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Tc-99m HMPAO Brain SPECT for ischemic strokes

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= 국문초록 =

Tc-99m HMPAO Brain SPECT의 장점은 핵의약품인 HMPAO을 항시 보관하고 있으면 모든 핵의학 laboratory에서 필요시 언제든지 labelling 하여 쉽게 사용할 수 있고 또 Tc-99m의 favorable physical character 때문에 high quality, high resolution imaging을 얻을 수 있다는데 있다. Control group으로서 8명의 nonischemic neurologic 증상을 호소하는 환자와 15명의 neurosis환자를 택했으며 ischemic stroke 환자 38명을 대상으로 Tc-99m HMPAO Brain SPECT slice image에서 양측을 비교하여 얼마만큼의 activity count의 저하(decrease % of activity : DPA)가 ischemic stroke을 진단하는데 필수적인가를 관찰하였다. Control group 중에서 nonischemic neurologic symptoms을 호소하는 환자군에서는 DPA가 5.7±4.14%(mena±s.d.)였고 neurosis 환자에서는 DPA가 10.3±2.3%(mean±s. d.)였다. 이상과 같이 DPA 15% 이상에서는 abnormal DPA(정상 DPA±2 s.d. 이상)로 간주하였다. 모든 ischemic stroke 환자는 DPA 15% 이상을 나타내어 ischemic stroke의 진단율은 100%이다. DPA 20% 이상을 ischemic stroke을 진단하는 기준 DPA로 간주하는 것이 적당한 것을 발견하였으며 TIA인 경우에는 임상적인 정보와 종합하여 DPA 15% 이상인 것을 진단의 기준으로 삼는 것이 좋지만 nonischemic disorder인 dehydration, cerebral atrophy, neuropsychiatric disorder 및 subarachnoid hemorrhage 등에서 발생할 수 있는 vascular spasm 등의 가능성도 생각해야만 할 것이다. Complete stroke인 경우에는 대부분 DPA 30% 이상을 나타내며 DPA 30% 이상일 때는 진단된 ischemic stroke는 complete stroke인 것을 발견하였다.(예건을 90%) 그래서 DPA 30% 이상은 ischemic stroke 환자의 예후판단에 임계점으로 사용할 수 있을 것이다. Complete stroke이 Brain atrophy와 동반되어 있을 때 특히 Brain atrophy가 오래전부터 계속 반복되는 ischemic strokes에 의해서 나타낼 때는 ischemic stroke에 의한 hypoperfusion이 뚜렷하지 않고 낮은 DPA을 나타내었다. 그 이유는 반대쪽 혹은 주위 뇌조직의 atrophy 때문에 뇌혈류의 저하와 atrophy된 뇌조직의 glutatione의 양의 감소 때문에 HMPAO의 분포저하를 나타내기 때문으로 생각되었다. Tc-99m HMPAO Brain SPECT는 뇌의 허혈성 혈

관질환의 진단에 있어서 다른 진단 방법인 X선 전산단층촬영술(X-raya CT)와 뇌혈관조영술(invasive digital angiography)과 비교하여 훨씬 우수한 것을 발견하였으며 X-ray CT의 진단율은 46.4%였고 invasive digital angiography의 진단율은 58.8%였다.

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= Abstract =

Major advantage of Tc-99m HMPAO SPECT was easy availability at every Nuclear Medicine laboratories and high quality of images by many favorable character of Tc-99m labeled agent. 8 patients of non ischemic neurologic disorders and 15 patients of neurosis as controls and 38 patients of ischemic strokes were studied for how much decrease % of activity (DPA) is abnormal for ischemic strokes. Mean DPA of neurologic control compared to contralateral side was 5.7±4.14% and mean DPA of neurotic control patients was 10.3±2.3%.

Higher than DPA 15% was counted as abnormal (above mean value+2 s.d.) .

Detection sensitivity of ischemic strokes was 100%. DPA more than 20% was adequate for prediction of ischemic strokes with acceptable false negative rate and DPA more than 15% for TIA with expected higher false positive rate. DPA more than 30% was prone to be complete strokes (prediction 90%) and would be critical level for prospect of prognosis. The complete strokes with cerebral atrophy and/or by old infarcts were prone to show the hypoperfusion less contrast to have DPA less than 20%. Tc-99m HMPAO SPECT was found to have much superiority in detection of ischemic strokes, compared to XCT (detection rate : 46.4%) and invasive digital angiography (detection rate : 58.8%) .

Key Words : Tc-99m HMPAO, Brain SPECT, ischemic strokes

INTRODUCTION

Relatively accurated quantitative mapping of regional cerebral blood flow has been possible by expensive, specially designed SPECT machine and using inert gas Xe-133.[12]

Tomographic images of Xe-133 using conventional SPECT system is not possible due to its slow count acquisition rate and rapid clearance of Xe-133 from brain.

Recently I-123 IMP(N-isopropyl-(123-I) iodoamphetamine) and HIPDM(N,N,N, trimethyl-N,

12-hydroxy-3-methyl-5-(I-123) iodobenzyl-1,3, propane diamine) have been widely used as brain SPECT agents due to their favorable characters for measuring regional cerebral blood flow in human.[3,7,9,10,15,16]

However, the major disadvantage of I-123 amines are expensive and not readily available in time whenever it is needed.

Newly developed Tc-99m labeled neutral lipophylic agent HMPAO(d,1,form of Hexamethyl propyleneamine oxime) has none of these disadvantages and is good promising agent for brain SPECT in mapping regional perfusion of brain because it deposits in brain proportional to blood flow and its deposit is quite constant during performance of SPECT.[13,24]

Several studies reported that the Tc-99m HMAPO SPECT are matching well to that of others(Xe-133, I-123-IMP and HIPDM) although minor disadvantages were reported.[1,18,19]

The high resolution SPECT imaging could be achieved by Tc-99m labelled HMPAO due to its advantages of physical characters and improved counting rate by administration of large doses.[4]

Normal distribution of Tc-99m HMPAO and side/side differences of counting rate and several clinical experiences of SPECT using Tc-99m HMPAO for various brain pathologies have been reported.[21,22]

The significance of decrease percentage of activity in SPECT using I-123 IMP in comparison with normal contralateral side has been reported in correlation with detection sensitivity of ischemic pathologies extending to prospect and prognosis of these ischemias.[15]

To define the sensitivity of decrease percentage of activity for visualizing changes of regional brain blood flow correlated to ischemic pathologies, our initial clinical experience of Tc-99m HMPAO Brain SPECT is outlined.

MATERIAL AND METHOD

38 patients of ischemic strokes were studied.

Among 38 patients of ischemic strokes 9 patients had transient ischemic attack (TIA), 3 had prolonged reversible ischemic neurologic deficit (RIND) and 26 had complete stroke(CS) resulting infarct. Table 1 shows time interval between various brain studies and ischemic strokes. 8 patients who had non-ischemic neurologic disorders and 15 patients of neurosis as

Table 1. Time of Brain studies after attack

Study time after attack	number of patients		
	99mTc-HMPAO spect	X-CT	Angio
1 day	4	15	3
2 days	3	2	0
3 days	4	1	2
1 week	3	2	2
2 weeks	5	1	1
3 weeks	3	1	0
8 weeks	3	0	0
6 months	1	0	0
3 years	1	0	0
Total	27*	22	8

*one patient had two studies

seen in Table 2 were selected as control groups.

