Spectrum of neurocognitive dysfunction in Indian population on FDG PET/CT imaging

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ABSTRACT

Background: A variety of neurodegenerative disorders produce significant abnormal brain function which can be detected using fluorodeoxyglucose positron emission tomography (FDG PET) scan even when structural changes are not detected on CT or MRI Scan. A study was undertaken at our institute to evaluate the FDG PET/CT findings in Indian population suffering from mild cognitive impairment (MCI), Alzheimer’s disease (AD), fronto-temporal dementia (FTD), dementia with Lewy body disease (DLBD) and other miscellaneous causes of dementia. Materials and Methods: 117 subjects having neurocognitive deficits and 36 normals were included in our study. All patients underwent a detailed history and clinical examination. This was followed by a mini mental state examination. Subsequently an FDG brain PET scan and an MRI were done. Results: In the patient population included in our study group 36 were normal, 39 had MCI, 40 had AD, 14 had FTD, and 13 had DLBD and 11 dementia due to other miscellaneous causes. MCI patients showed primarily reduced tracer uptake in the mesio-temporal cortex. AD patients showed reduced tracer concentration in temporo-parietal lobes, while patients with advanced diseases showed frontal lobe disease additionally. In subjects of FTD, reduced radiotracer uptake in the fronto-temporal lobes was noted. In addition, FTD patients also showed basal ganglia defects. In contrast the DLBD patients showed globally reduced FDG uptake including severely affecting the occipital cortices. Conclusion: In the current study the F18-FDG PET scans have been shown to be highly useful in the diagnosis of various neurocognitive disorders of the brain. AD was found to be the most common dementia in the Indian population followed by MCI. Diffuse Lewy body disease, FTD and other miscellaneous categories of dementia had a near similar incidence.

Keywords: Dementia, F18-FDG scan, Indian population

INTRODUCTION

One of the most important uses of PET in neurosciences has been in the workup of the patients of various dementing disorders. Criteria for diagnosis of AD were defined by the National institute of neurological and communicative disorders and the Alzheimer’s disease and related disorders. These require evidence of progressive, chronic cognitive deficits in middle aged and elderly patients with no identifiable cause. It is very difficult to differentiate between AD and various other causes of dementia. It has been reported that there is 20-30% decrease in the brain FDG uptake values in patients with various dementias when compared with the normal healthy population. The magnitude and extent of hypometabolism correlates with the severity of dementia symptoms. MCI is used as a diagnostic classification concept for patients with decline in cognitive performance, which is in excess of the expected age related changes but does not completely fit into the diagnosis of dementia. It has been reported that a substantial proportion of MCI patients subsequently may develop dementing disorder of the Alzheimer type (DAT). Hence, it is important to detect patients of MCI as it includes a sizeable number of subjects with pre-dementia of AD. If patients of MCI are diagnosed on the basis of clinical diagnosis only, then there is a very high possibility of including the population suffering from cerebrovascular disease or depression.
FDG PET assessment of cerebral glucose metabolism is a measure of synaptic activity and can identify the presence and localization of a neurodegenerative process in the brain. Different criteria have been laid down for differential diagnosis of dementia.[11,13-18]

AD patients typically show hypometabolism in parieto-temporal cortices and in fronto-limbic in the advanced stage of the disease.[19] While the FTD patients show hypometabolism in the frontal and temporal cortices,[20-22] the DLBD patients show primary hypometabolism in the parieto-occipital cortex.[23,24] The present study was undertaken to evaluate the spectrum of various neurocognitive dysfunction in the Indian population.

MATERIALS AND METHODS

The study comprised of 117 subjects including 39 MCI, 40 AD, 14 FTD, 13 DLBD patients and 11 belonging to miscellaneous group.

The subjects were referred from a tertiary neurological centre after detailed history (corroborated by a close informant), clinical examination and mini mental state examination (MMSE). All patients were subjected to FDG PET scan and MRI of brain. All participants provided written informed consent. Approval of the local ethics committee was taken. None of the patients had any evidence of organic brain pathology or organic illness affecting the brain, significant head injury, systemic illness, psychosis or history of drug or alcohol intake.

Normal population

The control normal population included in our study had no functional impairment based on detailed neurological examination. These subjects had a clinical dementia rating (CDR) = 0 or global deterioration scale (GDS) ≤ 2. They all had a MMSE score of more than 28. The subjects were matched to the cases on the basis of age, sex and educational status.

