Accuracy of Bedside Diagnosis in Stroke

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SUMMARY The clinical diagnosis of the type of acute cerebrovascular diseases is often considered unreliable, although this has not been validated prospectively in representative patients. The accuracy of bedside diagnostics was, therefore, tested in 206 patients consecutively admitted to the Stroke Unit of the Serafimerlasarettet in Stockholm. Bedside diagnosis turned out to be correct in 69%. In 24% the diagnoses were altered after hospital investigation and in the remaining 7% no defined preliminary and/or final diagnosis could be made. When the diagnoses were considered “fairly certain” they were accurate in 87%, compared to 53% when regarded as only “probable”. The diagnostic accuracy improved during the period studied. Sensitivity in identifying hemorrhages was much lower (39%) than for cerebral infarctions (83%). It is suggested that new investigational methods should be compared with what can be accomplished with bedside methods alone.

SPECIFIC ACUTE and secondary preventive therapy has become available for some forms of cerebrovascular diseases (CBVD). It is, therefore, important not only to differentiate between hemorrhagic and ischemic disorders but also to separate the subgroups within these entities.

Many qualified methods for investigating stroke have, accordingly, come into use. Some are associated with great costs and require equipment not available in all hospitals or for all patients. The benefit of such specific investigations for the aged patient has also been questioned. Although many authors support the value of new diagnostic examinations with the observation that bedside diagnosis is not reliable in CBVD, there is no prospective validation of this idea in a living patient population. Therefore, we have evaluated the accuracy of diagnoses at the bedside made on clinical grounds alone. The present study, over 2 years, was aimed at answering the following questions:

1) How often can we correctly identify the type of stroke with bedside methods only?
2) Are the bedside diagnoses more often correct when initially considered “certain” than when considered “probable”?
3) Which clinical factors guided our preliminary judgement and which factors were involved in the final diagnoses?
4) Did our diagnostic ability improve during the two year study period in the Stroke Unit?

Patients and Methods

Serafimerlasarettet is a university hospital of the Karolinska Institute. It serves a defined population of 120,000 inhabitants in greater Stockholm. Two hundred and six patients (97 men and 109 women, mean age 73 years) were consecutively admitted from the Emergency Ward to the Stroke Unit of the Medical Department during the period October 1976 to July 1978. Criteria for admission to the Stroke Unit were: acute to subacute appearance of focal neurological deficit, either as transitory ischemic attacks (TIA) within the last month or as persistent symptoms within the last week. Doctors on duty were informed about the study and checked that the patients fulfilled these criteria. The patients were then sent to the Stroke Unit if there was a bed available. The admission procedure has been described and evaluated earlier as well as the general organization of the Stroke Unit.

Bedside Diagnoses

The patients arrived in the Stroke Unit with a mean delay of 16 hours after the onset of symptoms. A bedside diagnosis was made and irretrievably designated...
within the first 24 hours after the patients' arrival. At least one senior, clinically experienced internist and one junior doctor, both members of the CBVD research group, participated. The diagnosis was based solely on the patient's history and physical findings. Working from the International Classification of Diseases, \(^8\) the following diagnostic alternatives were used: subarachnoid hemorrhage, cerebral hemorrhage, occlusion of precerebral arteries, cerebral thrombosis, cerebral embolism, transient cerebral ischemia (TIA) and acute but ill-defined cerebrovascular disease.

A record was also kept of whether the physicians considered the bedside diagnosis "fairly certain" or only "probable". The study did not deal with the clinical ability to localize or estimate the size of the different lesions.

**Final Diagnoses**

After discharge of the patient a final diagnosis was made at a concluding discussion of the case within the CBVD research group. The minimum diagnostic requirements were:

Cerebral hemorrhage: macroscopically hemorrhagic cerebrospinal fluid or bleeding pattern when spectrophotometry was performed.

Cerebral thrombosis: patients with findings in accordance with an ischemic lesion when spectrophotometry was performed.

Cerebral embolism: sudden onset of symptoms in patients with an embolic source (rheumatic heart disease, atrial fibrillation, acute myocardial infarction or angiographically verified ulceration in the relevant carotid artery) and findings at spectrophotometry in accordance with an ischemic lesion.

