

Transient Disappearance of Cerebral Infarcts on CT Scan, the So-Called Fogging Effect

Elisabeth Bech Skriver and T. Skyhøj Olsen

Department of Neuroradiology, Hvidovre Hospital, and Neurology, Bispebjerg Hospital, Copenhagen, Denmark

Summary. The fogging effect, whereby initially hypodense infarcts become isodense during the second and third week after the onset of stroke, was investigated in a prospective and consecutive series comprising 50 cases with completed stroke. CT scans were performed approximately 3 days, 10 days and 6 months after stroke. The fogging effect was found in 54% of cases. The hypodense areas reappeared on all scans at 6 months. Cerebral infarcts therefore may be overlooked or grossly underestimated if the scan is performed during the second and third week after stroke.

Key words: Computed tomography – Fogging effect – Completed stroke – Cerebral infarcts

Introduction

The finding of a normal plain CT scan, despite unquestionable symptoms of cerebral infarction in stroke patients, is well known [1, 3, 7, 9]. This is commonly seen in the second and third week after the onset of stroke but rarely in the first week [2, 4]. This phenomenon, that initially hypodense infarcts become isodense during the second and third weeks after stroke, or close to isodense on sequential scans, has been denoted as the fogging effect by Becker et al. 1978 [1].

The present study attempts to investigate the incidence and elucidate the clinical significance of this fogging effect in a sequentially studied series of stroke patients.

Material and Methods

The present study is prospective and consecutive comprising 50 patients below the age of 75, all suffer-

ing from completed stroke. Transient ischemic attacks and intracerebral hematoma were not included. The diagnosis was established in all cases by clinical neurological examination, electroencephalography, ⁹⁹Techetium isotope scintigraphy, cerebral angiography and computed tomography.

Plain CT scan was performed in all cases within 1 week after the onset of stroke. A second CT scan was performed approximately 2 weeks later, and a third scan approximately 6 months after the stroke. All patients were examined with an EMI CT 1010 scanner using the 160 × 160 matrix.

The density was measured in the center of the infarcts, using a circle with a radius varying from 3.8–9.5 mm, depending on the size of the lesion. The absorption values thus obtained on the first, second and third examination were compared in order to evaluate the fogging effect quantitatively. The density of normal areas symmetrical to the infarcts were likewise determined on the first, second and third examinations.

Results

The interval between stroke and the first CT scan averaged 79 h (range 38–144 h); 18 days (range 13–25) between the stroke and second CT scan and 6 months between the stroke and third CT scan.

The CT scan revealed infarcts as hypodense areas on the first examination in 31 (62%) of the 50 cases investigated. In the remaining 19 cases the infarcts were not seen as hypodense areas until the second or the third examination in 9 cases (18%), while no focal changes were demonstrated on any scan in 10 cases (20%). Only cases showing hypodense lesions on the first examination were analyzed to evaluate the fogging effect. Three patients died between the first and

Table 1. The density (HU) of the center of the infarcted area on the first (CT I), second (CT II) and third (CT III) CT examinations and the density of the normal symmetrical area. x indicates cases fullfilling the first criterion for fogging effect

Case	HU of lesion			HU of symmetrical area CT II	Increase of HU from CT I to CT II	Difference of density between lesion and symmetrical area on CT II
	CT I	CT II	CT III			
1	23.92	29.48	-	30.51	5.56x	1.03 ^a
2	13.74	18.96	4.69	31.01	5.22x	12.05
3	25.30	46.66	15.35	46.77	21.36x	0.11 ^a
4	11.20	24.37	-	32.59	13.17x	8.22
5	23.16	28.97	20.86	30.89	5.81x	1.92 ^a
6	19.43	31.59	14.86	32.27	12.16x	0.68 ^a
7	19.97	25.75	11.14	28.19	5.78x	2.44 ^a
8	15.67	27.27	9.00	26.49	11.60x	-0.78 ^a
9	21.48	26.86	7.08	33.30	5.38x	6.44
10	21.33	36.27	6.00	32.75	14.94x	1.48 ^a
11	18.17	23.98	11.99	30.49	5.81x	6.51
12	21.75	33.01	12.23	32.08	11.26x	-0.93 ^a
13	24.52	25.38	10.33	28.43	0.86	3.05
14	26.58	21.70	-	41.78	-4.88	20.07
15	15.49	15.62	14.43	36.68	0.19	14.38
16	22.61	32.55	23.71	31.23	9.94x	-1.32 ^a
17	14.78	28.04	18.45	27.54	13.26x	-0.50 ^a
18	28.52	27.76	-	31.51	-0.76	5.95
19	25.67	35.56	10.67	35.49	9.89x	-0.07 ^a
20	17.89	28.78	12.89	32.06	10.89x	3.26 ^a
21	19.00	30.14	7.67	33.61	11.14x	3.47 ^a
22	26.90	32.19	15.95	35.07	5.29x	2.88 ^a
23	27.14	24.96	19.27	25.49	-2.18	0.54
24	26.43	25.66	14.67	28.76	-0.77	3.10
25	16.89	32.66	13.44	33.33	15.77x	0.67 ^a
26	15.48	19.52	12.67	25.67	4.04	6.15
27	20.76	18.71	19.10	25.90	-2.02	7.19
28	20.43	27.95	22.11	33.48	7.52x	5.53