MCI

The criteria for MCI were based on clinical examination showing impaired cognitive function, ability to perform normal daily activities, no evidence of dementing disease. They had a CDR = 0.5 or GDS = 3 and they all had normal activity of daily living (ADL). The MMSE score of these patients was equal to or more than 24.

Dementia

All subjects fulfilled the diagnostic and statistical manual of mental disorders (DSM-IV),[25] criteria for dementia. They all had significant ADL defects, and had a CDR ≥ 1 or GDS ≥ 4. Standard clinical criteria were used to characterize the type of dementia. Consensus criteria were used for the diagnosis of DLB[26] and FTD.[27]

F18-FDG PET

All patients were fasting for at least 4 hours before the study and advised adequate hydration for rapid tracer excretion. The studies were done in a resting state with eyes closed. Ear plugs were used to prevent any auditory stimulus. The PET/CT study was performed on a Discovery STE 16 (GE) camera. F18-FDG was injected intravenously in a dose of 370 MBq and a brain scan was obtained after an interval of 60 minutes with patient in supine position and head immobilized in a head rest. An initial scan of the head with localizer positioning was followed by a low dose CT acquisition at 110mA and 120 KV for attenuation correction. This was followed by a static 20 minute single bed position 3 dimensional emission scan. Data was reconstructed using 3-dimensional VUE algorithms (GE) and images were viewed for interpretation on a Xeleris workstation using volumetric protocol (GE). Visual image interpretation was independently performed by 3 PET physicians (Dr. MT, Dr. RS and Dr. AJ) for FDG PET brain scans using fused PET/CT images. Any tracer activity which was noted as abnormal by all the 3 physicians was reported as abnormal.

MRI

MRI studies were undertaken for all patients to rule out morphological abnormalities, vascular insults and intracranial space occupying lesions. This was undertaken on a 1.5 T-Magnetom Vision (Seimens) scanner with a standardized protocol consisting of axial T1 weighted images (TR 655ms, TE 24 ms, NEX 2) axial and sagittal, T2 weighted images (TR - 3800 ms, TE 90 ms, NEX 2) and axial and coronal FLAIR images (TR - 9000 ms, TE 110ms, NEX 2).

RESULTS

Details of the subjects included in the study have been depicted in Tables 1 to 5 for the MCI, AD, FTD, DLBD and Miscellaneous Dementia Category respectively. Among MCI patients 17 out of 39 (43.5%) showed cortical hypometabolism indicative of neurodegenerative disease. Mesio-temporal hypometabolism was the most common defect noted in patients of MCI [Figure 1]. The remaining 22 subjects did not show significant cortical hypometabolism. Out of 17 patients found to have abnormal FDG Brain scan 11 were labeled as MCI, 4 AD and 2 had FTD pattern. 2 (18.8%) MCI patients showed bilateral F18-FDG uptake reductions. 6 (54.5%) showed predominantly left and 3 (27.2%) showed a predominant right reductions. The AD group included 17 patients with mild and 23 patients with moderate to severe dementia. Among the AD patients 17/40 (42.5%) showed prominent parieto-temporal hypometabolism [Figure 2]. Symmetric F18-FDG uptake reductions were found in 26/40 (65%) AD, 8/40 (20%) showed severe hypometabolism in left hemisphere and 6/40 (15%) showed more severe hypometabolism in right hemisphere. 23/40 (57.5%) AD patients showed additional frontal cortex hypometabolism. No extension into occipital cortex was noted in AD patients in our series of patients.

The DLBD group included 13 patients, out of which 4 were with mild dementia and 9 patients had moderate to severe dementia. Among the DLBD patients, all patients showed hypometabolism in the occipital cortex of the brain besides affecting temporal,
### Table 1: Clinical and diagnostic characteristics of MCI subjects

