Upsala J Med Sci 93: 63–69, 1988

Discrepances between CT and EEG Findings after Acute Cerebrovascular Disease

Martin Knibestöl, Erik Hägg and Bengt Liliequist

Departments of Clinical Neurophysiology, Internal Medicine and Neuroradiology, University Hospital, Umeå, Sweden

ABSTRACT

Combined EEG and brain CT examinations were performed in 33 patients during the 1st and 2nd week after stroke. CT was abnormal in 17 patients (51%) and EEG was abnormal in 24 patients (72%). In 17 patients CT and EEG showed conflicting results; in 5 patients with normal EEG findings CT was pathological, and 12 patients had normal CT but pathological EEG findings. In this latter group, there were 5 particularly interesting cases with normal CT and a prominent unilateral EEG abnormality. Recently patients with this combination of findings have been described where further investigations disclosed internal carotid occlusion, which could be treated surgically. It is suggested that EEG should be more extensively used when CT findings are negative after stroke, and if a major unilateral EEG abnormality is encountered in such cases, further investigations with angiography should be considered in order to exclude surgically treatable internal carotid occlusion.

INTRODUCTION

In some recent studies (3, 9, 10, 11, 12) the relative usefulness of computerized tomography (CT) and electroencephalography (EEG) in investigation of patients with acute cerebrovascular disease (CVD) has been discussed. There is general agreement that both methods are useful and may complement each other: CT gives information on the extent and localization of a lesion and can differentiate between cerebral infarction and hemorrhage, whereas EEG reflects mainly the functional state of the brain and correlates better with metabolic and circulatory function. CT is superior for diagnostic purposes, whereas EEG may have greater value for prognostic information concerning survival and neurological recovery (6).

It is important to point out, however, that both CT and EEG can be normal in a substantial number of CVD cases, depending on such factors as size and localization of a lesion and its nature (TIA, infarction or hemorrhage). Furthermore, CT and EEG may not necessarily both be pathological in the same patient. Recently Yanagihara et al. (15) described in detail three interesting cases with a prominent unilateral EEG abnormality but normal CT findings, and on angiography these patients proved to have occlusion of the carotid system which could be treated surgically with subsequent clinical improvement and normalization of EEG. They noticed that the frequency with which

discrepancy between CT and EEG occurs is not known, but suggested that the number could be considerable, and they emphasized that recognition of this kind of discrepancy may help to establish the diagnosis by directing attention to appropriate neurovascular evaluation and treatment. In the present study we performed combined CT and EEG examinations in a series of CVD patients, with particular attention focused on the frequency of major discrepancies between the two methods, including the type described by Yanagihara et al. (15).

MATERIAL AND METHODS

The clinical material consisted of 33 consecutive patients aged 45-87 years (mean 64), admitted to the Stroke Unit at the Medical Clinic, Umeå University Hospital, during a six month period for acute CVD. The patients were included in the study irrespective of the initial severity or course of the disease. Four patients were classified as having TIA, whereas 29 patients developed completed stroke with neurological deficits of more than 24 hours' duration. Of the latter, a definite diagnosis of cerebral infarction or hemorrhage could be established (by typical CT findings) in 11 respectively 3 patients, whereas the remaining 15 were considered to have an infarction (symptom duration more than 24 hours, lack of bleeding on CT and CSF spectrophotometry).

Every patient in the stroke unit was subjected to a standardized scheme of investigation. Repeated clinical assessments were performed at admission, after one and four days and at discharge. All data were registered in a protocol for computer use. The patients usually kept their previous medication (mostly antihypertensive drugs, digitalis and oral hypoglycemic drugs) and no special medication was given for their stroke. Of clinical data, only the state of consciousness and side and type of hemisymptoms were used in the present study.

CT examination was performed with an EMI mark 1 head scanner with a 160 x 160 matrix. EEG was recorded on a 16-channel EEG mingograph, using both unipolar and bipolar derivations including longitudinal and transversal montages. At least one CT and EEG examination was performed in 30 and 24 of the patients respectively within four days after admission and in all patients within eight days. An additional CT and EEG examination was performed in 24 and 28 patients respectively on day 4-11 after admission.