All were at rest recumbent with eye close. Tc-99m HMPAO was injected i,v,as a bolus of 5 ml with an activity of 15mCi(555 Bq) immediately after the commercial kit of HMPAO(ceretec) was labeled by 99mTc'04. Data acquisition started at 20-60min. after i,v,injection of Tc-99m HMPAO on single detector rotating gamma camera using LEAP collimator and dedicated gamma 11 computer. The patient's head was fastened by elastic bandage to scaning table. The detector of gamma camera was adjusted with 10 degree cephalocaudal angle to close the patient's head and shoulder to improve spatial resolution. The SPECT study was required 25 min. with 6 degree stepwise rotation in 60 projection and data collection time of each step required 30 sec. for accumulating more than 200K counts. The data acquisition starts on posterior projection. The data acquisition was carried out by using 64×64 word mode. Reconstruction was performed by filtered back projection method employing shepp-Logan No 2 shaper filter for keeping better resolution. The attenuation collection was not used. Following reconstruction of transverse images, images were mirrored and matrix size expanded from 64×64 to 128×128 by 2 times interpolations. The coronal and sagittal slice images were reconstructed from raw data of transverse reconstruction images and image size was expanded to 128×128 matrix size by 2 times interpolations. Image interpretation was done as decrease % of activity (DPA) in each slice in comparison with normal contralateral side and ipsilateral normal region by visual estimation of color coded images. In 15 cases of neurotic patients in the control group both DPAs by computer calculation in ROI of maximal hypoperfusion seen on SPECT slices and by visual estimation of color coding of perfustion activity were compared as seen on Table 2. The visual estimation of color coding was found to have less standard deviation, therefore subsequent interpretation of cases of ischemic strokes was performed by visual estimation of color coding.

RESULT

In the control groups, among 8 patients who had non ischemic neurologic disorders 5 patients showed less than 5% DPA(decrease percentage of activity) and 3 showed 10%, 15% and 20% each. However 15% & 20% DPA were seen in severely dehydrated and mentally stuporous patients. One patient having 20% DPA exhibited atrophy on XCT(X-ray computerized tomography) that was excluded on study. Patients of neurosis that showed DPA less than 10% in the area of hypoperfusion, were 13 and remainder showed DPA 15%. Mean DPA of neurotic patients was 10.3± 2.3(mean ±s.d.). The DPAs by visual estimation in color coded images better lesion to normal contrast while the calculation of rt./lt.differences by drawing regional region of interest(ROIs) in computer was less lesion contrast due to volume average in count density as expected and seen on Table 2. Mean value of DPA in the control of nonischemic neurogic disorders was 5.7±4.14%. DPA more than 15% was counted to be abnormal (above mean value ± 2 s.d.) Among 38 ischemic strokes as Table 3, TIA showed 15−30% DPA in 2 of 9 patients(22.2%) as seen in fig. 2 A-B. In 3 RIND patients 15−20% DPA in 7 out of 9 patients (77.8%) as seen in fig. 1 and 20 −30% DPA in 2 of 9 patients(22.2%) as seen

Table 2. Summary of Data in the Controls(Neurosis)

Case No	Age/ Sex	Diagnosis psychiatric	Site of Maximal decreased	Activity abnormal	Count/ROI normal	Decreased% computer calculation	Activity(DPA) estimation by color code
1	25/F	conversion disorder	bilateral post. vertex	post.part : 10209	ant.part : 12638	19.2%	15%
2	22/F	conversion disorder	Lt.vertex	Lt.side : 11048	Rt.side 12102	8.7%	10%
3	40/F	conversion disorder	bilat.inferior temporal	Lt.ant : 10325	Lt.post : 10393	6.1%	10%
4	30/F	conversion disorder	Lt.parietal	Lt.7213	Rt.8618	16.3%	15%
5	48/M	anxiety disorder	Lt.fronto-temporal	Lt.9914	Rt.11670	15.0%	10%
6	33/F	anxiety disorder	Lt.parietal	Lt.9061	Rt.10036	9.7%	10%
7	39/M	anxiety disorder	Rt.post. vertex	Rt.post : 13048	Lt.ant : 14400	9.4%	10%
8	13/M	conversion disorder	Lt.post. parietal	Lt.post : 15932	Rt.post : 18461	13.7%	10%
9	34/F	conversion disorder	Rt.temporal	Rt.14012	Lt.15637	10.4%	10%
10	59/F	conversion disorder	bilat.parietal	Lt.temp : 7022	Lt.parietal : 7569	7.2%	5%
11	36/M	anxiety disorder	bilat.parietal	Rt.ant. parietal : 7291	Rt.mid parietal 8301	12.2%	10%
12	19/F	dysthymic disorder	Lt.temporo-frontal &	Lt.8845	Rt.9469	6.6%	10%
13	10/M	conversion	Rt.posterior	Rt.10844	Lt.11603	6.5%	10%
14	10/M	conversion disorder	Lt.vertex	Lt.11603	Rt.11844	8.6%	10%
15	30/M	conversion	bilat.post.	post : 7695	ant : 9380	18.0%	10%
						11.2±4.3 (mean±s.d.)	10.3±2.3 (mean±s.d.)

Table 3. DPA of Tc-99m HMPAO perfusion in ischemic strokes

	DPA(decreased % of activity)		
	15%−20%	20%−30%	more than 30%
TIA	*7/9(77.8%)	2/9(22.2%)	0
RIND	1/3(33.3%)	0	2/3(66.7%)
Complete stroke	*5/26(19.2%)	*2/26(7.7%)	*19/26(73.1%)
Total ischemic disease	13/38(34.2%)	2/38(5.3%)	23/38(60.5%)

*with cerebral atrophy : 3 cases of TIA and 3 cases of complete stroke which have cerebral atrophy shows less than 20% decreased perfusion.

in fig. 2 A-B. In 3 RIND patients 15−20% DPA was seen in one(33.3%) and more than 30% DPA in 2 patients(66.7%). 26 complete strokes (CS) showed 15−20% DPA in 5 patients(19.2%), 20−30% DPA in 2 (7.7%) and more than 30% DPA as seen in fig. 3A in 19(73.1%). One of 15−20% DPA showed hypoperfusion in the large area of subcortical white matter with suggestion of functional decreased uptake in ipsilateral parietal cortex and one of 15−20% DPA was small hypoperfusion in external capsule of white matter as old infarct. 8 of 38 strokes exhibited cerebral atrophy on XCT with several times ischemic attacks clinically prior to this study. 6 patients of cerebral atrophy(75%) showed 15−20% DPA and was found to have old strokes. 2 cerebral atrophies seen in each patient of CS with 20−30% and more than 30% DPA were studied with persistent neurologic symptoms for 6 months and 3 years after initial attack.

Table 4 displays the detection sensitivity of ischemic strokes in various thresholds of DPA. The threshold of DPA greater than 15% revealed 100% detection sensitivity of ischemic strokes but false positive finding by non-ischemic neuro-psychiatric disorers, local vasospasm in subarachnoid hemorrhage or head injury was expected to be high. The threshold of DPA more than 20% showe detection sensitivity 22.2% of TIA, 66.7% of RIND and 80.8% of CS, and false finding 77.8% for TIA, 33.3% for RIND and 19.2% for CS. By the threshold of DPA more than 30%, 66.7% of RIND and 73.1% CS but non TIA were included. Table 5 exhibits DPA value for prediction of ischemic strokes. The prediction of TIA with DPA of 15 to 20% was 53%.

