

# Indium-111-Granulocyte Scintigraphy in Brain Abscess Diagnosis: Limitations and Pitfalls

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The scintigrams and records of 28 patients referred for indium-111-granulocyte scintigraphy ( $^{111}\text{In-GS}$ ) because of a suspected brain abscess were studied retrospectively. The final diagnosis was brain abscess in 8 patients, brain tumor in 18 patients, and infarct and hematoma in 1 patient each. Five patients not on corticosteroid treatment showed intense focal  $^{111}\text{In}$  accumulation in abscesses, whereas an abscess patient receiving a high steroid dose showed no uptake. Two patients studied twice showed intense uptake in abscesses when not on steroid therapy or on a low dose, whereas no uptake was seen when they received high or medium doses. Weak or moderate  $^{111}\text{In}$  uptake was observed in nine tumors. Microscopically assessed degree of tumor granulocyte infiltration, vessel proliferation, and hemorrhage did not correlate with the outcome of  $^{111}\text{In GS}$ . Our results suggest that intense focal cerebral  $^{111}\text{In}$  uptake favors the abscess diagnosis. Abscesses may go undetected, however, in patients on high- or medium-dose steroid therapy.

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**A**n abscess within the brain is a serious condition, and its successful treatment requires considerable diagnostic and therapeutic skills. With the wider availability of computerized tomographic (CT) scanning and more appropriate antibiotic regimens, the outcome of treatment for brain abscesses has improved substantially (1). However, CT may be unable to differentiate abscesses from other types of lesions such as tumors, infarction or hematoma (2-4). Even if clinical history, physical examination, and laboratory findings may aid in differential diagnosis (2,5), diagnostic problems often remain. Indium-111-granulocyte scintigraphy ( $^{111}\text{In-GS}$ ) is a sensitive method for the detection of soft-tissue infection (6,7). So far, limited experience has been gained in the study of suspected cerebral abscesses (8-10). The present paper reviews the results of a retro-

spective study of patients with focal intracerebral lesions subjected to  $^{111}\text{In-GS}$ , with special references to the value and the pitfalls of this method in brain abscess diagnosis.

## MATERIALS AND METHODS

### Patients

We reviewed all  $^{111}\text{In}$  granulocyte scintigrams and the corresponding records from patients with a suspected brain abscess, studied during a five-year period. The patients were referred for  $^{111}\text{In-GS}$  because of clinical and CT findings compatible with or suggestive of brain abscess. The CT-based suspicion was founded on a low-density lesion with a ring of uniform contrast enhancement, surrounded by hypodense edema.

Thirty-two patients (corresponding to 35 scintigraphic studies) were evaluated. Of these, four patients (each studied once) were excluded because of diagnostic uncertainty, leaving 28 patients (31 studies) for detailed evaluation. The four patients excluded presumably suffered from postoperative hematomas (two patients,  $^{111}\text{In-GS}$  neg.), septic infarction ( $^{111}\text{In-GS}$  neg., remnants of a small abscess at autopsy five weeks later), and multiple small abscesses or infarcts ( $^{111}\text{In-GS}$  weakly pos.) resolving on antibiotic treatment. The age composition of the remaining 28 patients (17 females, 11 males) is shown in Table 1, which also shows which patients were treated with steroids and/or antibiotics.

The abscess diagnosis was based on the demonstration of pus in the intracerebral lesion in four patients, and in the remaining four patients on the course, including clinical recovery and disappearance of the CT abnormalities during follow-up. The tumor diagnosis was based on microscopic examination of tumor tissue obtained by craniotomy or autopsy in 13 cases, and by stereotactic biopsy in 5 cases.

### Granulocyte Isolation and Labeling

The isolation and  $^{111}\text{In}$ -oxine labeling in a plasma-buffer mixture of ACD anticoagulated autologous granulocyte-enriched leukocytes has been described previously (11,12).

The median dose injected was 15.2 MBq (range 5.0-18.8 MBq). The median fraction of cell-bound radioactivity in the injected samples (calculated in 12 cases) was 99.3% (range 96.9%-99.7%). The corresponding erythrocyte-bound fraction was 1.7% (range 0.5%-10.7%). The granulocyte/mononuclear cell ratio in the injected sample was calculated in 12 cases. It ranged from 1.1 to 41.8 (median 7.7).

