Criteria (DSM-III, ADDTC, ICD-10, NINDS-AIREN, DSM-IV) for the Diagnosis of Vascular Dementia

T. Pohjasvaara, MD, PhD; R. Mäntylä, MD; R. Ylikoski, MA; M. Kaste, MD, PhD; T. Erkinjuntti, MD, PhD

Background and Purpose—The criteria for vascular dementia (VaD) include definition of the cognitive syndrome and the vascular cause. Different criteria for dementia identify different frequencies and clusters of patients. In addition, variation in defining the cause and etiology may have an effect. We compared different clinical criteria for VaD in series of patients with poststroke dementia.

Methods—The study group comprised 107 patients fulfilling the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) definition for dementia from a cohort of consecutive patients with ischemic stroke who completed a comprehensive neuropsychological test battery and MRI. The mean age (SD) of the patients was 71.4 (7.6) years. The definitions of vascular cause of VaD were those of the DSM-III (1980), Alzheimer’s Disease Diagnostic and Treatment Centers (ADDTC; 1992), International Statistical Classification of Diseases, 10th Revision (ICD-10; 1992), National Institute of Neurological Disorders and Stroke–Association Internationale pour la Recherche et l’Enseignement en Neurosciences (NINDS-AIREN; 1993), and Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; 1994).

Results—The number of cases that could be classified as VaD according to the different criteria varied considerably: 36.4% (n=39) by DSM-III, 86.9% (n=93) by ADDTC, 32.7% (n=35) by NINDS-AIREN, 36.4% (n=39) by ICD-10, and 91.6% (n=98) by DSM-IV criteria. The concordance between DSM-III/ICD-10 was perfect (100%; κ=1.0), between ICD-10/NINDS-AIREN and ADDTC/DSM-IV good to moderate (85.0% and 87.3%; κ=0.87 and 0.37, respectively), but otherwise poor between the other criteria. Only 31 patients fulfilled all the criteria for VaD applied. Major discriminating factors between the criteria were requirement of (1) focal neurological signs, (2) unequal distribution of deficits in higher cortical functions, and (3) evidence of relevant CVD based on brain imaging findings.

Conclusions—Current criteria of VaD identify different frequencies and clusters of patients and are not interchangeable. Optimally, prospective studies with clinicopathological correlation could identify new criteria. Meanwhile, focus on more homogeneous subtypes (eg, small-vessel subcortical VaD) and detailed neuroimaging criteria could improve the diagnostics. (Stroke. 2000;31:2952-2957.)

Key Words: dementia ■ diagnosis ■ stroke

Critical elements in the concept and diagnosis of vascular dementia (VaD) incorporate defining the cognitive syndrome and the vascular cause. Recently, the effect of different criteria for defining the cognitive syndrome, the dementia syndrome, has been shown.1,2 Accordingly, different definitions determine different prevalence estimates and identify different groups of subjects. Furthermore, differences in defining the vascular cause and etiology may add to the variation.3–6 A limitation has been that all the clinical criteria applied are consensus criteria that are neither derived from prospective community-based studies with clinicopathological correlates of vascular factors affecting the cognition nor based on detailed natural histories.7 Currently, the most widely used criteria for VaD include the Diagnostic and Statistical Manual of Mental Disorders (DSM), Alzheimer’s Disease Diagnostic and Treatment Centers (ADDTC), International Statistical Classification of Diseases (ICD), and National Institute of Neurological Disorders and Stroke–Association Internationale pour la Recherche et l’Enseignement en Neurosciences (NINDS-AIREN) criteria.8–10

In the present study we studied the effect of different clinical definitions of VaD in case finding among patients with poststroke dementia.
Subjects and Methods