| Age | Sex | Clinical diagnosis | MMSE Score | Symptoms | MRI | PET | Final diagnosis |
|-----|-----|-------------------|------------|----------|-----|-----|----------------|
| 55 M | MCI | 26 | Memory loss | Normal | Normal | Normal | MCI |
| 82 M | MCI | 26 | Memory loss | Cerebral atrophy | reduced tracer uptake in left mesial temporal cortex | MCI |
| 66 M | MCI | 28 | Forgetfulness | Cerebral atrophy | Normal | Normal | MCI |
| 81 M | MCI | 28 | Forgetfulness | Normal | Normal | Normal | MCI |
| 50 F | MCI | 24 | Forgetfulness | Gyiotic Scar left Parietal Lobe | Normal | Normal | MCI |
| 50 M | MCI | 28 | Forgetfulness | Normal | Normal | Normal | MCI |
| 80 M | MCI | 27 | Memory loss | Diffuse cerebral atrophy | Normal | Normal | MCI |
| 67 M | MCI | 26 | Forgetfulness | Diffuse cerebral atrophy | Left temporo mesial hypometabolism | MCI |
| 49 M | MCI | 25 | Forgetfulness | Diffuse cerebral atrophy | B/L Fronto-Tempo Hypo Metab | FTD |
| 65 F | MCI / AD | 27 | Memory loss | Diffuse cerebral atrophy | Right Mesial temp Hypo metb | MCI |
| 62 M | MCI | 27 | Forgetfulness | Normal | Normal | Normal | MCI |
| 55 M | MCI | 28 | Memory loss | Normal | Normal | Normal | MCI |
| 72 M | MCI | 28 | Change in Speech | Normal | Normal | Normal | MCI |
| 63 M | MCI | 29 | Forgetfulness | Normal | Normal | Normal | MCI |
| 74 M | AD / MCI | 25 | Forgetfulness | Diffuse cerebral atrophy | B/L temp-ro-parial frontalhypometab | AD |
| 71 M | MCI | 24 | Forgetfulness | Diffuse cerebral atrophy | Reduced Tracer uptake in B/L mesio-temporal lobes | MCI |
| 80 F | MCI | 28 | Forgetfulness | Normal | Normal | Normal | MCI |
| 74 M | MCI | 26 | Memory loss | Normal | Right Mesial temp Hypo metb | MCI |
| 37 M | FTD / MCI | 25 | Forgetfulness | Diffuse cerebral atrophy | B/L frontal hypometab | FTD |
| 58 F | MCI | 24 | Forgetfulness | Diffuse cerebral atrophy left Parito-temporal Hypo metab | AD |
| 61 F | MCI | 28 | Forgetfulness | Normal | Normal | Normal | AD |
| 65 M | MCI | 28 | Forgetfulness | Normal | Normal | Normal | AD |
| 81 M | MCI | 25 | Forgetfulness | Normal | Reduced tracer uptake in mesio-temporal and Left parietal cortex | AD |
| 56 F | MCI | 26 | Memory loss | Normal | Right Mesial temp Hypo metb | MCI |
| 81 M | MCI | 27 | Forgetfulness, attention defect | Diffuse cerebral atrophy | Normal | Normal | AD |
| 50 M | MCI | 28 | Forgetfulness | Normal | Normal | Normal | AD |
| 75 M | MCI | 28 | Forgetfulness, naming difficulty | Diffuse cerebral atrophy reduced tracer uptake in temporal and parietal cortex | AD |
| 65 M | MCI | 28 | Forgetfulness | Normal | Normal | Normal | AD |
| 64 M | MCI | 29 | Forgetfulness | Normal | Normal | Normal | AD |
| 82 M | MCI / AD | 26 | Memory loss | Normal | left mesial temporal hypometabolism | MCI |
| 78 F | MCI | 29 | Memory loss | Normal | Normal | Normal | AD |
| 64 F | FTD / MCI | 27 | Memory loss | Normal | left mesial temporal hypometabolism | MCI |
| 71 M | MCI | 30 | Forgetfulness | Normal | Normal | Normal | AD |
| 79 M | MCI | 29 | Memory loss | Normal | Normal | Normal | AD |
| 55 M | MCI | 29 | Forgetfulness | Normal | Normal | Normal | AD |
| 69 F | MCI | 26 | Forgetfulness | Normal | left mesial temporal hypometabolism | MCI |
| 45 F | MCI | 28 | Forgetfulness | Normal | Normal | Normal | AD |
| 62 M | MCI | 30 | Forgetfulness | Normal | Normal | Normal | AD |
| 72 M | MCI / AD / VCI | 24 | Forgetfulness | Diffuse cerebral atrophy | Reduced Tracer uptake in B/L mesio-temporal lobes | MCI |

Average of MMSE Index 27.02564
SD of MMSE Index 1.693442

MCI- Mild Cognitive Impairment AD- Alzheimer’s Disease
FTD- Fronto-temporal Dementia