The prediction of neurologically positive stroke with DPA more than 30% was 91% and

Table 4. Detection sensitivity of ischemic strokes by various threshold of DPAs.

Brain disorders	DPA(decreased % of activity)		
	more than 15%	more than 20%	more than 30%
TIA	9/9(100%)	2/9(22.2%)	−
RIND	3/3(100%)	2/3(66.7%)	2/3(66.7%)
Complete Stroke	26/26(100%)	21/26(80.8%)	19/26(73.1%)
Total ischemic Brain disorders	38/39*(97.4%)	25/38(65.8%)	23/38(60.5%)

*1 case of false positive finding ; severe dehydration

Table 5. DPA value for prediction of ischemic stroke

	DPA		
	15%−20%	20%−30%	more than 30%
TIA	7/13(54%)	2/4(50%)	−
	9/17(53%)		
RIND	1/13(8%)	−	2/21(10%)
		* (84%)	@ (91%)
CS	5/13(40%)	2/4(50%)	19/21(90%)

*If DPA value more than 20% is taken for CS, prediction is 84%
@If DPA value more than 30% is taken for neurologically positive ischemic stroke,predictive value is 91%

Table 6. Comparative sensitivity for detection of ischemic disorders in other diagnostic procedures

Clinical DX	CT	digital angiography
TIA	0/5(0%)	2/5(40.0%)
RIND	0/1(0%)	0/0(0%)
Complete Stroke	13/22(59%)	8/12(66.7%)
Total	13/28(46.4%)	10/17(58.8%)

Table 7. XCT positive rate in time interval after attack for detection of complete stroke.

1 day after attack	:	1/15(6.7%)
2 days after attack	:	1/2 (50 %)
3 days after attack	:	5/5 (100%)

that of complete stroke 90%. In Complete stroke, old infarct and/with cerebral atrophy showed DPA value below 30%. Table 6 shows comparature sensitivity for detection of ischemic disorder by XCT and invasive digital angiography. XCT and invasive digital angiography were studied in few days before and after Tc-99m HMPAO SPECT in 5 out 7 TIA patients, only 2 patients showed evidence of TIA on angiography (40%) but no patients was positive on XCT. 22 out of 26 CS patients had XCT and 13 of these 22 patients(59%) exhibited hypodensity area. Table 7 shows positive rate of XCT in time interval after attacks for detection of complete stroke(CS). In first day after attack only 1 out of 15 patients was positive to show hypodensity and all 5 patients revealed positive in third day. Invasive digital angiography was done in 12 CS patients and 8 of these 12 showed evidence of vascular occlusion(66.7%).

12 out of 26 CS patients dipicted asymmetrical cerebellar activity and 50% of these 12 CS showed contralateral cerebellar hypoactivity. The cross cerebellar diaschisis was found in 6 out of 21 supratentorial CS(29%). 4 of 12 patients revealed focal more than 30% DPA indicating cerebellar infarct corresponded to neurologic findings. 7 of 26 CS dipicted focal more than 30% DPA in basal ganglia while 3 of them were seen on XCT, others were found to have associated infarct in the other areas on Tc-99m HMPAO SPECT. The regions of DPA in Tc-99m HMPAO were found to be larger and more than the regions of hypodensity found in XCT.(Fig. 3,4)

DISCUSSION

Tc-99m HMPAO is neutral lipophylic agent, able to cross the blood brain barrier and retained proportionally according to regional blood flow.[24]

It has advantages of stable deposit in brain, use of large dosage for better image quality and is readily available in time whenever it is needed while I-123 labeled amines do not. The autoradiographic study reported that the regional cerebral uptake pattern of Tc-99m-d, 1-HMPAO in comparison with the distribution of C-14 iodoantipyrine(IAP) is proportional to regional cerebral blood flow in rat brain and this finding could interpolate to human.[24,26]

Distribution of Tc-99m HMPAO compared to Sn-113-labeled microsphere in dogs found a close correlation.[25]

Comparison to cerebral blood flow measured by X-133, showed that 99mTc-HMPAO de-

posit does not exactly identical to CBF. The extraction efficiency of Tc-99m HMPAO measured 70% at very high flow and 90% at normal flow and deposit of the Tc-99m HMPAO was 40—50% of initial peak value of passage of bolus, the contrast between the regions of high and low flow is less than measured by Xe-133 because high blood flow region would have lowest retention because in the these regions back diffusion and clearance by efferent venous blood would be more effective.[1] The distribution of HMPAO in the brain decreases by approximately 10% over the first 10 min. and for following 5 hours it does not show significent changes but about 5% decrease of activity and slight less contrast between gray and white matter. The regional hypoperfusion with 10—20% decrease compared to the nonaffected area was reported not to be clearly visualized as a reduced perfusion in Tc-99m HMPAO image while the IMP imae demonstrated the ischemic area with better contrast.[19] The Tc-99m HMPAO images were reported to be similar in control patients compared to I-123 HIPDM and the flow defects were equally well detected by both, however Tc-99m HMPAO was superior to have less background by counting advantage.[13] The mechanism of the Tc-99m HMPAO deposit in brain differs from I-123 IMP, that IMP requires living brain cells in order to extract from blood but Tc-99m HMPAO is not brain cell specific and is depending to GSH(glutathione) level in tissues.[18] % of activity in white matter was measured 56% of gray matter at 1 hour and 58% at 5 to 6 hours.[13]

The absolute rCBF can not be expressed by Tc-99m HMPAO. In addition visual estimation of rt./lt. % difference of activity above 15% was considered as pathologic.[15,21] In our study detection sensitivity for ischemic stroke was very high, 100%, however we felt that the DPA less than 20% would have high false positive without clinical information because other functional hypoperfusion by neurologic and psychiatric disorders such as seizure focus, functional vasospasm and localized atrophy could exhibit similar DPA.

Higher than 20% DPA showed high false negative for TIA but relatively high detection rate, 80.8% for complete stroke. When DPA was found to be more than 30%, the prediction of complete stroke was estimated as 90% probability and probability of neurologically positive stroke was 91%. 30% DPA would be critical for prospecting prognosis.[15] RIND however displayed that the region of DPA was much smaller and in 2 cases of this study DPAs were in temporal operculum that DPA could be falsely aggrevated by irregularity of tissue. By less than 30% DPA ischemic stroke prefered to TIA, 53%. Old cerebral infarct with cerebral atrophy was more likely seen by less than 30% DPA, that reason is not clear but it could be suggested that areas of hypoperfusion were less contrasted by diffusely decreased blood flow in contralateral brain, shrinking areas of hypoperfusion and DPA[23] and less amount of GSH level in atrophic brain.[18]

Local hypoperfusion of white matter showed less DPA in contrast to low density of activity in contralateral or ipsilateral white matter that exhibits 56—58% of gray matter. Large infarct in subcortical white matter exhibited however absolute low density of activity with ipsilateral adjacent cortex showing functioning hypoperfusion as diaschisis.(fig 5.) No patient of TIA and RIND was depicted as a hypodense area of XCT. Only 59% of complete stroke patients was positive XCT, that matched to others.[6,28] It

is well known that regions of infarct start to be seen as hypodensity by edema on XCT between 24 hours and 72 hours after ischemic attack.[8] Fig. 4 shows the large area of hypoperfusion, more than 30% DPA involving rt. temporo-parietal and occipital region with an irregular small area of 20% DPA with contralateral parietal cortex at vertex, but CT(fig. 4B) show no hypodensity. We found 12 patients of complete stroke exhibiting asymmetrical cerebellum and cerebellar activity. 50% of these 12 patient had contralateral cerebellar hypoactivity, however only 6 out of 21 patients of supratentorial infarct was counted to have functinal cross cerebellar diaschisis, 29%[17]

