CT DENSITY DYNAMICS IN CEREBRAL INFARCTIONS

S. Kovachev

Key-words: computer tomography — density image — cerebral infarction — diagnosis

Brain computer tomography (CT) has established itself as a method of choice in the diagnosis of vascular diseases and their differentiation from other pathological processes. The use of quantitative computerized evaluation of X-ray absorption enables the determination of the density of single cerebral regions. Thus CT image of cerebral infarction (CI) as a low-density zone is well investigated (4, 9, 11). Some authors (12, 16) pay attention to the possible negative CT in case of well-manifested clinical neurological symptomatics. They report together with other investigators (8, 10) data from single investigation of different patients at a given time. Serial studies at different time intervals are scanty (5, 7).

There are few literature data available from multifold examinations at equal time intervals concerning the change of CT density with cerebral ischemia.

The purpose of the present study is to get information about the dynamics of CT density with cerebral infarctions.

Material and methods

We performed repeated CT investigation of 94 brain infarction patients (61 males and 33 females) aged between 21 and 83 years (mean age of 57 years). The initial 4 examinations were carried out at one-week intervals in all the patients. The time lag between the accident and the first examination varied between 2 and 144 hours (at the average 80 hours). The second examination was done during the second week, at the average on the 9th day, the third one — at the average on the 19th day, the 4th one — on the 30th day. The 5th examination was realized in 65 patients between the 3rd and 6th month (at the average in the 4th one). 19 patients were followed-up after 18 months (at the average in the 22th month).

The examinations were performed by using of the apparatus «Siretom — I» with matrix image transformer of 256×256 pixels and 10 mm thick sections without contrast matter injection. We determined visually the pathologically altered density. The gradual expressiveness of hypodensity was read according to a three-degree scale as presented on table 1.

Table 1

CT density changes

Degree	Characteristics
0 degree	no density changes
Ist degree	slight density reduction
IInd degree	moderate density reduction
IIIrd degree	density equal to that of cerebro-spinal fluid

Results and discussion

CT was negative during the whole examination series in 9 patients. The hypodensic finding did not show any dynamic changes in another patient. We con-

Table 2

CT density dynamics according to the time of examination

Time of examination	Patients n	Density dynamics					
		increase		without changes		decrease	
		n	%	n	%	%	n
8th—14th day	84	40	47.6	23	34.5	21	25
15th—21st day	84	22	26.1	32	38.0	30	35.7
22nd—30th day	84	5	5.9	17	20.2	62	73.8
3rd—6th month	65			47	72.3	18	27.6
after 12th month	19			19	100		

sidered this case a cyst after vascular accident. In fact, CT density changes were discussed in a total of 84 patients. CT density dynamics in relation to the period of examination was shown on table 2 and fig. 1.

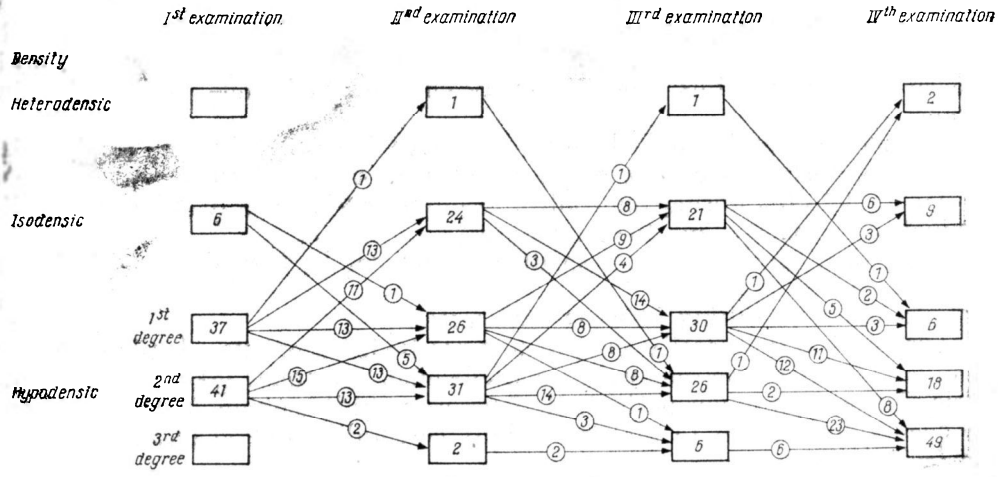

Fig. 1.