TIA: focal neurological deficit with a duration of less than 24 hours.

Acute but ill-defined cerebrovascular disease: patients with CBVD not fulfilling the above criteria.

The examinations which served as a basis for final diagnoses are presented in table 1. Spinal fluid analysis, skull x-ray, echoencephalography and brain scan were done in almost every patient. In about half cerebral angiography, computed tomography (CT-scan), or, in the deceased, autopsy, were also carried out. In 7 patients 2 of these investigations were done.

To check the reliability of the final diagnoses, Kyushu University scores for differential diagnosis in stroke, recommended by WHO, were applied retrospectively. \(^7\) Certain parameters (sex, age, history of previous stroke, blood pressure, consciousness level, conjugated eye deviation, anisocoria, speech disorder, neck stiffness, motor- and sensory deficit, xanthochromia of the cerebrospinal fluid) are scored differently for cerebral infarction and hemorrhage respectively. This yields 2 sums, one for infarction and the other for hemorrhage, and the higher of these indicates the most likely diagnosis. This score confirmed our final diagnosis in 92% of the patients where values for cerebral infarction were higher than for hemorrhage (table 2). As can be seen, the consistency in patients with the diagnosis of cerebral infarction based on CT-scan, angiography or autopsy, was similar to results without these examinations.

**Validation Procedure and Statistical Methods**

A validation measurement based on comparison between bedside diagnosis and final diagnosis was computed. The validity of bedside diagnosis is expressed in terms of sensitivity, specificity and confirmation rate. Sensitivity is defined as the ability of the diagnostic method to find true positives, specificity as the ability to find true negatives. \(^8\)

Occurrence of certain factors in patient history and physical examination, earlier described as important for differential diagnosis, \(^9\) \(^11\) was calculated for each diagnostic group. Differences between the groups were
analysed by the chi-square test. Significance levels considered were 5%, 1% and 0.1%. A cluster analysis was performed of the factors which proved to discriminate best between hemorrhage and infarction.

**Results**

**Agreement Between Bedside and Final Diagnoses**

Table 3 shows the relation between bedside and final diagnosis in the 206 patients. Bedside diagnosis was correct in 69% of the total. Correctness was similar in the patient group with diagnosis based on spinal fluid analysis and brain scan (71%) as in the group where CT-scan, angiography or in the deceased autopsy, had also been performed (68%). In 24% the diagnosis was changed after hospital investigation. For the remaining 7% of patients it was not possible to make a preliminary diagnosis (9 patients), a final diagnosis (4 patients) or either (one patient).

Twenty patients were suspected of having a cerebral hemorrhage, but this was confirmed in only 6. An additional 12 were found. Of all 18 patients with hemorrhage one was identified with CT-scan (3% of all CT-scans). Two others were found by angiography (10% of all performed angiographies). Twenty-one percent of all autopsies (8 cases) showed hemorrhages. In the remaining 7 patients the diagnoses were based on spinal fluid analyses and brain scan.

In 145 patients cerebral infarction (thrombosis and embolism) was thought to be present; this was confirmed in 82%. Table 3 also shows that for 12 patients the diagnosis was altered from one category of infarction to the other. In the bedside diagnoses of TIA, 72% were correct; in a few patients, symptoms that were presumed likely to pass within 24 hours actually did not. None of the diagnostic sub-groups was consistently more over- than under-diagnosed nor was the opposite true.

Of 6 patients (here called A, B, C, D, E, F), 3% proved not to have cerebrovascular disease. In 2 of them (A, B), one with a suspected cerebral hemorrhage and one with a clinical picture of cerebral thrombosis, autopsy revealed acute myocardial infarction. In one patient (C) with a suspected hemorrhage, spinal fluid analysis was inconclusive, with a slight rise of protein, white and red corpuscles, and a somewhat low glucose level. Since meningitis could not be excluded, the patient was treated with antibiotics but died 24 hours after arrival. Autopsy and bacterial cultures showed listeria meningitis. In 2 other patients the true diagnosis was revealed by further clinical observation. One of them (D) developed several epileptic attacks with subsequent post-ictal neurological deficit of the type which had initially prompted the suspicion of a stroke. The other patient (E) was included due to anisocoria and facial palsy; the pupil abnormality had probably existed for many years and the paresis was of the peripheral type. In the last patient (F), with a typical TIA, a meningioma was found.