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## Image Acquisition

Following injection of the <sup>111</sup>In-labeled granulocytes, their appropriate *in vivo* behavior was secured by one or more anterior view recordings on Polaroid film of their initial lung transit pattern (13). Following this, repeated static imaging of the head (corresponding to the 173-keV and 247-keV photo-peaks of <sup>111</sup>In, using a Maxi Camera 535, General Electric, Milwaukee, WI) was carried out in the 4-hr postinjection (p.i.) period (2–5 images, median 4, all patients imaged 2 and 4 hr p.i.), and 18–24 hr p.i. as well. At least two projections, including a lateral one, were obtained routinely. If judged necessary for image interpretation and allowed for by the patient's condition, four projections (anterior, posterior, left lateral, right lateral) were obtained. The images were obtained for preset time (300 sec on Day 1, 375 sec on Day 2, at injected

doses of 15–18 MBq, for a correspondingly longer time at lower doses).

Abnormal intracranial foci of <sup>111</sup>In activity were graded as weak, moderate, or intense on the 18–24-hr p.i. images. Intense uptake corresponds to that of the skull base, moderate activity to that of the cranial wall, weakly positive lesions are definitely seen, but with lesser activity than the cranial wall.

## Microscopic Method

The degree of neutrophil infiltration in tumors and abscesses was graded, using a scoring system (0-1-2-3) as previously described (12). A similar scoring system was adopted for the assessment of tumor vessel proliferation and non-artificial hemorrhage. A combined score was obtained by adding half of the vessel proliferation and hemorrhage scores to the granulocyte score.

**TABLE 1**  
Patient Characteristics and Results of <sup>111</sup>In-Granulocyte Scintigraphy

Patient	Age	<sup>111</sup> In-GS <sup>*</sup>	Final diagnosis <sup>†</sup>	Confirm. study <sup>‡</sup>	Steroid therapy <sup>§</sup>	Antibiotic therapy <sup>¶</sup>
1	52	Pos(i) [60']	Abscess (1 mo)	C [1 yr]	—	Amp, Metro
2	33	Pos(i) [60']	Abscess (12 d)	C [3 yr]	—	Pen, Str, Chl
3	17	Pos(i) [4 hr]	Abscess (2 wk)	S [5 d]	—	Pen, Str, Metro
4a	27	Pos(i) [5']	Abscess (3 wk)	S [7 d]	Dxm (4 mg) [1 d]	Amp, Metro
4b		Neg	Abscess (6 wk) +infarct (2 d)	A [4 d]	Dxm (8 mg) [1 d]	Amp, Metro
5a	71	Neg	Abscess (18 d)	C [22 mo]	Dxm (24 mg) [12 d]	—
5b		Pos(i) [2 hr]	Abscess (3.5 mo)	C [25 mo]	Pred (2.5 mg) [2 d]	—
6	31	Neg	Abscess (9 d)	S [3 d]	Pred (75 mg) [8 d]	Amp, Str, Metro
7	76	Pos(i) [2 hr]	Abscess (1 mo)	C [6 mo]	—	—
8	31	Pos(i) [2 hr]	Abscess (2 d)	S [2 d]	—	Amp, Metro
9	62	Neg	Infarct (18 d)	C [6 mo]	—	—
10	43	Neg	Recent hematoma	C [8 mo]	—	—
11	70	Pos(w) [20 hr]	Gliomas (3 wk)	S [30 d]	Dxm (12 mg) [3 d]	Pen, Metro
12a	27	Pos(w) [2 hr]	Glioma (2 wk)	S [20 d]	—	Pen, Metro
12b		Neg	(6 wk)	S [10 d]	Pred (40 mg) [1 mo]	—
13	56	Neg	Glioblastoma (1 mo)	S [7 d]	Dxm (24 mg) [9 d]	—
14	54	Neg	Glioma (1 mo)	S [7 d]	—	—
15	54	Neg	Metastasis (1 mo)	S [5 d]	Pred (75 mg) [1 d]	Amp, Genta
16	48	Neg	Metastases (24 d)	A [30 d]	—	Pen
17	64	Neg	Glioma (6 d)	S [12 d]	Dxm (24 mg) [5 d]	Amp, Metro
18	60	Neg	Metastasis (7 d)	S [1 d]	—	—
19	45	Neg	Glioblastoma (2 mo)	S [3 d]	Pred (100 mg) [8 d]	—
20	59	Pos(w) [2 hr]	Metastasis (3 mo)	S [9 d]	—	—
21	60	Pos(m) [4 hr]	Metastases (2 mo)	S [7 d]	Dxm (24 mg) [7 d]	Amp, Genta, Metro
22	43	Pos(w) [2 hr]	Gliomas (1 mo)	S [3 d]	—	—
23	63	Pos(w) [20 hr]	Glioma (13 d)	S [7 d]	Dxm (24 mg) [2 d]	—
24	80	Neg	Glioblastoma (2 mo)	A [8 mo]	—	—
25	69	Pos(m) [2 hr]	Glioblastoma (3 wk)	S [1 d]	Dxm (24 mg) [3 d]	—
26	68	Pos(m) [4 hr]	Glioblastoma (1 mo)	S [2 d]	Dxm (24 mg) [1 d]	—
27	70	Pos(w) [20 hr]	Glioblastoma (6 mo)	S [11 d]	Dxm (6 mg) [2 mo]	—
28	29	Neg	Metastasis (1 mo)	S [5 d]	—	—