Remainders were thought to be an anatomical variant of cerebellar size as normal functional dominance. 4 of 12 patients revealed focal more than 30% DPA were diagnosed to be cerebellar infarct corresponded to neurologic finding while only one of 4 cerebellar infarct was seen as a local hypodensity on XCT(25%).[5,11]

Isolated 3 infarcts in basal ganglia seen on XCT were clearly seen as a focal more than 30% DPA on Tc-99m HMPAO SPECT but 4 more patients exhibiting focal hypoperfusion on Tc-99m SPECT had associated infarct in other areas, those 4 basal ganglia infarct were not found on XCT, however hemorrhagic infarct from non hemorrhagic could not be differentiated on Tc-99m SPECT. As others[2,29] the areas of hypoperfusion in complete stroke were larger and more than those of hypodensity seen on XCT as seen on fig. 3A and 3B due to ischemic penumbra. Tc-99m HMPAO SPECT visualized reversible ischemic zone but CT showed only substantial change of encephalomalacia indicative of irreversible damage.

The Tc-99m HMPAO SPECT image was much superior to XCT and angiography in detection of ischemic strokes regardless of severity of disease as seen on table 6 and 7. In summary Tc-99m HMPAO SPECT shows promising high resolution perfusion tomographic imaging by conventional rotating gamma camera using high resolution collimator and large dosage of agent.[4] Major advantage of Tc-99m labeled agents is daily availability differe from I-123 labeled amines. Tc-99m labeled agents is daily availability differed from I-123 labeled amines. Tc-99m HMPAO SPECT seems to be superior to other methods(XCT and angiography) as non invasive screening and investigating of ischemic strokes. DPA more than 30% was critical for prospecting prognosis in Tc-99m HMPAO Brain SPECT in ischemic strokes.

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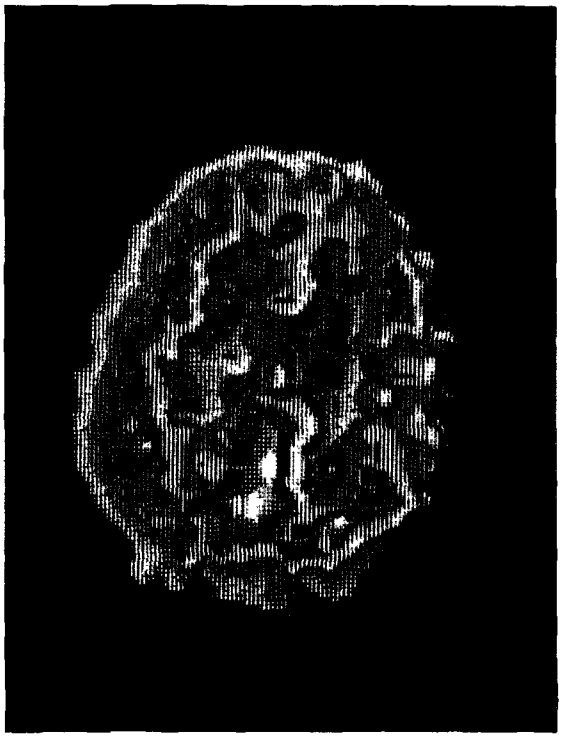


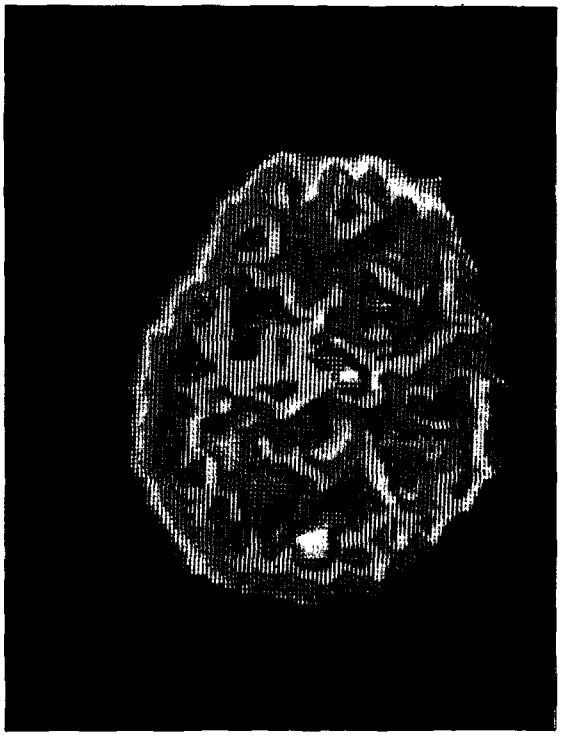


Fig. 1. A case of TIA : Hypoperfusion(DPA 15 to 20%) in lt. hemisphere at parietal region.

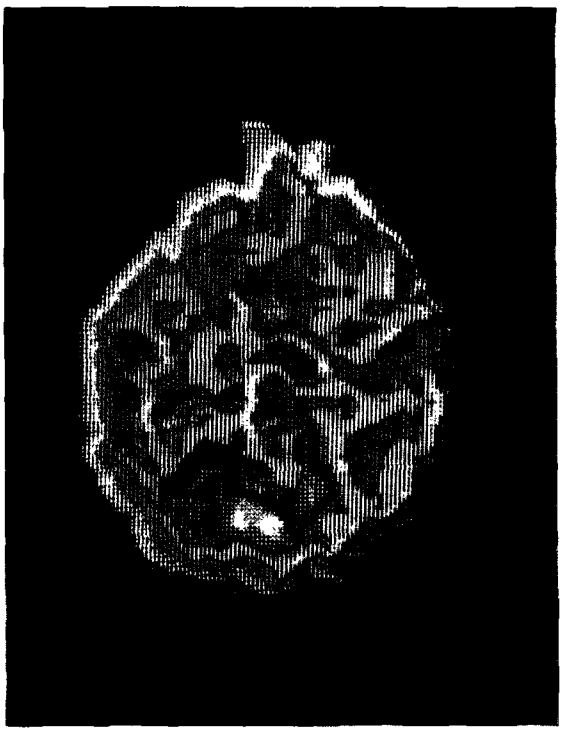


Fig 1

Fig. 2. TIA : Hypoperfusion in rt. temporoparietal region(DPA 20 to 30%)
64 years ol man had near total thyroidectomy and rt. neck dissection for thyroid papillary carcinoma with metastases to rt. nect being adherent to rt. carotid sheath. Subsequently I-131 therapy with 130mCi and electron beam radiotherapy due to no iodine uptake in the residual disease in rt. neck at rt. carotid sheath. After above therapy he had persistent intermittent dizzy spells with no neurologic focal & lateralization sign.
XCT was completely normal but 99mTc-HMPAO SPECT on next day of XCT depicted clearly hypoperfusion in entire right hemisphere, higher perfusion defect in rt. posterior temporoparietal region(A).
Dizziness has improved one year later and follow up 99m-HMPAO SPECT revealed improvement of hypoperfusion in rt. cerebral hemisphere(B).