^a indicates cases fullfilling both criteria

second examination. The study therefore comprises 28 patients.

The following criteria must be fullfilled if fogging is considered to be present:

1. There must be a significant increase in the density from the first to the second examination.

2. The infarcted area should be isodense or close to isodense on the second examination, i.e. the density of the infarcted area should not differ significantly from the density of the corresponding symmetrical region.

The difference of density between the first and second examination in the normal contralateral area was measured and the random experimental error, calculated as the standard deviation $\sqrt{\frac{\sum d^2}{2n}}$, was 2.12 Hounsfield units (HU). Thus an increase of density from the first to the second examination of more than 4.24 HU (2 SD) was considered significant ($p = 0.05$), and a difference between the density of the le-

sion and the corresponding symmetrical region below 4.24 HU (2 SD) was considered to be within normal limits. The density in the center of the infarcted areas on the first and second CT examinations appears in Table 1. The density increased more than 4.24 HU from the first to the second scan in 20 cases (71%). Hence these cases fullfill our first criterion for considering the fogging effect to be present. Of these 20 cases 15 also fullfilled our second criterion. Thus, the fogging effect was present in 15 of the 28 cases investigated (54%).

This result was concordant with our experience from visual inspection of the CT scans. Examples of cases showing the fogging effect are seen in Figure 1. The second examination of the cases showing the fogging effect was carried out from the 13th to the 21st day after stroke (average 16 days). The second examination of the remaining 13 cases in which the fogging effect was not seen was performed from the 15th to the 24th day after stroke (average 19 days). No significant difference was seen between the time elapsed