<sup>\*</sup> Scintigraphically assessed degree of abscess and tumor accumulation of <sup>111</sup>In: w = weak, m = moderate, i = intense. The time of <sup>111</sup>In GS turning positive (min/h postinfection) is shown in square brackets.

<sup>†</sup> Duration of disease shown in bracket (difficult to assess in patient 10).

<sup>‡</sup> C = clinical and sequential CT-studies (with follow-up period in square bracket). S = surgery, A = autopsy. (Time from <sup>111</sup>In-GS to invasive diagnosis in bracket).

<sup>§</sup> Dxm = dexamethasone, Pred = prednisolone (daily dosage and duration of therapy at the time of <sup>111</sup>In-GS in brackets). Patients 4a, 4b, and 8 received 24 mg Dxm until 1–2 days prior to <sup>111</sup>In-GS.

<sup>¶</sup> Amp = ampicillin, Metro = metronidazole, Pen = penicillin, Str = streptomycin, Chl = chloramphenicol, and Genta = gentamicin.

## RESULTS

Table 1 summarizes our results. The final diagnosis was brain abscess in 8 patients, brain tumor or metastasis in 18 patients, and infarct and hematoma in 1 patient each. Non-beta-hemolytic streptococci were cultured from three abscesses, peptococcus from one abscess. Patient 4b developed a large cerebral infarct in addition to an abscess. Two abscess patients (Nos. 4 and 5) were studied twice, as was a patient with a cerebral tumor (No. 12). These patients showed focal  $^{111}\text{In}$  uptake when not on steroid therapy (patient 12a) or on low-dose steroid therapy (Patients 4a and 5b), whereas no uptake was seen when they were on high- or medium-dose therapy (4b, 5a, and 12b) (Figs. 1 and 2). One additional steroid-treated abscess patient (No. 6) did not accumulate  $^{111}\text{In}$  in an abscess containing numerous neutrophils on microscopic examination (around 40% being morphologically intact). The remaining five abscesses in patients not treated with steroids showed intense  $^{111}\text{In}$  accumulation (Fig. 3). To summarize, three abscesses in patients treated with a daily dexamethasone dose of 8 mg or more, or an equipotent prednisolone dose (14) did not accumulate  $^{111}\text{In}$ , whereas all seven abscesses in patients receiving 4 mg dexamethasone or less per day, or not on steroid treatment, took up  $^{111}\text{In}$  avidly.