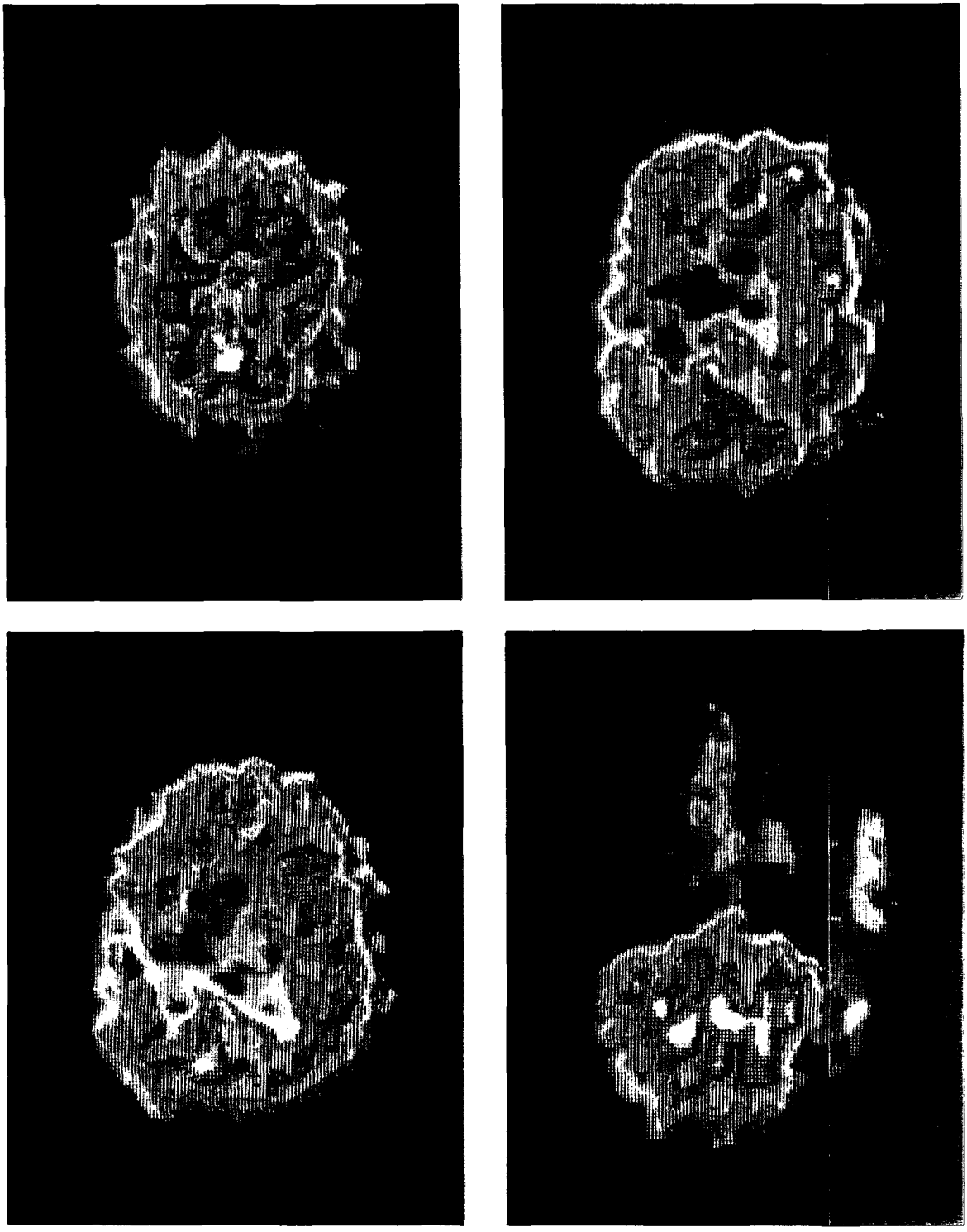


Fig 2-A

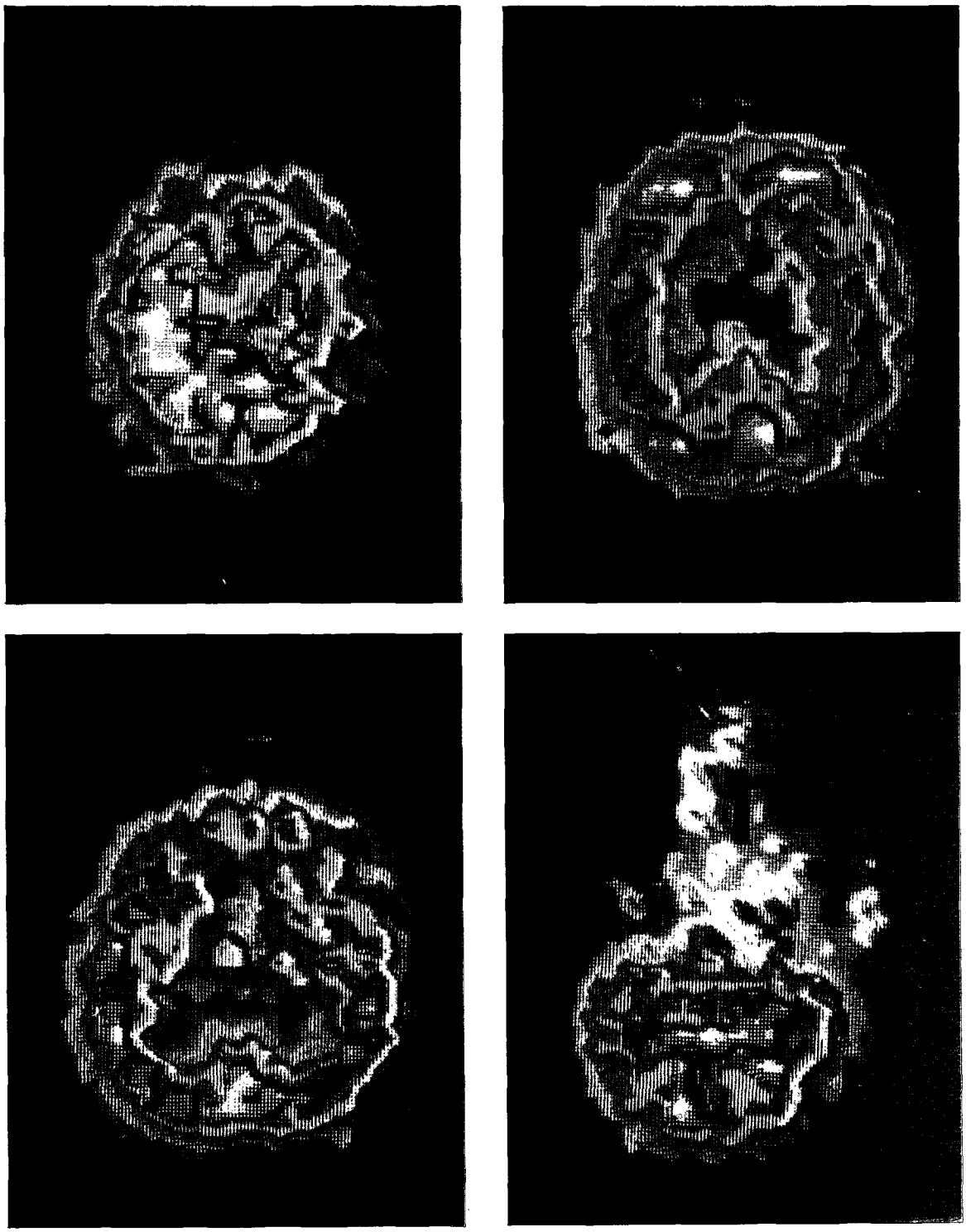


Fig 2-B

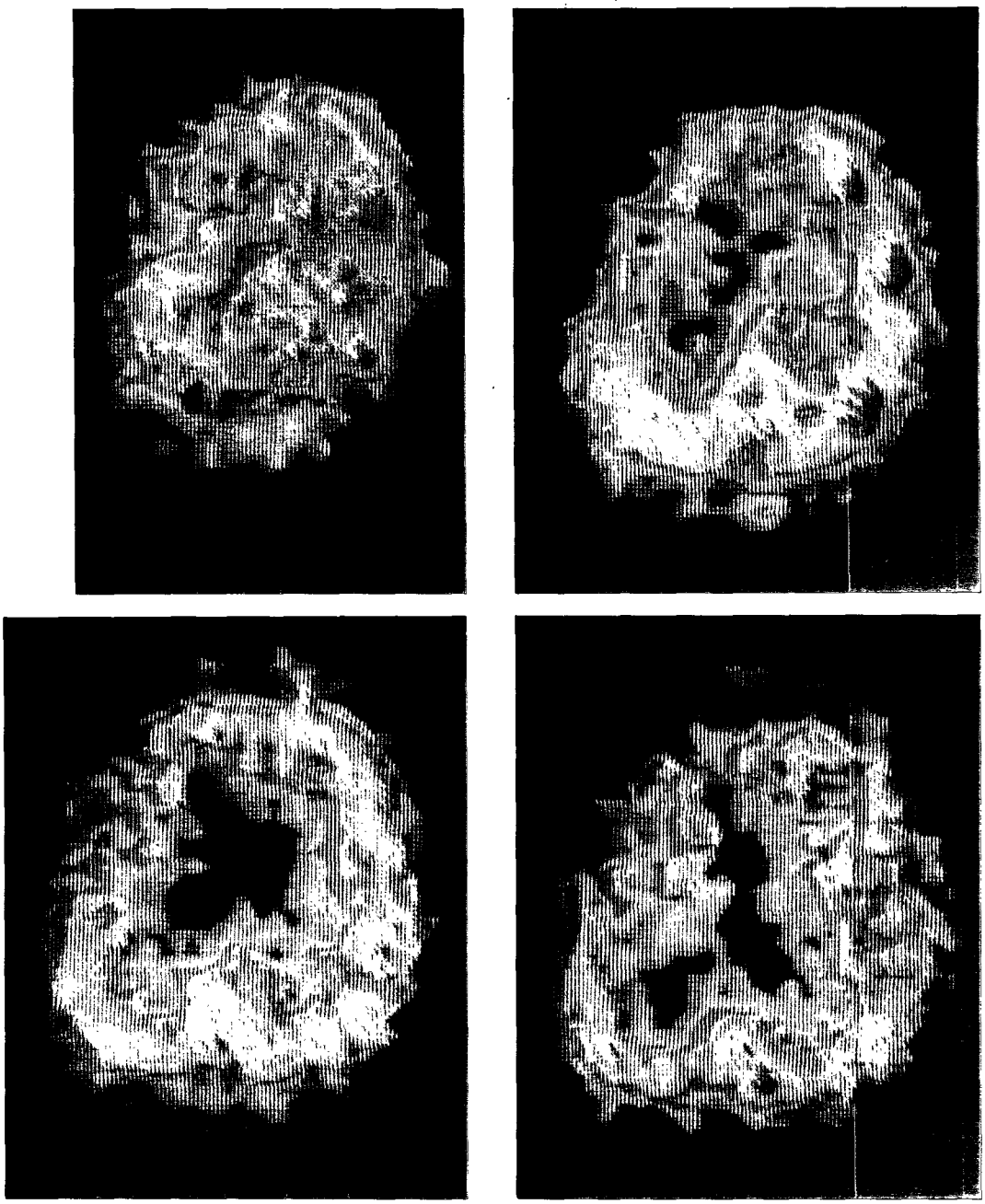


Fig. 3. A case of infarct : larger ischemic zone on Tc-99m-HMPAO and small irreversible infarct on CT.
68 year old hypertensive female(BP 160/110) came to emergency room with sudden attack of global aphasic rt. facial weakness and paralysis of rt. soft palate. She had been on somnolent state before attack. XCT was immediately obtained at ER and demonstrated illdefined hypodensity in lt. anterior