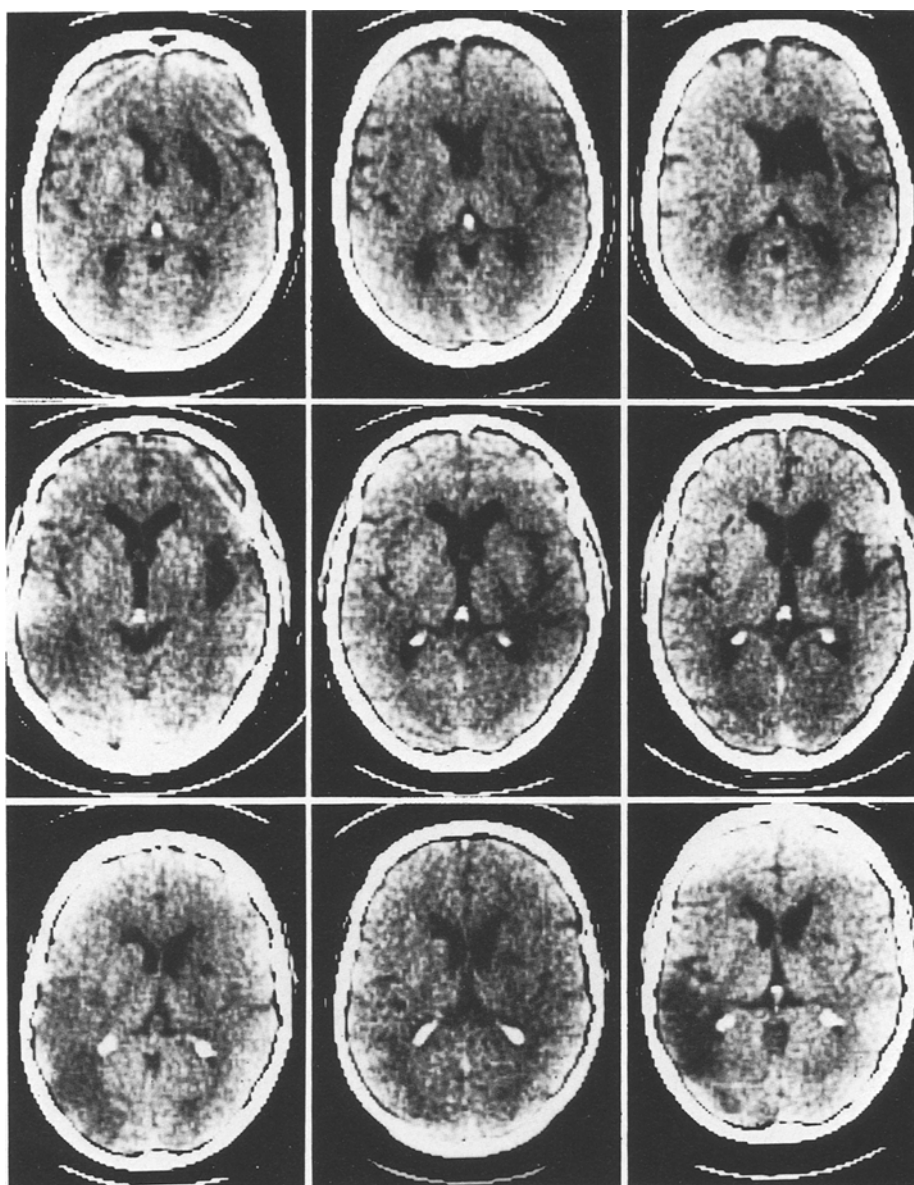


Fig. 1. Three cases showing the fogging effect on CT scans performed during the first week, between the second and third weeks, and 6 months after stroke. The hypodense area, which had almost disappeared on the second scan, returned to hypodensity on the third scan

from the stroke to the second examination in these two groups (unpaired t-test, $t = 1.79$ $p > 0.05$). A third CT scan performed 6 months after stroke was available in 14 of the cases showing the fogging effect. The lesion had returned to hypodensity in all these cases as shown in Table 1. The late scans typically show a more pronounced and sharper demarcated hypodense area.

Discussion

The fogging effect was seen in the present series in 54% of the cases investigated.

This figure is far above the frequency reported in other studies in which the fogging effect was manifested as a false negative on CT scans performed within 4 weeks after stroke; Yoch et al. 1978, 10–20% [9]; Wing et al. 1976, 37% [7]; Masdeu et al. 1977, 20% [3]. However, these studies were not designed to investigate the fogging effect: serial CT scan was not performed within the second and third weeks after stroke and most cases were investigated only once within the first 4 weeks.

Becker et al. 1978 [1] studied 10 selected patients with cerebral infarction. CT scans were performed on admission and on the 3rd, 7th, 14th, 21st, 28th and 42nd day after stroke. In this series a fogging effect

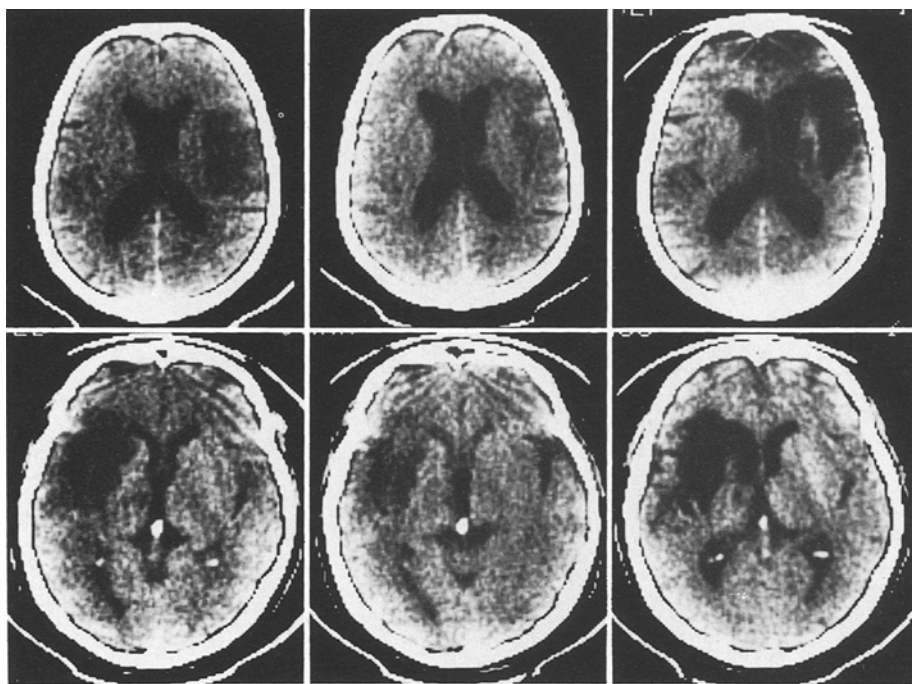


Fig. 2. Two cases showing some degree of fogging on CT scans performed during the first week, between the second and third weeks, and 6 months after stroke. The hypodense area is still visible on the second examination, but the density has increased

was reported to be a constant phenomenon during the second and third weeks after stroke.