On the basis of the analysis of these data four main types of density changes can be differentiated as follows:

Type 1. Progressive reduction of the density from slightly decreased density down to that equal to CSF one. There are 27 patients (32 per cent) here. There is a hypodensic finding in all the examinations and at the last stage a cyst is formed.

Type 2. At the onset the finding is hypodensic and then in a definite interval it becomes isodensic or almost isodensic. Then it reappears and advances like

that one of the first type. This phenomenon is described by H. Becker et al. (6) as a fogging effect. These authors accept that it occurs in all the patients during the second and third week after stroke. There are 47 patients (55.9 %) here. These data are close to those reported by E. Skriver et al. (14), namely of 46 per cent of the cases. If the examinations is carried out during this time interval the ischemic lesion visualization can also be overlooked. However, this effect is not completely pathomorphologically explained in the literature. Density reduction in the first several days is due to oedema (13). In the 2nd and 3rd week capillary proliferation and macrophage extravasation reaches its maximal level (15, 17). On the 7th day reparative processes set in manifested by glial hyperplasia (3). This restoration process is opposite to dystrophic and destructive alterations more severely expressed during the 2nd week. It is possible that the prevalence of some of these processes during a sertain period of time can be the reason for density changes. Besides the stage of necrosis resorption during the 2nd and 3rd week is characterized by activation of blood cells which begin to move from borderline zones and then together with hypertrophied astrocytes implant in the necrotic area. At this moment granular cell formation sets in. Fibril-forming or fibrillose astrocytes with different fibril content appear. Round necrotic focus vascular new-formation starts (2). Brain perifocal oedema decreases strongly during this period, too. Reparative processes, oedema reduction as well as nervous fibre demyelinization reduce X-ray absorption in the damaged region and thus probably induce CT density increase.

Type 3. Progressive increase of the low density from slightly or moderately reduced one up to an equal to normal one of brain tissue. There are 6 of our patients (7.1 per cent) in this group. No cysts are established during control examinations. This density change can be due to long-lasting restorable ischemic states. At the beginning neurological disorders occur which resemble an insult but restitution sets in for 2—7 days (1). Probably, their CT density is equal to that of brain parenchyma. Besides this type of density change can be due to the following reasons:

— small lesion located closely to massive bone structures around which at the onset there has been a perifocal oedema. After oedema disappearance it can not be detected during the control examination because it is below the discrimination capacity of the apparatus;

— cortical microinsult. Its small cyst can not be then distinguished from convex sulci;

— small cyst falling between two sections.

Type 4. The low density sharply becomes isodensic or heterodensic with more densified areas on the periphery or as a whole zone. Later on it again becomes hypodensic. There are 4 of our patients (4. 7 per cent) in this group. This type of density change corresponds to the transition of cerebral infarction into red encephalomalacia.

This moment namely can be detected during dynamic CT investigation. These processes during single examination and in case of isodensity both can be overlooked, indeed. On table 3 the dynamic type of the patients studied is demonstrated.

We can draw the following conclusions:

1. Kind and topic diagnosis with type 1 density changes is not difficult when multifold examined.

2. In case of type 2 density change, if the first examination is performed in the 2nd and 3rd week and it is negative a repeated one is required after 30 days or contrast mattery injection should be used.