temporal region extending to deep white matter and a focal hypodensity in rt. anterior parietal cortex(B).

99mTc-HMPAO SPECT on next day depicted irregular hypoperfusion, DPA more than 30%, in lt. anterior temporal region corresponded to the finding of XCT.
Surrounding larger ischemic hypoperfusion zone(DPA 20—30%) are noted(A).
The focal additional ischemic hypoperfusion is seen in lt. putamen.
Functional cross cerebellar diaschisis is noted in lt. cerebellum.

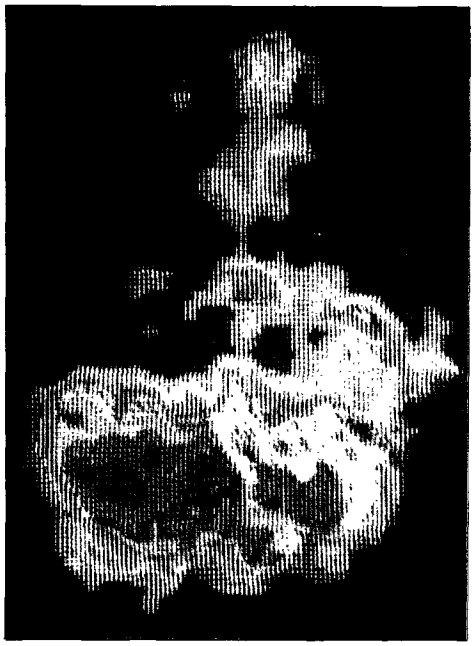
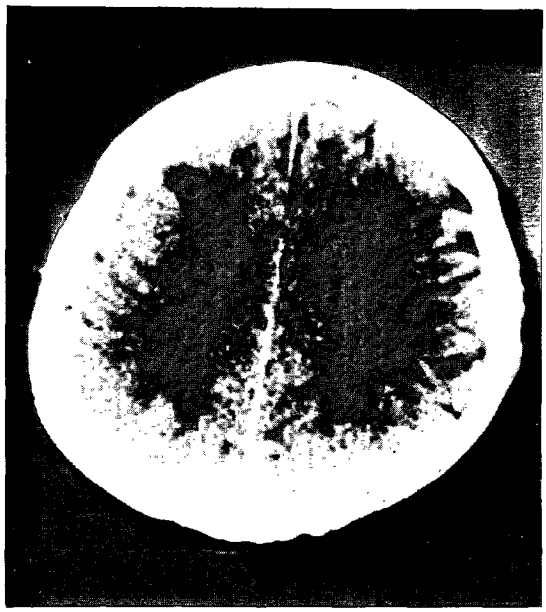