Weak focal  $^{111}\text{In}$  accumulations were recorded in six tumors, moderate activity was seen in three (Fig. 4). Six of the nine patients showing tumor uptake were on

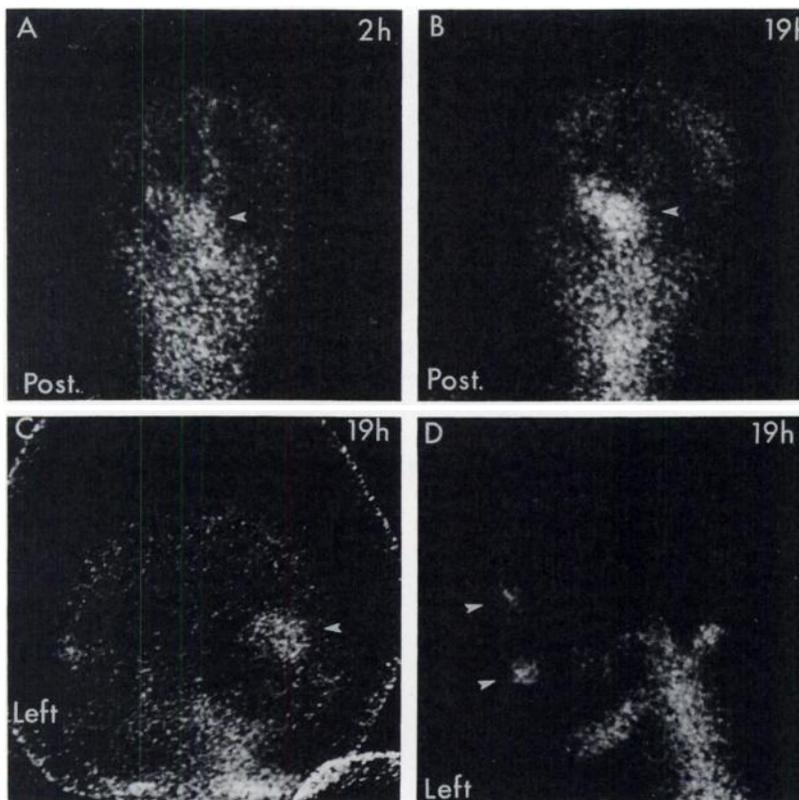
steroid treatment. Of the ten patients without tumor visualization, five received steroids. Two tumors (without  $^{111}\text{In}$  uptake) were cerebellar, the remainder being located in the cerebral hemispheres. The hematoma and the infarct did not take up  $^{111}\text{In}$ .

The abscesses tended to accumulate  $^{111}\text{In}$  more rapidly and with considerably greater intensity than did the tumors. Even if steroid therapy appeared not to affect the outcome of  $^{111}\text{In}$ -GS in the tumor group, it seemed to influence the speed of  $^{111}\text{In}$  accumulation, as can be seen in Table 1.

Table 1 results indicate antibiotic treatment did not seem to influence the result of  $^{111}\text{In}$ -GS.

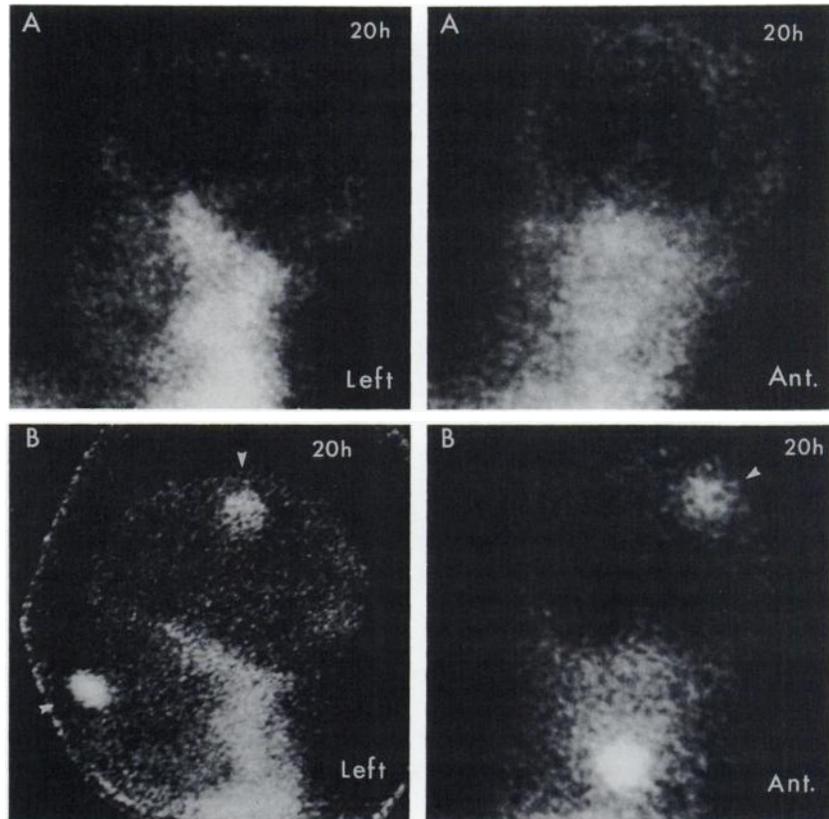
We had the opportunity to observe the effect on  $^{111}\text{In}$ -GS of recent burr-hole aspiration or craniotomy performed 4–19 days prior to scintigraphy in five patients (not treated with steroids). Rather intense activity was seen corresponding to two burr holes made four and seven days prior to  $^{111}\text{In}$ -GS and to a scar, following craniotomy performed nine days before  $^{111}\text{In}$ -GS. Two craniotomy scars (surgery 11 and 19 days before  $^{111}\text{In}$ -GS) did not accumulate  $^{111}\text{In}$ .