### Table 2: Clinical and diagnostic characteristics of AD subjects

| Age | Sex | Provisional Diagnosis | MMSE Score | Symptoms | MRI | PET | Diagnosis |
|-----|-----|-----------------------|------------|----------|-----|-----|-----------|
| 68 M | AD / AADC / MCI | 26 | Forgetfulness, thought disorder | Diffuse cerebral atrophy Right parietal-temporal hypometabolism | AD |
| 78 M | AD | 24 | Forgetfulness, behaviour disturbance | Diffuse cerebral atrophy Reduced tracer uptake in Left Fronto- parieto- temporal uptake | AD |
| 80 M | PSP with dementia | 19 | Thought disorder, disorientation | Diffuse cerebral atrophy B/L Mesiotemporal and parietal hypometabolism | AD |
| 63 F | AD | 24 | Forgetfulness, thought disorder | Diffuse cerebral atrophy B/L Fronto-parietal hypometabolism | AD |

Contd.....
### Table 2: (Contd....)

| Age | Sex | Provisional Diagnosis | MMSE Score | Symptoms | MRI | PET | Diagnosis |
|-----|-----|-----------------------|------------|----------|-----|-----|-----------|
| 70  | M   | AD/VD                 | 22         | Forgetfulness, confusion | B/L Frontal and parietal hypometabolism | Left fronto-parieto-temporal lobe | AD |
| 58  | M   | AD                     | 22         | Forgetfulness, thought disorder, uncooperative | Diffuse cerebral atrophy | B/L Mesio-frontal and parietal hypometabolism | AD |
| 68  | F   | AD/FTD                | 21         | Memory loss, disorientation | Diffuse cerebral atrophy | Left fronto-parieto-temporal hypometabolism | AD |
| 74  | M   | AD/MCI                | 20         | Forgetfulness, confusion thought disorder | Diffuse cerebral atrophy | B/L tempo-parietal frontal hypometabolism | AD |
| 45  | F   | AD                     | 26         | Forgetfulness | Diffuse cerebral atrophy | Reduced tracer uptake in Left temporo-parietal lobe | AD |
| 65  | F   | AD                     | 25         | memory loss, thought disorder | Diffuse cerebral atrophy | Reduced tracer uptake in Right temporo-parietal lobe | AD |
| 48  | M   | AD                     | 26         | Memory loss, social withdrawal | Diffuse cerebral atrophy | Reduced tracer uptake in Right temporo-parietal cortex | AD |
| 62  | M   | AD                     | 25         | Forgetfulness, thought disorder | Diffuse cerebral atrophy | Reduced tracer uptake in Left parieto-temporal cortex | AD |
| 26  | M   | FTD                   | 23         | Cognitive decline, confusion, disorientation, memory loss | Hippocampus normal, S/o neuro degenerative disorder | B/L Fronto-parieto-temporal lobes | AD |
| 67  | F   | AD                     | 22         | Forgetfulness | Diffuse cerebral atrophy | B/L Parieto-temporal hypometabolism | AD |
| 60  | M   | AD                     | 26         | Forgetfulness | Diffuse cerebral atrophy | Reduced tracer uptake in Right parieto-temporal cortex | AD |
| 50  | F   | AD                     | 25         | Memory loss, thought disorder | Cerebellar atrophy | Reduced tracer uptake in Right parieto-temporal cortex | AD |
| 58  | F   | MCI                   | 26         | Forgetfulness | Diffuse cerebral atrophy | B/L Fronto-parieto-temporal hypometabolism | AD |
| 65  | F   | AD                     | 20         | Forgetfulness, disorientation | Diffuse cerebral atrophy | Reduced tracer uptake in B/L temporo-parietal and frontal cortex | AD |
| 73  | M   | AD                     | 20         | Memory loss, confusion | Diffuse cerebral atrophy | Reduced tracer uptake in B/L parietal and temporal lobes | AD |
| 80  | M   | AD                     | 26         | Memory loss | Diffuse cerebral atrophy | Reduced tracer uptake in B/L parietal and temporal lobes | AD |
| 55  | M   | AD                     | 26         | Forgetfulness | Diffuse cerebral atrophy | Reduced tracer uptake in B/L parietal and temporal lobes | AD |
| 78  | F   | AD                     | 19         | Memory loss, confusion, uncooperative | Diffuse cerebral atrophy | B/L frontal-parietal and temporal hypometabolism | AD |
| 78  | M   | AD                     | 20         | Memory loss, confusion | Diffuse cerebral atrophy | B/L frontal-parietal and temporal hypometabolism | AD |
| 81  | M   | MCI                   | 20         | Memory loss, confusion disorientation | Diffuse cerebral atrophy | B/L frontal-parietal and temporal hypometabolism | AD |
| 55  | M   | FTD                   | 26         | Forgetfulness | Diffuse cerebral atrophy | Reduced tracer uptake in right parieto-temporal cortex | AD |
| 45  | M   | AD                     | 22         | Forgetfulness, disorientation | Diffuse cerebral atrophy | Reduced tracer uptake in B/L frontal-parietal and temporal cortex | AD |
| 61  | F   | AD                     | 26         | Forgetfulness | Non specific ischaemia | Reduced tracer uptake in Left parietal lobes | AD |
| 30  | F   | AD                     | 20         | Forgetfulness, confusion | Diffuse cerebral atrophy | B/L reduced perfusion in frontal parietal and temporal and basal ganglia | AD |
| 60  | M   | AD                     | 19         | Forgetfulness, confusion, uncooperative | Diffuse cerebral atrophy | B/L reduced perfusion in frontal parietal and temporal and thalamus | AD |
| 50  | F   | AD                     | 22         | Forgetfulness, confusion | Diffuse cerebral atrophy | Reduced tracer uptake in B/L parietal and temporal lobes | AD |
| 75  | M   | AD                     | 26         | slurring of speech | Diffuse cerebral atrophy | Reduced tracer uptake in Right parieto-temporal lobes | AD |
| 45  | M   | AD                     | 20         | Forgetfulness, behaviour change, irritability | Diffuse cerebral atrophy | B/L Parieto-temporal-frontal hypometabolism | AD |
| 71  | F   | AD                     | 20         | Forgetfulness, difficulty in speech, naming difficulty | Diffuse cerebral atrophy | Parietal-temporal- frontal cortex | AD |
| 75  | M   | MCI                   | 28         | Forgetfulness | Diffuse cerebral atrophy | Reduced tracer uptake in B/L frontal-temporal cortex | AD |
| 65  | M   | FTD                   | 22         | Abnormal Behaviour, walking difficulty loss of memory, behaviour change | Diffuse cerebral atrophy | Reduced tracer uptake in B/L frontal-parietal - temporal lobes | AD |
| 71  | M   | AD                     | 25         | Abnormal Behaviour, walking difficulty | Diffuse cerebral atrophy | Reduced tracer uptake in B/L frontal-parietal - temporal lobes | AD |
| 56  | F   | AD/FTD                | 23         | Forgetfulness, does not recognize relatives | Diffuse cerebral atrophy | Reduced tracer uptake in B/L frontal-parietal-temporal hypometabolism | AD |
| 55  | F   | FTD/AD                | 19         | Abnormal Behaviour, forgetfulness | Diffuse cerebral atrophy | Reduced tracer uptake in B/L Parietal-temporal frontal cortex | AD |
| 60  | F   | FTD                   | 22         | Memory loss, confusion | Diffuse cerebral atrophy | Markedly tracer uptake in B/L frontal-parietal temporal cortices | AD |