The Helsinki Stroke Aging Memory Study included 486 consecutive patients aged 55 to 85 years with ischemic stroke. Of these 486 patients, 337 (69.3%) completed MRI of the head and a comprehensive neuropsychological examination 3 months after stroke.1,11,12 The mean age (SD) of the patients was 71.4 (7.6) years. All the subjects underwent a structured medical and neurological history and a structured clinical and neurological examination.2 Cognitive domains assessed by the neuropsychological test battery included memory functions (short- and long-term memory), abstract thinking, judgment, aphasia, apraxia, agnosia, and constructional difficulty, including visuospatial and constructional functions.11 The criteria for dementia were those of the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III),13 and the diagnosis required impairment in short- or long-term memory and in one other cognitive domain, as well as a cognitive decline sufficiently severe to impair everyday social functioning, including the patient’s ability to work and perform activities of daily living. Assessment of both basic and complex activities of daily living, reflecting functions before and 3 months after the index stroke, was based on 5 structured interview schedules.3 Cognitive domains assessed by the neuropsychological test battery included memory functions (short- and long-term memory), abstract thinking, judgment, aphasia, apraxia, agnosia, and constructional difficulty, including visuospatial and constructional functions.11 The criteria for dementia were those of the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III),13 and the diagnosis required impairment in short- or long-term memory and in one other cognitive domain, as well as a cognitive decline sufficiently severe to impair everyday social functioning, including the patient’s ability to work and perform activities of daily living. Assessment of both basic and complex activities of daily living, reflecting functions before and 3 months after the index stroke, was based on 5 structured interview schedules.3 Cognitive domains assessed by the neuropsychological test battery included memory functions (short- and long-term memory), abstract thinking, judgment, aphasia, apraxia, agnosia, and constructional difficulty, including visuospatial and constructional functions.11 The criteria for dementia were those of the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III),13 and the diagnosis required impairment in short- or long-term memory and in one other cognitive domain, as well as a cognitive decline sufficiently severe to impair everyday social functioning, including the patient’s ability to work and perform activities of daily living. Assessment of both basic and complex activities of daily living, reflecting functions before and 3 months after the index stroke, was based on 5 structured interview schedules.3 Cognitive domains assessed by the neuropsychological test battery included memory functions (short- and long-term memory), abstract thinking, judgment, aphasia, apraxia, agnosia, and constructional difficulty, including visuospatial and constructional functions.11

Definitions of vascular cause of VaD applied included the DSM-III,13 ADDTC for probable ischemic VaD,14 International Statistical Classification of Diseases, 10th Revision (ICD-10),15 NINDS-AIREN for probable VaD,16 and Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV).17 Variables included in the 5 diagnostic guidelines applied are detailed in Tables 1 and 2.

Focal neurological signs in the DSM-III and DSM-IV definitions include exaggeration of deep tendon reflexes, extensor plantar response, pseudobulbar palsy, gait abnormalities, and weakness of an extremity; in the ICD-10 criteria, unilateral spastic weakness of the limbs, unilaterally increased tendon reflexes, an extensor plantar response, or pseudobulbar palsy; and in the NINDS-AIREN criteria, hemiparesis, lower facial weakness, Babinski sign, sensory deficit, hemianopia, and dysarthria. In the present study presence of focal neurological signs included at least 1 of the following: hemianopia, lower facial weakness, dysarthria, motor or sensory hemisindrome, hemiplegic gait, or positive Babinski sign.

“Patchy” distribution of deficits (ie, affecting some functions, but not others) in DSM-III and deficits in higher cognitive functions “unequally” distributed (with some function affected and others relatively spared) in ICD-10 were recorded positive in the present study if at least 1 of the cognitive domains assessed was rated as normal.

MRI was performed with a 1.0-T device (Siemens Magnetom).12 The number, size, and location of infarcts were recorded. The sites included lobes, vascular territories, and specific locations.12 Infarction was defined as lacunar if situated in the deep white or gray matter areas irrigated by the deep perforants and if the diameter was 3 to 9 mm.18 Large-vessel infarcts were those located in the cortical or cortico-subcortical areas. White matter lesions were rated in periventricular, deep, watershed, and subcortical white matter areas, as detailed previously.12,19 Extensive periventricular white matter lesions included extending caps (hyperintensities classified on the basis of size and shape), or irregular halo, or diffusely confluent lesions, or extensive white matter change, or a combination thereof.20 Reliability of the visual rating was tested by reviewing 60 MR scans independently by the same rater (R.M.), by a board-certified neuroradiologist, and by a general radiologist. The weighted $k$ values for intraobserver agreement were 0.90 for periventricular caps, 0.93 for linings and halos, and 0.95 for deep white matter hyperintensities. The corresponding $k$ values for interobserver agreement were 0.82 to 0.84 for caps, 0.72 to 0.82 for linings and halos, and 0.77 to 0.84 for deep white matter hyperintensities.12 Clinically, all the patients were examined by the same neurologist (T.P.), and all the cases were reviewed together with the senior neurologist (T.E.).