We did not find the microscopic granulocyte score (17 tumor samples studied) or the combined granulocyte/vessel proliferation/hemorrhage score (12 samples) to correlate with the scintigraphic  $^{111}\text{In}$  uptake score, the correlation between  $^{111}\text{In}$  uptake and granulocyte infiltration score reaching a borderline significance, however ( $r = 0.42$ ,  $0.05 < p < 0.10$ ), Spearman's



**FIGURE 1**

Patient 4. A 27-yr-old woman suffering from a congenital heart disease with right-to-left shunt, now presenting with a surgically confirmed central abscess in the left parieto-occipital region, preceded by culture-negative meningitis 3 mo previously. The scintigrams A, B, and C were obtained 3 weeks prior to scintigram D. Seventeen and 7 days prior to the last scintigraphic study, respectively, 8 and 4 ml of pus had been obtained by aspiration. The first study shows early abscess uptake of  $^{111}\text{In}$  (dexamethasone dose: 4 mg/d), the late study (scintigram D) does not at all (dexamethasone dose: 8 mg/d). It shows uptake, however, corresponding to the frontal sinus and the nose (no equivalent overt clinical signs of infection). She expired 5 days after the late study (4 days prior to which she had developed a large infarct in the left hemisphere). At autopsy, the infarct and the previously diagnosed abscess were seen. The abscess contained numerous granulocytes. The nose and frontal sinus were not examined.



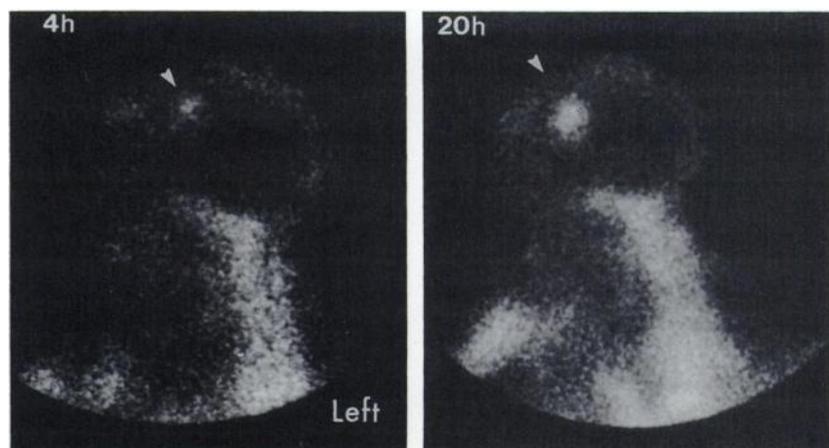
**FIGURE 2**  
 Patient 5. A 71-yr-old woman with clinical and CT signs of an abscess in the left fronto-parietal region. The time interval between the upper (A) and lower (B) scintigrams was 3 mo. At the time of the early (A) study, which shows no focal <sup>111</sup>In uptake, she received dexamethasone, 24 mg/d. This dose was gradually tapered off to 2.5 mg prednisolone per day at the time of the late (B) study, which shows abscess <sup>111</sup>In uptake (arrow), in addition to uptake corresponding to dental abscesses requiring subsequent tooth extractions. She made an uneventful recovery during a 2-yr observation period.

rank sum test). Five tumor diagnoses were based on microscopic examination of smear preparations, making impossible in these a semiquantitative assessment of hemorrhage and vessel proliferation. In addition, a patient (No. 24), in whom the time interval between <sup>111</sup>In-GS and autopsy was 8 mo, was excluded, leaving the 17 and 12 samples, respectively, for analysis. All abscesses contained numerous granulocytes.