Fig 3-A



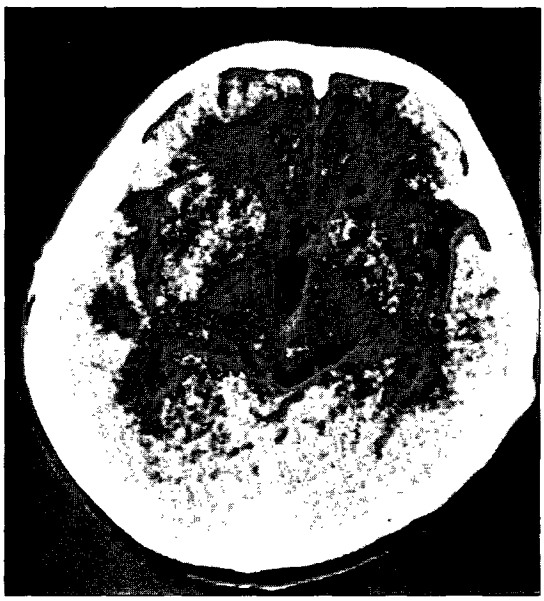
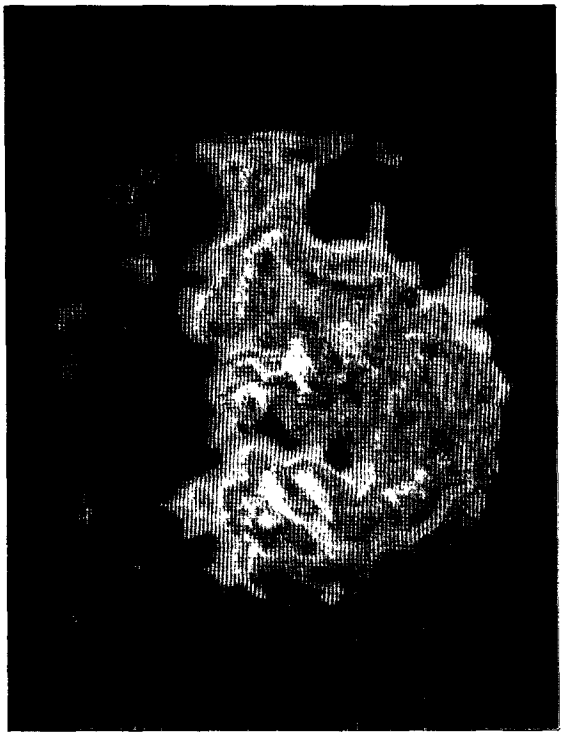
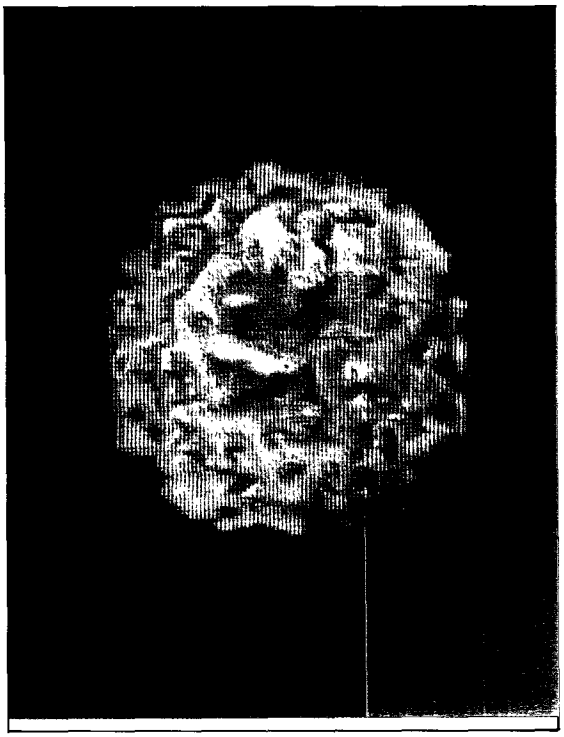


Fig 3-B





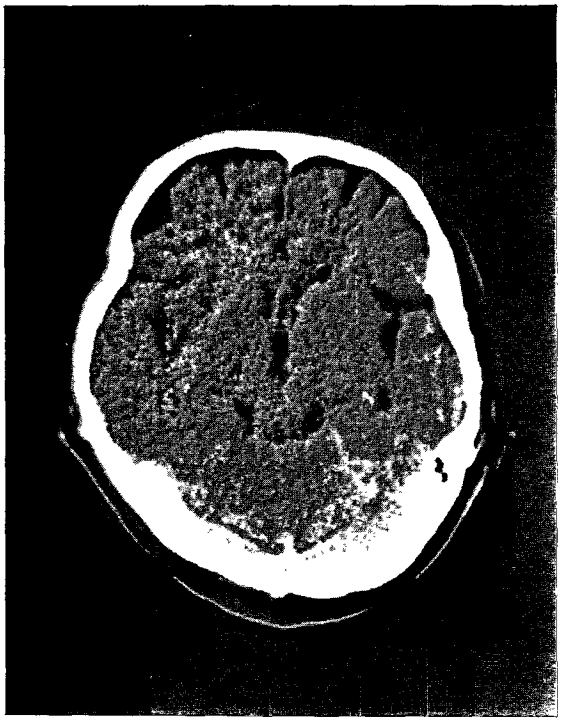


Fig. 4. A case of the large infarct with positive Tc99m-HMPAO SPECT and negative CT : 54 years old man came to ER with rapidly progressive left hemiparesis and memory disturbance on first day after attack. XCT was normal as seen on fig 4(B) and Tc99m-HMPAO SPECT exhibited the large perfusion defect in DPA more than 30% in rt. hemisphere as seen on fig 4(A-1).
Vertex slice shows irregular hypoperfusion in lt. posterior and rt. parietal vertical cortical regions(4A-2)
On secondary XCT demonstrated infarcted hypodensity in corresponding rt. temporal region.

