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# Computed Tomography Study of Cerebral Infarctions Treated With Vinpocetine

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## ABSTRACT

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A group of 20 persons with cerebral infarctions who had been treated with vinpocetine was compared with a group of 20 similar patients who had been treated with other vasoregulative drugs. Two computed tomography (CT) examinations were evaluated for each patient. The initial scan was done during the first ten days, and the second between the 20th and 50th day after the stroke. Three parameters were studied: extension of the lesion, intensity of hypodensity, and presence of postcontrast enhancement. In the first and second criteria, the vinpocetine-treated patients exhibited better results, whereas the third did not yield any explicit data.

**Key words: computed tomography, vinpocetine, cerebral infarctions**

## INTRODUCTION

Computed tomography (CT) proved to be a most valuable aid in observing the morphologic criteria of cerebral infarctions. Among the main advantages of the CT method, the following capacities dominate: determination of the lesion volume, determination of the degree of hypodensity and determination of postcontrast enhancement. These parameters can help to evaluate the influence of vasoregulative drugs on the development of cerebral infarctions. We performed a comparative study, based on CT results, between a group of 20

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**TABLE 1. Development of Volume of Ischemic Lesions From the First to the Second CT Examination***

| | Vinpocetine patients | Control group |
|---|---|---|
| Increase | 3 | 2* |
| No change | 6 | 8 |
| Decrease | 11 | 9 |

*Plus one who suffered hemorrhage into malacia.

patients treated with vinpocetine and another group of 20 patients treated with other vasoregulative drugs.

## PATIENTS AND METHODS

Forty patients with clinical and CT diagnosis of acute stage cerebral infarction were divided into two groups. Twenty patients of the first group (aged 28 to 80 years, with an average of 58.7 years) were treated with vinpocetine, whereas 20 other patients (aged 4 to 72 years, with an average of 43.8 years) were treated with another drug.

The first group (13 males and 7 females) was given 20 mg vinpocetine daily in a drip infusion during the first week of treatment, starting on the first day with 10 mg in order to observe the initial reaction. After the first week, therapy was continued with oral application $3 \times 5$ to 10 mg. The control group (14 males and 6 females), was composed of patients from three different departments who had been treated with either aethophyllin 400–800 mg/day or xanthinol nicotinate 300–600 mg/day parenterally during the first week, with continuation of 300–600 mg/day of aethophyllin or 1000 mg/day of xanthinol nicotinate orally respectively, beginning the second week. Dextran and manitol infusions were used during the first week in both groups; an auxiliary corticosteroid therapy, used sporadically in both groups, could not be uniform due to factors such as age or diabetes mellitus in some patients.

All persons from both groups were examined by CT at least twice during the first 50 days, the first examination within 10 days and the second between the 20th and 50th day after the stroke. Each examination evaluated 9 to 10 plain scans as well as the same postcontrast scans. Each patient received 1 ml Diatrizoate (Verografin 60% = 290 mg iodine per ml) per 1 kg body weight intravenously as a contrasting agent. The character of cerebral tissue lesions was lysed further with respect to the volume of cerebral infarction, degree of hypodensity, and intensity of contrast enhancement.

## RESULTS

### Extent of Infarcted Area

To avoid misinterpretations of data from the complicated field of the posterior fosse, only lesions in cerebral hemispheres were evaluated. Their extent varied from large infarctions involving several vessels of the circulus of Willis to smaller areas, but never less than 2 cm in diameter. Comparison of the malatic volume between the first and second examination is demonstrated in Table 1. One patient from the control group suffered a hemorrhage into malacia, which led also to an increase in volume due, however, to a different mechanism.

### Degree of Tissue Hypodensity

The process of tissue deterioration is expressed in CT by the lowering of density values in the hypodense district. We scrutinized the number of patients in both groups who underwent a decrease in hypodensity between the first and second CT examination. Further, we noted some separate cases in which the hypodensity decreased to zero; such findings indicate a complete coliquation of the tissue, a sign of full necrosis. These results are presented in Table 2. Among the patients whose lesion density increased between the first and second examination, two in the vinpocetine group and one in the control group tended to have an

**TABLE 2. Development of Hypodensity in Infarcted Area From the First to the Second CT Examination**

| | Vinpocetine patients | Control group |
|---|---|---|
| Decrease in density of hypodense ischemic lesion | 12 | 15 |
| Decrease to necrotic values near O Hounsfield units | 6 | 9 |
| No change | 2 | 2 |
| Increase in density of hypodense ischemic lesion | 6 | 2[a] |

[a]Plus one who suffered hemorrhage into malacia.

increase to normal values. Another patient in the first group was found to have nearly normalized values following the second examination, but the third examination (separate from out study) revealed a secondary loss of tissue density, indicating later destruction of the malatic zone.

### Intensity of Postcontrast Enhancement

In both groups, a nine times postcontrast enhancement was found in the first CT. In most cases, this enhancement disappeared until the second examination only twice in the vinpocetine group and five times in the control group enhancement increased in comparison with the first examination; this occurred not only in patients previously demonstrating positive enhancement, but it also appeared in previously negative ones. Regarding postcontrast changes, we were unable to find any signs that could elucidate our results in relation to therapy.

## DISCUSSION

Factors that influence the development of cerebral infarctions are many. In contrast to the general state of the patient, the etiology of the disturbed perfusion of cerebral tissue, factors like viscosity, blood cell deformability, the character of vascular stenoses, and all given conditions, drug treatment probably has only a modest role in the destiny of the damaged tissue. Nevertheless, we believe that vasoregulative drugs are able to stop spreading of hypoxia into neighbouring areas, restrain intracellular edema by improving collateral circulation, and limit blood-brain-barrier disturbances. CT follow-up provides an opportunity to differentiate factors contributing to the resulting state.

The interpretation of the volume of the infarcted area must be understood in terms of temporal development. A diminution of malatic area—for example, in later stages after the stroke—is quite a normal phenomenon, produced by the retraction of the malatic pseudocyst. In early stages, however, is it a sign of normalization of edematous tissue in the peripheral fringe of the infarction. In most cases, our CT examinations were performed during stages when no atrophy was present.

The evaluation of tissue density is complicated by two major circumstances. First, the hypodensity is produced by a combination of intracellular edema, extracellular edema appearing several days later [Katzman et al., 1977], and tissue necrosis [Inoue et al., 1980; Valk, 1982]. Second, sometimes the hypodensity can be hidden by a temporary hyperemia—a so-called "fogging effect" [Becker et al., 1979]. To distinguish such cases requires more consecutive CT examinations. Also, it is important that various infarctions have their own different temporal development. Therefore, the follow-up of blood-brain-barrier damage in terms of postcontrast enhancement in our investigations yielded no significant results. We compared, therefore, the presence of postcontrast enhancement with data on temporal development of this phenomenon, studied in our previous analysis [Kalvach and Bauer, 1984],

as well as in the literature [Valk, 1982]. We were unable to find an exact temporal dependence to elucidate our findings.

Most of the individual differences in the development of cerebral infarctions can be overcome in comparative drug studies only by using larger numbers and larger groups of patients. We are of the opinion, however, that our Ct analysis confirms our previous positive clinical results [Kalvach et al., 1982] in patients treated with vinpocetine.

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