The erythrocyte sedimentation rate and neutrophil count in peripheral blood were available in most patients. They were not able in any way to discriminate between abscesses and tumors.

## DISCUSSION

The encapsulated cerebral abscess, preceded by the cerebritic, presuppurative phase of ~8–10 days' duration, typically is a trilayered structure with a necrotic core containing degenerating neutrophils, and surrounded by a layer of morphologically intact neutrophils, external to which is a vascularized zone of granulation tissue containing inflammatory cells of different types, and varying amounts of collagen, depending on the age of the abscess. These structures are surrounded by vascularized gliotic reactive brain tissue adjacent to



**FIGURE 3**  
 Patient 8. Scintigrams from a 31-yr-old woman showing <sup>111</sup>In uptake of increasing intensity from 4 hr to 20 hr postinjection, corresponding to a surgically confirmed abscess in the left fronto-parietal region. She was not treated with steroids.



**FIGURE 4**  
 Patient 21. A 60-yr-old man with multiple processes in the right hemisphere, showing ring contrast enhancement on CT scans. An open biopsy (7 days after the  $^{111}\text{In}$ -GS study) neither revealed tumor cells nor showed signs of bacterial growth. Microscopic granulocyte score: 1. The scintigram shows irregular  $^{111}\text{In}$  uptake (arrows) in the right hemisphere. Dexamethasone dose: 24 mg/d. He deteriorated slowly and died 5 mo later. Autopsy revealed multiple metastases from an adenocarcinoma. No primary tumor was found.

the surrounding edematous brain substance (15–17). As the abscess grows older, the neutrophil response in the necrotic center declines (18).

CT scanning is a valuable diagnostic modality in brain abscess diagnosis. With repeated CT studies, it is possible to follow the evolution and resolution of brain abscesses and to determine the appropriate timing for surgical intervention (2,5,19). However, it may be difficult on CT to differentiate between suppurative and neoplastic processes, and the CT findings in the presuppurative stage of abscess evolution may be atypical (5). Indium-111-GS would thus a priori seem a valuable method in this diagnostic situation. The application of  $^{111}\text{In}$  GS in brain abscess diagnosis has been reported sporadically (20,21) and described in greater detail in two published studies (8,9). Rehncrona et al. (8) observed  $^{111}\text{In}$  uptake in four of five brain abscesses (all treated with betamethasone, 12–16 mg/d) imaged 24 and 48 hr postinjection. Bellotti et al. (9), who used the activity of the base of the skull as reference, i.e., suggested only very intense accumulation of  $^{111}\text{In}$  activity to represent abscess formation, claimed a sensitivity of this method for brain abscess detection of 100% and a specificity of 94%. By this method of image interpretation, two tumors showing  $^{111}\text{In}$  uptake were “negative.” Two  $^{111}\text{In}$  accumulating encapsulated abscesses apparently did not take up activity when previously

studied in the cerebritis stage, and in two positive abscesses restudied two weeks after surgical aspiration neither took up  $^{111}\text{In}$  (no invasive confirmatory studies were performed at that time). Thus, the diagnostic accuracy may be somewhat lower than claimed by the authors. No mention was made of steroid therapy.

Our previous results of  $^{111}\text{In}$ -GS for the detection of soft-tissue inflammation (7), and the fact that all abscesses in the present study would be expected to be encapsulated, one of them accumulating  $^{111}\text{In}$  after 3.5 mo, testifies to the sensitivity of this method. Accordingly, our results suggest that  $^{111}\text{In}$ -GS may be a useful adjunct to CT in brain abscess diagnosis. There are some obvious pitfalls and shortcomings, however. Most important, our study shows that cerebral abscesses in patients on high-dose steroid treatment may not be detected by  $^{111}\text{In}$ -GS. To our knowledge, this is the first in vivo demonstration in humans of what has been suspected from animal experiments. Quartey et al. (22) in rabbit experiments found dexamethasone to impede bacterial killing in brain abscesses in animals on antibiotic treatment. This was accompanied by sparse infiltration by granulocytes and compromised granulation tissue and fibrous capsule formation. Neuwelt et al. (17) found high- and medium-dose dexamethasone to suppress macrophage and glial response and to decrease collagen formation. The neutrophil response apparently was unaffected. The results of their study are somewhat difficult to interpret, however, because of simultaneous antibiotic treatment which in this early cerebritic phase suppressed abscess formation very effectively. Schroeder et al. (18) did not find dexamethasone to affect the inflammatory response in experimental brain abscesses in rats. The only effect observed was a delay in collagen deposition in the abscess wall.