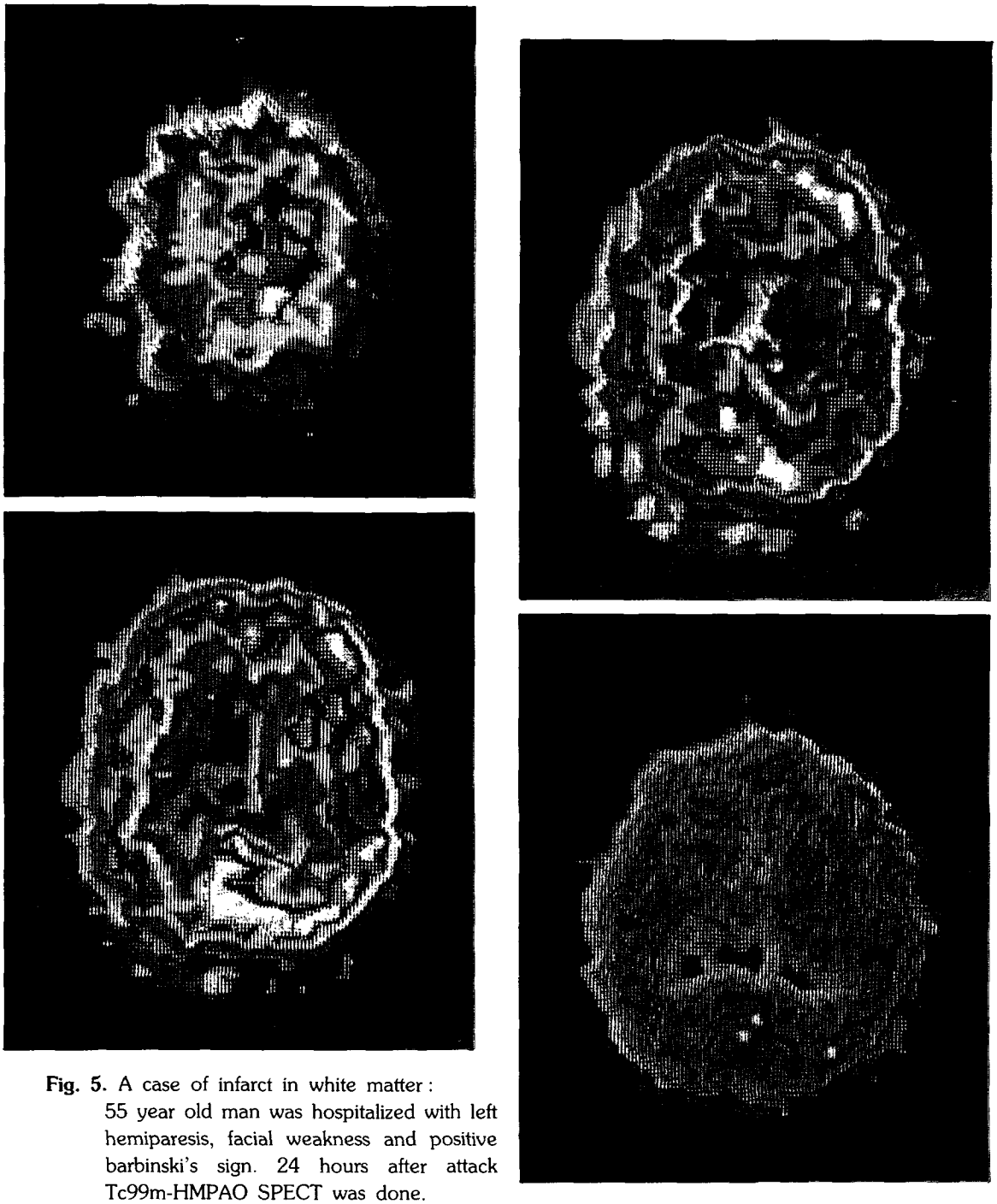
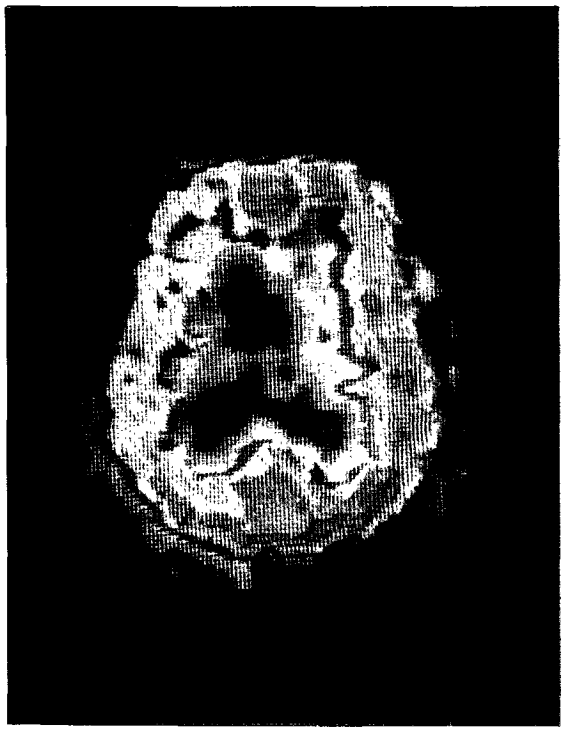
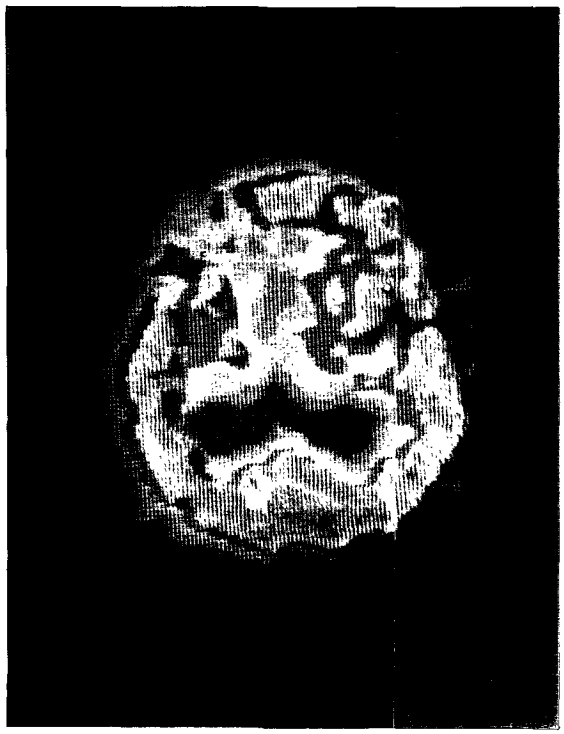


Fig. 5. A case of infarct in white matter :
55 year old man was hospitalized with left hemiparesis, facial weakness and positive barbinski's sign. 24 hours after attack Tc99m-HMPAO SPECT was done.
A) top left : cerebral vertex level of SPECT slice B) top right : mid-parietal level of SPECT slice C) low left : upper temporal level of SPECT slice D) low right : low temporal level of SPECT slice
A large perfusion defect(DPA more than 30%) is seen in right white matter.
Ipsilateral hemispheric cortex shows functional level of hypoperfusion,(DPA less than 10%) that suggests unilateral diaschisis.





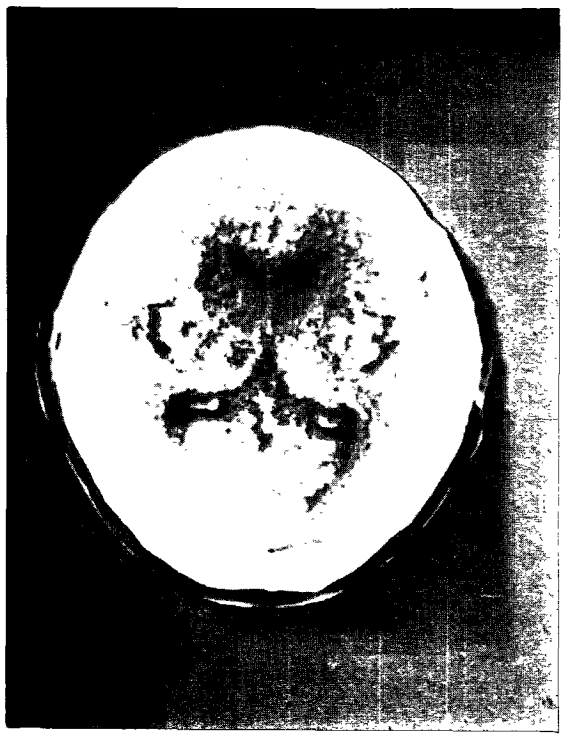


Fig. 6. A case of infarct in the head of caudate nucleus :
67 year old man was hospitalized with neurologic deficits of rt. leg monoparesis and dizziness.
Tc99m-HMPAO SPECT depicts a focal perfusion defect in DPA 15 to 20% level in the head of rt. caudate nucleus in comparison with opposite site.(A : slice level of mid-temporal region B : slice level of low temporal region)
XCT of same slice level (C) reveals better density difference of infarcted at caudate nucleus from adjacent brain and CSF in the anterior horne of lateral ventricles.