The study was approved by the ethics committee of the Department of Clinical Neurosciences, Helsinki University Central Hospital, Helsinki, Finland. The study design was first explained fully; written information was offered to the patients and a knowledgeable informant, and if they agreed to participate, a written consent form was signed by a patient or a knowledgeable informant if the patient was obviously demented or unable in any other way to sign a consent form.

In the statistical procedure we compared the effect of the definition of vascular cause on the 107 patients with a diagnosis of DSM-III dementia. The $\chi^2$ test was applied for categorical data comparing patients diagnosed with VaD by different paradigms. Concordance (overlapping cases) was computed as the quotient of the cases classified as VaD by both of the criteria applied and the number of cases classified as VaD by either of the criteria. Agreement between the criteria was also calculated with the use of $\kappa$ statistics, indicating how much better the agreement is than that of chance, with a value of zero indicating no agreement better than chance. The statistics were analyzed with the BMDP program.21

Results

The frequency of patients fulfilling definitions for vascular cause varied considerably when algorithms for different
<table>
<thead>
<tr>
<th>Diagnosis of Vascular Cause of VaD According to DSM-III, ADDCT, ICD-10, NINDS-AIREN, and DSM-IV Criteria in Patients With DSM-III Poststroke Dementia (n=107) in the Helsinki Stroke Aging Memory Study Cohort</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No.</strong></td>
</tr>
<tr>
<td><strong>DSM-III</strong></td>
</tr>
<tr>
<td>Stepwise deterioration, and with “patchy” distribution of cognitive deficits and focal neurological signs and symptoms and evidence from history, physical examination, or laboratory tests of significant CVD that is judged to be etiologically related to the disturbance</td>
</tr>
<tr>
<td>VaD according to DSM-III criteria</td>
</tr>
<tr>
<td><strong>ADDCT</strong></td>
</tr>
<tr>
<td>Evidence of ≥2 ischemic strokes by history, or neurological signs, and/or neuroimaging studies (CT or T1-weighted MRI), or occurrence of a single stroke with a clearly documented temporal relationship to the onset of dementia and evidence of ≥1 infarct outside the cerebellum by CT or T1-weighted MRI</td>
</tr>
<tr>
<td>VaD according to ADDTC criteria</td>
</tr>
<tr>
<td><strong>ICD-10</strong></td>
</tr>
<tr>
<td>Deficits in higher cognitive functions are unequally distributed and clinical evidence of focal brain damage (signs) and evidence from the history, examination, or tests of a significant CVD, which may reasonably be judged to be etiologically related to the dementia (e.g., a history of stroke, evidence of cerebral infarction)</td>
</tr>
<tr>
<td>VaD according to ICD-10 criteria</td>
</tr>
<tr>
<td><strong>NINDS-AIREN</strong></td>
</tr>
<tr>
<td>CVD defined by the presence of focal signs on neurological examination and evidence of relevant cerebrovascular disease by brain imaging (MRI) including multiple large-vessel infarcts or single strategically placed infarct (angular gyrus, thalamus, basal forebrain, PCA or ACA territories) or multiple basal ganglia and white matter lacunes or extensive periventricular white matter lesions or combinations thereof A relationship between the above 2 disorders manifested or inferred by the presence of ≥1 of the following Onset of dementia within 3 mo after a recognized stroke Abrupt deterioration in cognitive functions, or fluctuating, stepwise progression of cognitive deficits</td>
</tr>
<tr>
<td>VaD according to NINDS-AIREN criteria</td>
</tr>
<tr>
<td><strong>DSM-IV</strong></td>
</tr>
<tr>
<td>Focal neurological signs and symptoms or laboratory evidence indicative of CVD that are judged to be etiologically related to the disturbance</td>
</tr>
<tr>
<td>VaD according to DSM-IV criteria</td>
</tr>
</tbody>
</table>

PCA indicates posterior cerebral artery; ACA, anterior cerebral artery.
definitions of VaD were used among the 107 patients fulfilling the DSM-III criteria for dementia: 36.4% (n=39) by DSM-III, 86.9% (n=93) by ADDTC, 32.7% (n=35) by NINDS-AIREN, 36.4% (n=39) by ICD-10, and 91.6% (n=98) by DSM-IV criteria (Tables 1 and 2).