An EEG abnormality, when encountered, could usually be classified as either U = unilateral, polymorphic theta-delta activity, or B = bilateral, mostly bursts of bilateral delta rhythms, commonly known as frontal intermittent rhythmic delta activity (FIRDA). The degree or severity of an EEG abnormality was further scaled according to a semiquantitative 5-grade scale, mostly based on the spatial extent of an abnormality, but to some extent also including temporal characteristics (short episodes versus continuous activity) and frequency aspects (content of theta or delta rhythms).

An approximate characterization of the 5 grades of EEG abnormality is as follows:

Grade 0 = Normal EEG

Grade 1 = Slight abnormality - typically a few episodes of short duration, mainly theta-rhythms,

restricted to one lobe

Grade 2 = Moderate abnormality - theta-delta activity covering about one half hemisphere, appearing continuously or as frequent episodes of moderate to long duration

Grade 3 = Severe abnormality - continuous mainly delta activity covering a whole hemisphere, or frequent bilateral delta episodes occupying nearly 50% of the recordering

Grade 4 = Very severe abnormality - continuous delta activity over both hemispheres

RESULTS

The results of the study are summarized in Table 1, where the principal clinical features (state of consciousness and hemisymptoms) as well as CT and EEG findings are presented. The clinical findings concerning hemiparesis and the degree of consciousness and orientation were the same during the first four days in all patients except for patient 061 (an initial dysphasia and paresis of the right arm had disappeared after four days) and 120 (disorientation on day 1, then fully orientated). The data in the table are based on the first CT and EEG examination during the first week after admission, except for one case (case 119) where CT was negative on day 0 and positive on day 5. In the table the patients are arranged in groups of increasing grade of EEG abnormality.

EEG was pathological in 24 patients (72,7%) and CT was positive in 17 patients (51,5%). CT and EEG were both negative in only four patients (12,1%), implying that the addition of EEG to CT examination increased the incidence of positive findings from 51,5 to 87,9 %.

Of the 12 CT negative cases with pathological EEG, the EEG abnormality was only slight in 3 cases, and in 9 cases of moderate to very severe degree. Of the latter, 5 cases had unilateral and 4 cases had bilateral EEG-abnormality.

Of the 30 patients with hemisymptoms, 27 had pathological CT and/or EEG, and in most of these cases there was good agreement between clinical findings and CT and/or EEG findings as far as laterality of the lesion was concerned. Of the 22 patients with hemisymptoms and pathological EEG, 14 had unilateral EEG abnormality in agreement with clinical findings, whereas 8 patients had bilateral EEG abnormality, which could give no objective support in lateralizing a lesion. Of the 17 patients with hemisymptoms and positive CT, there were 3 patients where the CT lesion was found in the "wrong" hemisphere: however, in these cases the CT findings were classified as an old infarction probably not related to the present illness (two of these were TIA cases, one case probable infarction).

It can be inferred from Table 1 that there was a correlation between the degree of EEG abnormality and the state of consciousness of the patient: there is an increasing incidence of impairment of consciousness with increasing EEG grade. (Spearman's rank correlation coefficient = 0.62, $P < 0.001$). In most patients the impairment of consciousness was of only slight to moderate degree: only one patient was comatose, and had also a grade 4 EEG abnormality.