Table 4 shows validity data regarding the clinical ability to separate patients with hemorrhagic disorders from those with ischemic lesions. Only 32% of the few hemorrhages were suspected at the bedside, compared to 88% of the infarctions.

**Impact of Certainty of Bedside Diagnoses**

The degree of certainty with which the bedside diagnosis was made was related to its correctness (table 5). Thus, 87% were correct when considered "fairly certain," compared to 53% when regarded only
TABLE 5  Correctness of the Different Bedside Diagnoses in 196 Patients. Correctness is Expressed in Relation to the Degree of Certainty with which the Diagnoses Were Made. Ten Patients, Where no Specified Bedside Diagnosis Could be Made, are Excluded.

<table>
<thead>
<tr>
<th>Bedside diagnosis</th>
<th>Total No.</th>
<th>Total % correct</th>
<th>Fairly certain n = 115 % correct</th>
<th>Probable n = 81 % correct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>2</td>
<td>0</td>
<td>—</td>
<td>0</td>
</tr>
<tr>
<td>Cerebral hemorrhage</td>
<td>20</td>
<td>30</td>
<td>50</td>
<td>21</td>
</tr>
<tr>
<td>Cerebral thrombosis</td>
<td>108</td>
<td>82</td>
<td>97</td>
<td>64</td>
</tr>
<tr>
<td>Cerebral embolism</td>
<td>37</td>
<td>73</td>
<td>82</td>
<td>44</td>
</tr>
<tr>
<td>TIA</td>
<td>29</td>
<td>72</td>
<td>75</td>
<td>67</td>
</tr>
<tr>
<td>Total</td>
<td>196</td>
<td>73</td>
<td>87</td>
<td>53</td>
</tr>
</tbody>
</table>

"probable" (p < 0.001). The accuracy within each of the diagnostic groups is also given in the table. Whereas “certain” diagnoses of cerebral hemorrhage were correct in only 50% of cases, a thrombotic lesion, suspected with fair certainty, was confirmed in 97%.

Test of Clinical Factors

Factors previously described as important for differential diagnosis between cerebral hemorrhage and infarction were investigated in relation to bedside as well as to final diagnosis. We could not find any factors which, alone or combined, were of absolute importance in determining our bedside diagnosis. In relation to final diagnosis, the only statistically significant findings in the 2 groups are presented in table 6. Information from the patient’s history (earlier hypertension, diabetes, hyperlipemia, smoking, heart failure, ischemic heart disease, way of falling ill and activity at onset of symptoms) appeared with the same frequency in both groups. So did physical findings (heart rate, blood pressure levels and neurological deficit) apart from those presented in the table. The cluster analysis did not reveal any combination of factors which significantly improved the differentiation between the groups.

Effect of Experience

The material was divided retrospectively into 3 consecutive, equally large, parts. During the first 2 periods the accuracy of the bedside diagnoses was 61% and constant. In the last period it improved to 87% (p < 0.001). A more detailed analysis showed there was a rise in confirmation rate as well as specificity as regards all the diagnostic sub-groups. Sensitivity also improved, but not for hemorrhages.

Discussion

Most earlier studies on the reliability of clinical diagnosis of CBVD have used autopsy findings as a reference. Heasman and Lipworth stated on this basis that hemorrhages were clinically overdiagnosed in 43% of patients and that the whole group of cerebrovascular diseases was overestimated. Similar experiences have been reported by others. In the present study the diagnostic accuracy was better than in these reports and hemorrhages were not overdiagnosed at the expense of infarction. This is probably explained in part by the growth of knowledge about the distribution of diagnoses. Furthermore, findings from autopsy series clearly differ from the present in several respects: the frequency of hemorrhages is much higher among deceased patients than in a clinical series. In addition questioning and clinical examination of the most severely ill patients is hampered by their poor condition. The clinical diagnosis might, therefore, be erroneous more often in this group.