Table 3

Distribution of patients according to the type of density changes

Density type \ Number	n	%	Δ
Type 1	27	32	±0.38
Type 2	47	55.9	±0.28
Type 3	6	7.1	±0.80
Type 4	4	4.7	±0.98
Total	84	100	

3. With the type 3 of density change the process can be diagnosed only while brain oedema exists.

4. The ascertaining of the moment of ischemia transition into red encephalomalacia contributes to correct diagnosis.

REFERENCES

1. Карагьозов, Л. Хирургично лечение на съдовите заболявания на мозъка. С., Мед. и физк., 1981. — 2. Туманов, В. П. *Арх. патол.*, 1983, № 1, 3—12. — 3. Чумаков, А. А., А. А. Истомин. *Арх. патол.*, 1986, № 4, 45—51. — 4. Alcala, H., M. Gade, K. M. Torack. *Arch. Neurol.*, 35, 1978, N 1, 1—7. — 5. Aulich, A. *Acta Neurol.*, 4, 1977, 129—140. — 6. Becker, H., H. Desch, H. Hacker, A. Penz. *Neuroradiology*, 18, 1979, 165—192. — 7. Campbell, J. K., O. M. Houser, F. C. Stevens, H. W. Wahner, H. L. Beker, W. M. Folger. *Radiology*, 126, 1978, 695—702. — 8. Constant, P., A. M. Renou, F. Vernhist. In: Europ. Semin. Comput. axial tomogr. in clin. pract., I. Berlin, Springer, 1977, 227—236. — 9. Ore, D. G., S. Perini, A. Benati, A. Beltramello. *Europ. Neurol.*, 1978, 53—59. — 10. Davis, K. R., J. M. Taveras, P. F. New, J. A. Schnur, G. H. Robertson. *Amer. J. Roentgenol.*, 124, 1975, 643—660. — 11. Fields, W. S., F. M. Bell, J. K. Campbell, M. S. Huckman, M. M. Schecheter, J. C. Stevens, A. G. Waltz, G. H. Wilson. *Stroke*, 6, 1975, N 1—2, 105—107. — 12. Masdeu, J. C., B. Azar-Kia, F. A. Rubino. *Arch. Neurol.*, 34, 1977, 471—481. — 13. Rieth, K. G., K. Fujiwara, G. Di Chiro, I. Klatzo, R. A. Brooks, K. G. Johnston, C. M. O'Connor, L. G. Mitchel. *Radiology*, 135, 1980, 343—348. — 14. Skriver, E. B., T. S. Olsen. *Neuroradiology*, 22, 1981, N 2, 61—65. — 15. Spatz H. *Z. Neurol.*, 167, 1939, 301—349. — 16. Wing, S. D., D. Norman, J. A. Pollock, T. H. Newton. *Radiology*, 121, 1976, 89—92. — 17. Yates, P. O. In: Greenfield's Neuropathology. London, E. Arnold, 1976, 86—147.

КТ ДИНАМИКА ПЛОТНОСТИ ИШЕМИЧЕСКИХ МОЗГОВЫХ ИНСУЛЬТОВ

С. Ковачев

РЕЗЮМЕ

Проведено системное серийное компьютертомографское исследование 94 больных с ишемическим мозговым инсультом. Из анализа полученных результатов можно оформить четыре основных типа изменений плотности. Первый тип — прогрессивное уменьшение плотности, которая постепенно доходит до плотности ликвора. К этой группе можно отнести

27 (32 %) больных. Второй тип — гиподенсная в начале находка в определенном интервале времени становится изоденсной, после чего снова появляется. К этой группе относятся 47 (55,9 %) больных. Третий тип — прогрессивное увеличение низкой плотности до плотности, которая не различается от нормальной мозговой паренгимы. Это обнаружено у 6 (7,1 %) больных. Четвертый тип — низкая плотность резко становится изоденсной или гетероденсной с наличием различных по величине более плотных участков. Такое изменение наблюдалось у 4 (7 %) больных.

Обсуждаются патоморфологические субстраты и возможности видового и топического компьютертомографского диагноза при каждом из них.