Table 1. Main clinical findings and results of CT and EEG examinations in 33 patients after stroke

EEG		Clinical findings		CT findings		EEG findings	
Case no.	Type of lesion	Impairment of consciousness	Hemi-symptoms	Positive or negative	Side	Side	Type
Grade 0							
032	PI	-	R	-		0	
069	TIA	-	L	-		0	
099	PI	-	-	-		0	
124	PI	-	R	-		0	
066	I	-	L	+	R	0	
090	I	-	L	+	R	0	
114	PI	-	R	+	R	0	
001	TIA	-	R	+	R	0	
065	I	-	R	+	L	0	
Grade 1							
054	PI	-	L	-		-	B
115	PI	+	R	-		L	U
042	PI	-	R	-		L	U
012	I	-	R	+	L	-	B
061	I	-	R	+	L	L	U
Grade 2							
009	PI	-	-	-		-	B
059	PI	-	R	-		-	B
004	H	+	R	+	L	L	U
079	I	-	L	+	R	R	U
Grade 3							
052	PI	-	R	-		L	U
063	PI	-	R	-		L	U
098	PI	-	L	-		-	B
112	PI	+	R	-		L	U
120	PI	+	R	-		L	U
136	TIA	+	-	-		L	U
002	I	-	L	+	R	R	U
040	I	+	R	+	L	-	B
038	TIA	-	R	+	R	-	B
067	H	-	R	+	L	L	U
087	H	-	R	+	L	L	U
113	I	+	R	+	L	L	U
119	I	-	L	+	R	R	U
Grade 4							
053	PI	+	R	-		-	B
003	I	+	R	+	L	-	B

Abbreviations: Clinical: I = infarction, H = hemorrhage, PI = probable infarction, TIA = transitory ischemic attack, R = right side of body, L = left side of body, CT: R = right hemisphere, L = left hemisphere, EEG: R = right hemisphere, L = left hemisphere, U = unilateral abnormality, B = bilateral abnormality.
The TIA patients reported hemisymptoms anamnestically but did not disclose any hemisigns at examination

DISCUSSION

The frequency of positive CT and EEG findings reported after stroke have been highly varying mainly depending on the composition of the clinical materials. CT has been reported positive in 0-18 % in TIA, 48-98 % in infarction and 77-100 % in hemorrhage (1, 7, 8, 12, 14), and correspondingly for EEG 66-86 %, 77-90 % and 100% (9, 10, 11, 12). In our material with a majority of infarction cases the incidence of positive findings was 72,7% for EEG and 51,5% for CT, or as low as 42% for CT if the three cases with old infarctions are disregarded. The frequency of negative CT is particularly high in TIA cases, and is generally higher in the first week than in the second week after stroke, particularly during the first 48 hours after onset (2, 7, 8, 11). In our series two TIA cases with negative CT contributed to a low figure, but the time of examination probably played no major role, since most patients also had a CT examination during the second week.

There was disagreement between CT and EEG findings in 17 patients, i.e. as high perecentage as 51. Of these the 5 cases with positive CT and negative EEG findings are of limited interest in the present context: two of these cases had an old infarction, and furthermore, a normal EEG is often reported in deep lesions (3, 12). In three of the 12 patients with negative CT and positive EEG findings the EEG abnormality was only slight (grade 1) and of questionable significance in relation to the present disease, since minor episodes of slow wave activity particularly in temporal areas are not uncommon even in the normal population, especially in elderly individuals (5).

The remained 9 patients with normal CT and EEG abnormality of moderate to very severe degree, which no doubt indicated substantial interference with cerebral blood flow, related to the clinical symptoms of acute CVD. However, we will not further discuss the 4 cases with bilateral EEG abnormality, since in these cases it is difficult to make precise statements about the localization and extent of circulatory disturbances. The FIRDA pattern has traditionally been regarded by electroencephalogaphers as generated from deep midline structures, but recent studies have shown that it may also appear in diffuse encephalopathies or focal hemispheric lesions (4, 13).

The remaining 5 patients had normal CT and a prominent unilateral EEG abnormality, i.e. a combination of findings identical to those of the three cases described by Yanagihara et al. (15). Thus our findings in a fairly small series of patients support the assumption by these authors that this type of discrepancy between CT and EEG findings after stroke may not be uncommon. Correct recognition of this category of patients is important, since at least some of them may have occlusion of the internal carotid arteries which can be successfully treated by surgical intervention (15). EEG is in general probably not part of routine investigations in patients after stroke, but the present findings together with those of Yanagihara et al. (15) suggest that it should be more extensively used, particularly in patients with normal CT examination. If the EEG examination in such cases shows a prominent unilateral abnormality, further investigations with cerebral angiography should be considered.