In a study on CT-scan in CBVD, Kinkel and Jacobs compared final diagnoses and preliminary judgments and found the latter were correct in 69%. However, the diagnostic distribution and low mortality are not in accordance with recent clinical series, nor is the low sensitivity of spinal fluid analysis in hemorrhage or exclusively normal findings on CT in TIA. This indicates that the study could be retrospective on selected cases. A comparison with our results would, therefore, not be adequate.

In the present study, bedside diagnoses were made

**Table 6**  Occurrence of Some Factors in Cerebral Hemorrhage and Infarction Respectively in 172 Cases of Manifest and Well-defined Stroke.

<table>
<thead>
<tr>
<th>Physical findings on the 1st day after admittance to the Stroke Unit</th>
<th>Final diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cerebral hemorrhage, n = 18</td>
</tr>
<tr>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Impaired consciousness</td>
<td>13</td>
</tr>
<tr>
<td>Presence of neck stiffness</td>
<td>5</td>
</tr>
<tr>
<td>Patient cannot walk independently or with mechanical aid</td>
<td>17</td>
</tr>
</tbody>
</table>
on unselected patients consecutively admitted from an emergency department to a stroke unit. These patients were representative of all patients with stroke arriving in the hospital. The bedside diagnosis was then compared with the final diagnosis made after thorough clinical investigation, examinations described earlier and the summing-up discussion in each patient. This should provide for high reliability of the final diagnosis. A similar consistency existed between our diagnoses and the Kyushu diagnostic score both when CT-scan, angiography and, in the deceased patients, autopsy was done, and when none of these examinations was performed. This indicates that the final diagnoses were probably highly reliable. The diagnostic distribution is in accordance with that of other recent clinical series.

The bedside diagnosis thus was correct in 69% of patients and similar in the 2 groups with a different basis for final diagnosis. For ischemic lesions, the sensitivity and confirmation rate were very high. Diagnoses sometimes were changed from one entity to another within the group. For example, ECG revealed a clinically unnoticed atrial fibrillation in 4 patients with slow and regular ventricular rates. In keeping with our definitions, the diagnosis was then altered from cerebral thrombosis to embolism if the onset of symptoms had been sudden. This definition is being debated but is generally accepted. In the fifth and sixth patients alteration of diagnosis was due to findings at angiography and autopsy respectively.

For the hemorrhagic lesions, sensitivity was 39% and even when the diagnosis was considered fairly certain the confirmation rate was only 50%. In the early part of the study we were inclined strictly to follow what has been described as a characteristic clinical picture, which led to a tendency to over-diagnose cerebral hemorrhage. After a preliminary survey of the first two-thirds of the material we changed our policy and required much stronger evidence before this diagnosis was made. In the first period we thought 19 patients suffered from cerebral hemorrhage, but this was confirmed in only 5. During the last third of the study we presumed hemorrhage in only one case, which was confirmed. But we failed to diagnose hemorrhage in 3 patients.

In this group 6 patients turned out not to have CBVD, which indicates that patients with apparent CBVD may have entirely different conditions. The frequency and type of such diagnostic errors are in accordance with findings in other studies. Two of the patients were diagnosed correctly on a clinical basis (peripheral facial palsy, epilepsy). The 2 patients where autopsy revealed acute myocardial infarction had non-informative results from the special neurological investigations. These studies revealed the true diagnosis in only 2 of the 6 misdiagnosed patients who had a meningioma and meningitis.

A purpose of the study was to find a combination of clinical factors which would discriminate between hemorrhage and infarction with statistical significance. Impaired consciousness, neck stiffness and inability to walk independently occurred more often in patients with hemorrhage, but no factors or combination of findings were truly discriminatory for hemorrhage. Thus, most differences between the clinical characteristics are quantitative rather than qualitative and not really diagnostic.

A non-selected group of CBVD patients is overwhelmingly dominated by infarction. It is, therefore, of theoretical interest to calculate the validity data assuming a constant diagnosis of cerebral infarction. The confirmation rate for infarctions in this group would then be 75% and the sensitivity, accordingly, 100%. On the other hand, the specificity (the ability of the method to identify true negatives) would be nil. Furthermore, the sensitivity for hemorrhages would also be nil. With present therapeutic alternatives, the main requirements for a diagnostic method are high sensitivity for hemorrhages and high specificity for infarctions. In these respects the bedside method proved much superior to the "constant guess of infarction" method.