The DSM-III and the ICD-10 criteria for VaD had a concordance of 100%. The requirements of these criteria are detailed in Table 2. The ADDTC criteria for the diagnosis of probable ischemic VaD and the NINDS-AIREN criteria for probable VaD are also shown in Table 2, as are the DSM-IV criteria. A total of 5 subjects did not fulfill the definition of vascular cause for VaD according to any of the 5 diagnostic criteria, and only 31 subjects were diagnosed by all 5 criteria (Figure, Table 3).

The concordance (percentage of overlapping cases) between the definitions varied (Table 4): it was excellent between DSM-III and ICD-10 criteria (100%), good between ICD-10 and NINDS-AIREN criteria (85.0%) and between DSM-IV and ADDTC criteria (87.3%), but poor between the other criteria (<40%). Agreement between the guidelines was also calculated by the κ statistic, which indicates how much better the agreement is than by chance. The agreement was excellent between DSM-III and ICD-10 criteria (κ=1.0), good between ICD-10 and NINDS-AIREN criteria (κ=0.87), and fair or poor between the other criteria, being at the level of chance between the ICD-10 and ADDTC criteria (κ=−0.03) (Table 4).

Focal neurological signs required to be present in the DSM-III, ICD-10, and NINDS-AIREN criteria were recorded in 40 patients (37.4%). In the present series, presence of focal signs was the main discriminating factor between these 3 criteria and ADDTC or DSM-IV criteria (Table 4). Further important discriminating factors included requirement of unequal distribution of deficits in higher cognitive functions and evidence of relevant CVD based on brain imaging findings (Table 4).

**Discussion**

We evaluated the effect of different definitions of vascular cause in current clinical criteria for VaD in a series of 107 patients with DSM-III poststroke dementia. Different definitions for VaD gave different frequency estimates, and overlap in the cases diagnosed was considerable, except between the ICD-10 and DSM-III criteria, where it was perfect. The origins of these differences include the following: (1) requirement of focal neurological signs and symptoms to be present in DSM-III, ICD-10, and NINDS-AIREN criteria; (2) absence of brain imaging requirements of relevant cerebrovascular disease (CVD) in DSM-III, ICD-10, and DSM-IV criteria; and (3) requirement of patchy or unequal distribution of higher cognitive functions in DSM-III and ICD-10 criteria. Additional factors include (4) qualifying extensive white matter lesions as radiological evidence of relevant CVD in NINDS-AIREN but not in ADDTC criteria and (5) requiring one CT or T1-weighted MRI infarct outside the cerebellum in ADDTC criteria.

The DSM-III and ICD-10 criteria had a concordance of 100%. Neither of these guidelines specifies brain imaging requirements. They require evidence from history, physical examination, or laboratory tests of significant CVD that is judged to be etiologically related to the disturbance. They also require focal neurological signs and symptoms on neurological examination to be present and require unequal distribution of cognitive deficits. We did not study possible ICD-10 subtypes of VaD in the present study.22

The agreement between NINDS-AIREN and ICD-10 was good (concordance, 85%; weighted κ=0.87), as well. They both require focal neurological signs to be present, which were infrequent in the present patients with a history of ischemic stroke. The main origin of difference between these 2 criteria is requirement of unequal distribution of deficits in higher cognitive functions by ICD-10 and detailed radiological criteria by NINDS-AIREN.