Corticosteroids suppress inflammation by impeding the access of neutrophils and monocytes to an inflammatory site (23). They can inhibit granulocyte adherence (24,25), suppress the production of chemoattractants derived from macrophages (26), block the binding of certain chemotactic agents to the granulocyte surface (27), and prevent the release of granulocyte activating granule constituents from granulocytes (28). In addition, they may reduce the permeability of the capillary endothelial cells (29). That corticosteroids may affect the blood-brain barrier is further evidenced by the decreased ring enhancement on postcontrast CT scans (5).

The abscesses of two patients on high-dose steroid (4b and 6) did not accumulate  $^{111}\text{In}$  despite microscopic demonstration of neutrophil infiltration. In Patient 4b, this discrepancy may be explained by cessation of steroid therapy three days prior to her death. Other explanations could be a greatly prolonged granulocyte turnover across the abscess membrane (in which case the finding of a larger number of disintegrating neutrophils

would have been expected) or a modification of granulocyte function induced by the in vitro manipulations, making them particularly susceptible to the in vivo effects of corticosteroids.

Considering the 1–2-day biologic half-life of dexamethasone (14), and the fact that the steroid effect on the inflammatory response may last for days (30), it seems remarkable that patients on high-dose dexamethasone until 1–2 days prior to <sup>111</sup>In-GS (Patients 4a and 8), showed intense <sup>111</sup>In abscess uptake. However, a shorter duration of antiinflammatory effect of steroids has been reported (23). The practical consequence of our findings is that the diagnostic accuracy of <sup>111</sup>In-GS in brain abscess diagnosis may be “restored” by a 1–2-days reduction in steroid dosage prior to scintigraphy.

The previously described tumor visualization during <sup>111</sup>In-GS (12,31,32) also applies to brain tumors (8–10,12,33). The results of the present study suggest this phenomenon to be more frequent than previously thought. The mechanism by which tumors accumulate <sup>111</sup>In granulocytes may be different from that of abscesses. The typical abscess pattern is that of an early accumulation of activity of increasing intensity (7). In contrast, the tumor-associated activity remained of weak or moderate intensity throughout the imaging period. Besides, steroid therapy did not affect the frequency of <sup>111</sup>In accumulation in tumors. However, it did seem to delay their visualization. Intense early <sup>111</sup>In uptake in a vascularized cerebral tumor, decreasing in intensity in the imaging period, was described recently (10).

We found a rather poor correlation between the intensity of <sup>111</sup>In activity in tumors and the corresponding microscopic granulocyte score (whether supplemented with tumor vessel proliferation and hemorrhage scores or not). Among several possible explanations, one could be the inevitable fluctuations in steroid dosage in the period lapsing between scintigraphy and microscopy.

Early diagnosis of brain abscesses must be the goal before the establishment of a core of necrosis that may be inaccessible to the effect of antibiotics (34). In the cerebritis stage, <sup>111</sup>In-GS may be a useful adjunct to CT, which means that its inclusion in the diagnostic work-up should be considered early in patients with focal cerebral lesions at a time when corticosteroid therapy may be indicated to control the mass effect of the abscess and its associated edema, and the omission of concomitant antibiotic therapy could have detrimental consequences. In the distinction between an encapsulated abscess and other space occupying brain lesions, an intense accumulation of <sup>111</sup>In definitely favors the former diagnosis. Diagnostic problems may arise, however, in steroid-treated patients showing no or only modest <sup>111</sup>In uptake. With these shortcomings in mind, <sup>111</sup>In-GS undoubtedly may add to the quality of non-

invasive brain abscess diagnosis, being of value especially in cases where surgery is deferred.

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