Bedside diagnosis seems valid and acceptable in some situations without further confirmation, especially if the diagnosis was made with fair certainty. Further clinical investigations augment the ability to find the few patients with cerebral hemorrhages. For patients where therapy with anticoagulant or platelet-aggregation inhibitors is considered, bleeding must be ruled out with additional certainty. When evaluating diagnostic methods used for this purpose it is suggested that the results should be compared with what can be achieved with bedside methods alone.

Conclusions

1) The initial bedside diagnosis of specific CBVD proved correct in 69% of patients. In 24% they were altered after hospital investigation. For 7% of the patients no defined preliminary and/or final diagnosis could be made.

2) When the diagnoses were considered fairly certain, they were accurate in 87%, compared with 53% when they were regarded as only probable.

3) Impaired consciousness, neck stiffness and inability to walk independently occurred more often in patients with hemorrhage than in those with infarction. However, because of the diagnostic distribution, these differences are quantitative rather than qualitative and not truly diagnostic.

4) Correctness of the bedside diagnoses increased from 61% during the first 2 periods of the study to 87% during the last.

Acknowledgment

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References

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A MULTICENTER STUDY of Reversible Ischemic Attacks has been carried out in 8 Italian neurological and neurosurgical centers as part of the Italian National Research Council, Special Preventive Medicine Project for Atherosclerosis. Four hundred and sixty consecutive patients have been entered into the study. These were evaluated by standardized neurological, cardiological, laboratory and neurovascular procedures. All patients had cerebral angiography, with multiple vessel visualization through femoral catheterization.

Recruitment of patients took place between 1977 and 1978 and the 3 year follow up will end in 1981. The aim of the study is to determine the present status of the disease in Italy and its relation to the main risk factors, the type of clinical events, modification of cardiovascular state and evolution of the angiographic picture.

Data available to date were presented in Rome, Oct. 14-16, 1980, at a 3-day conference devoted to a review of atherosclerotic vascular disease. This conference covered reports of other aspects of the Special Preventive Medicine Project for Atherosclerosis, including multicenter studies on pathological aspects of atherosclerosis, coordinated by Prof. G. Baroldi; and on myocardial ischemia, by Prof. A. Maseri, professor of cardiology of the Postgraduate Medical School, Hammersmith Hospital, London.

The conference included lectures given by invited guests. Among them were “Clinical pathology and risk factors of TIAs,” A. Bes (Toulouse); “Tissue metabolism in TIAs and in ischemia,” J.M. Fein (New York); “Contribution of CBF studies to diagnosis and prevention of cerebral ischemia,” J.M. Orgogozo (Bordeaux); “Metabolic aspects of cerebrovascular disorders studied with positron tomography,” G.L. Lenzi, (Rome); “Natural history and causes of death in TIAs,” J.F. Toole (Winston-Salem, NC); “Cardiac dysfunction and brain ischemia,” S. Lavy (Jerusalem); “Contribution of angiography in preventive studies, risks and complications,” O.M. Reinmuth (Pittsburgh, PA); “Brain hemorrhage: present status of diagnosis, prevention and treatment,” J.T. Robertson (Memphis, TN); “Clinical management of TIA patients,” A. Heyman (Durham, NC); “Medical preventive trials in cerebral ischemia,” J.P. Whisnant (Rochester, MN); “Surgical preventive trials in cerebral ischemia,” P. Conforti (Naples).

The results of the Italian cooperative study on reversible ischemic attacks were presented by C. Fieschi (Rome) and others.

Dr. De Zanche (Padua) described the population analyses. The average age at time of entry was 49 with 51% of the cases below 55. The present study differs from others which included primarily older patients. Sixty-four percent had 2 or more ischemic attacks at the time of study. Fifty-six percent of the attacks lasted longer than 24 hours. Identification of the arterial territory involved was determined clinically by the participating authors: 60% were carotid attacks.

Italian Study of Reversible Ischemic Attacks

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