**TABLE 3. Agreement on the Diagnosis of a Vascular Cause of Dementia According to Various Clinical Criteria in Patients With DSM-III Poststroke Dementia (n=107) in the Helsinki Stroke Aging Memory Study Cohort**

<table>
<thead>
<tr>
<th>No Vascular Cause</th>
<th>NINDS-AIREN</th>
<th>DSM-IV</th>
<th>DSM-III/ICD-10</th>
<th>ADDTC</th>
</tr>
</thead>
<tbody>
<tr>
<td>No vascular cause</td>
<td>5</td>
<td>72</td>
<td>9</td>
<td>68</td>
</tr>
<tr>
<td>NINDS-AIREN</td>
<td>72</td>
<td>35</td>
<td>35</td>
<td>34</td>
</tr>
<tr>
<td>DSM-IV</td>
<td>9</td>
<td>35</td>
<td>98</td>
<td>39</td>
</tr>
<tr>
<td>DSM-III/ICD-10</td>
<td>68</td>
<td>34</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>ADDTC</td>
<td>14</td>
<td>32</td>
<td>89</td>
<td>33</td>
</tr>
</tbody>
</table>
In addition to the NINDS-AIREN criteria, the other criteria defining the radiological findings are the ADDTC criteria, which require at least 1 infarct outside the cerebellum detected on CT or T1-weighted MRI, but white matter lesions do not qualify for support of probable ischemic VaD. The NINDS-AIREN criteria require focal signs to always be present, and the ADDTC criteria require evidence of ≥2 ischemic strokes by history, neurological signs, and/or neuroimaging studies (CT or T1-weighted MRI); accordingly, the agreement between these 2 criteria was poor (concordance, 33%; weighted $\kappa=0.05$). As evaluated neuropathologically, the ADDTC criteria seem to be more sensitive and the NINDS-AIREN criteria more specific, but neither is perfect.23

The DSM-IV criteria were the most liberal; a total of 98 subjects (91.6%) fulfilled these criteria. The DSM-IV criteria do not require focal neurological signs and symptoms to be present and do not specify brain imaging criteria clearly. In a recent study of 25 demented subjects, the DSM-IV criteria showed the best overall agreement with other criteria for VaD ($\kappa$ range, 0.32 to 0.60) and gave the greatest overlap with the combined ADDTC for probable and possible ischemic VaD.6

In the present study the agreement between DSM-IV and the other criteria varied to a greater extent (concordance, 33% to 87%; weighted $\kappa$ range, 0.08 to 0.37), which related to the frequency of focal neurological signs.

In the present cohort, in patients with ischemic stroke, only 40 patients (37.4%) showed focal signs on neurological examination (hemianopia, lower facial weakness, dysarthria, motor or sensory hemisindrome, hemiplegic gait, or positive Babinski sign) 3 months after stroke. In accordance with our findings, the small percentage of cases classified as VaD depended on the small number of subjects showing focal signs on the neurological examination in the series of Wetterling et al.5 In particular, patients with small-vessel subcortical VaD frequently do not show clear-cut focal signs. Thus, neuroimaging criteria could increase sensitivity and specificity in case finding,7,24 as suggested in the recent research criteria for subcortical small-vessel VaD.20

In the DSM-III, ICD-10, and DSM-IV criteria, no clear specification of an underlying vascular process is given; the ADDTC criteria are limited to ischemic brain injury, and the NINDS-AIREN criteria compile a description of many possible etiologies of VaD. Thus, it is not surprising that the concepts underlying the definitions of vascular cause are rather heterogeneous, with agreement at the level of chance (between the ADDTC and ICD-10 criteria, $\kappa=-0.03$).

In conclusion, the clinical criteria for VaD are not interchangeable. Despite a degree of overall similarity, the case finding will vary significantly depending on the criteria. Our study thus strengthens the earlier findings in a large, well-defined stroke cohort.4,5 Furthermore, we focused only on the vascular cause of dementia in patients already found to be demented. At present, the lack of comparability between diagnostic criteria is a barrier to research and clinical care, and further debate based on these and earlier findings is needed. The differences we found will influence not only estimates of prevalence and incidence of VaD but also clinical recognition and treatment of the condition. Ideally, in constructing new criteria the component parts of the guidelines should be tested with prospective longitudinal studies with clinicopathological correlation.25 Meanwhile, focus on more homogeneous subtypes of VaD (eg, small-vessel subcortical VaD) and on uniform reproducible imaging criteria may be a solution.7,20

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References


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