REFERENCES

1. Campbell, J.K., Houser, O.W., Stevens, J.C., Wahner, H.W., Baker, H.L. & Folger, W.N.: Computed tomography and radionuclide imaging in the evaluation of ischemic stroke. Radiology 126: 695-702, 1978.
2. Davis, K.R., Taveras, J.M., New, P.F.J., Schnur, J.A. & Robertson, G.H.: Cerebral infarction diagnosis by computerized tomography. Am J Roentgenol 124: 643-660, 1975.
3. van der Drift, J.H.A., Visser, S.L., Jonkman, E.J. & v.d. Steen, A.: Correlations and discrepancies between clinical aspects, EEG and CT brainscan data in ischaemic cerebral disease. EEG and Clinical Neurophysiology (eds. H. Lechner & A. Aranibar), pp. 163-172. Excerpta Medica, Amsterdam, 1980.
4. Fariello, R.G., Orrison, W., Blanco, G. & Reyes, P.: Neuroradiological correlates of frontally predominant intermittent rhythmic delta activity (FIRDA). Electroenceph & Clin Neurophysiol 54: 194-202, 1982.
5. Hughes, J.R. & Cayaffa, J.J.: The EEG in patients at different ages without organic cerebral disease. Electroenceph & Clin Neurophysiol 42: 776-784, 1977.
6. Kayser-Gatchalian, M.C. & Neundorfer, B.: The prognostic value of EEG in ischemic cerebral insults. Electroenceph & Clin Neurophysiol 49: 608-617, 1980.
7. Kinkel, W.R. & Jacobs, L.: Computerized axial transverse tomography in cerebrovascular disease. Neurology 26: 924-930, 1976.
8. Ladurner, G., Sager, W.D., Iliff, L.D. & Lechner, H.: A correlation of clinical findings and CT in ischaemic cerebrovascular disease. Eur Neurol 18: 281-288, 1979.
9. Logar, C., Martischnig, R., Enge, S., Sager, W.D. & Ladurner, G.: Zur Wertigkeit von EEG and Computertomographie bei Ischämischer Insulten. Z. EEG-EMG 10: 161-166, 1979.
10. Moglia, A., Tartara, A., Mola, M., Manni, R., Rognoge, F. & Martelli, A.: Value of EEG and computerized tomography in ischemic cerebrovascular disorders. EEG and Clinical Neurophysiology (ed. H. Lechner & A. Aranibar), pp. 226-230. Excerpta Medica, Amsterdam, 1980.
11. Ott, E., Lechner, H., Marguc, K., Bertha, G., Aranibar, A., Ladurner & Sager, W.D.: Correlation of EEG and CT findings with cerebral blood flow measurements in patients with cerebrovascular disorders. EEG and Clinical Neurophysiology (eds. H. Lechner & A. Aranibar), pp. 143-147. Excerpta Medica, Amsterdam, 1980.
12. Ottonello, G.A., Regesta, G. & Tanganelli, P.: Correlation between computerized tomography and EEG findings in acute cerebrovascular disorders. EEG and Clinical Neurophysiology (eds. H. Lechner & A. Aranibar), pp. 148-162. Excerpta Medica, Amsterdam, 1980.
13. Schaul, N., Lueders, H. & Sachdev, K.: Generalized, bilaterally synchronous bursts of slow waves in the EEG. Arch Neurol 38: 690-692, 1981.

14. Söderström, C.E.: Diagnostic significance of CSF spectrophotometry and computer tomography in cerebrovascular disease. A comparative study of 231 cases. Stroke 8: 606-612, 1977.
15. Yanagihara, T., Houser, O.W. & Klass, D.W.: Computer tomography and EEG in cerebrovascular disease. Arch Neurol 38: 597-600, 1981.

Address for reprints:
Martin Knibestöl, M.D.
Department of Clinical Neurophysiology
University Hospital
901 85 UMEÅ
Sweden