In our unselected series the second CT scan was also performed within the second and third weeks, but although the fogging effect was frequently seen (54% of the cases), it was not a constant phenomenon. The time elapsed from stroke to the second scan was identical in our two groups with and without the fogging effect. Therefore a difference in the interval from stroke to the second CT in these two groups cannot explain the absence of fogging in 46% of our cases (Fig. 2).

However, the time range within which the fogging effect reaches its maximum is probably variable. Therefore the fogging effect has not been demonstrated in some cases as the second CT scan was not performed when this maximum was reached. In fact, almost half of the cases without fogging (18% of the entire material) showed a significantly increased density from the first to the second CT examination. Although not satisfying both our criteria for fogging, the increased density in these cases probably represents the same pathophysiological process. It should also be emphasized that the ischemic episodes leading to infarction most likely differ in duration and severity from case to case. Therefore infarcts may develop differently changing the ordinary course and appearance on CT. The frequency therefore might be higher than indicated in this study. The fogging during the

second and third weeks is not yet satisfactorily explained in the literature. Our study cannot explain the pathophysiological process leading to the fogging effect. Some suggestions may, however, be put forward. The decreased density seen in the first few days after infarction is mainly due to the development of edema [5]. The well known diminution of edema and mass effect during the second and third weeks could explain the return to isodensity. Furthermore, the proliferation of capillaries and extravasation of macrophages reaches a maximum during this period of the absorption process of necrotic material [6, 8].

This might also contribute to the increase of density. The gradual transformation of the infarcted necrotic area into a cystic space filled with fluid explains the final return to hypodensity also seen in our study [6, 8].

Clinical Implications

If CT scan is performed in the period when the fogging effect reaches a maximum, i.e. during the second and third weeks after stroke, cerebral infarcts may be overlooked or the size of the infarct may be grossly underestimated. A normal plain CT scan during this period does not rule out cerebral infarcts and must be repeated or performed after contrast administration to confirm or exclude the diagnosis.

References

1. Becker H, Desch H, Hacker H, Pencz A (1979) CT fogging effect with ischemic cerebral infarcts. *Neuroradiology* 18: 185-192
2. Drayer PD, Dujovny M, Boehnke M, Wolfson SK, Barrionuevo PJ, Cook EE, Rosenbaum AE (1977) The capacity for computed tomography diagnosis of cerebral infarction. *Radiology* 125: 393-402
3. Masdeu JC, Azar-Kia B, Rubino FA (1977) Evaluation of recent cerebral infarction by computerized tomography. *Arch Neurol* 34: 417-421
4. Palmers Y, Staelens B, Baert AL, Termote L (1978) Cerebral ischemia. In: Baert A, Jeanmart L, Wackenheim A (eds) *Clinical computer tomography head and trunk*. Springer, Berlin Heidelberg New York, pp 113-127
5. Rieth KG, Fujiwara K, Di Chiro G, Klatzo I, Brooks RA, Johnston GS, O'Connor CM, Mitchell LG (1980) Serial measurements of CT attenuation and specific gravity in experimental cerebral edema. *Radiology* 135: 343-348
6. Spatz H (1939) Pathologische Anatomie der Kreislaufstörungen des Gehirns. *Z Neurol* 167: 301-349
7. Wing SD, Norman D, Pollock JA, Newton TH (1976) Contrast enhancement of cerebral infarcts in computed tomography. *Radiology* 121: 89-92
8. Yates PO (1976) Vascular disease of the central nervous system. In: Blackwood W, Corsellis JAN (eds) *Greenfield's neuropathology*. Edward Arnold, London, pp 86-147
9. Yock DH, Marshall WH (1975) Recent ischemic brain infarcts at computed tomography: appearances pre- and postcontrast infusion. *Radiology* 117: 599-608

Received: 31 March 1981

Dr. Elisabeth Bech Skriver
 Department of Neuroradiology
 Hvidovre Hospital
 Kettegaard Alle 30
 DK-2650 Hvidovre
 Denmark