Average of MMSE Index: 22.84615
SD of MMSE Index: 2.716403
Table 3: Clinical and diagnostic characteristics of FTD subjects

| Age | Sex | Provisional Diagnosis | MMSE Score | Symptoms | MRI | PET | Diagnosis |
|-----|-----|-----------------------|------------|----------|-----|-----|-----------|
| 69  | F   | FTD                   | 25         | Abnormal behaviour, speech disorder | Lacunar Infarct | Mild Right fronto-temporal hypometabolism | FTD |
| 55  | F   | DLBD                  | 26         | Abnormal behaviour, forgetfulness | Normal | Left Fronto-temporal Hypometabolism | FTD |
| 49  | M   | MCI                   | 24         | Forgetfulness, abnormal behaviour | Diffuse cerebral atrophy | Left Fronto-Tempo Hypo Metab | FTD |
| 77  | M   | AD                    | 28         | Forgetfulness | Normal | reduced right frontal tracer uptake | FTD |
| 63  | F   | FTD                   | 27         | Mild social withdrawal | Diffuse cerebral atrophy | Reduced tracer uptake in left frontal lobe | FTD |
| 37  | M   | FTD/MCI               | 24         | Abnormal behaviour, speech disorder | Diffuse cerebral atrophy | Right frontal hypometab | FTD |
| 63  | M   | FTD                   | 14         | Hallucination and Delusion | Frontal temporal atrophy | B/L Reduced tracer uptake in fronto-parieto-temporal lobes | FTD |
| 65  | F   | FTD                   | 27         | Forgetfulness | Diffuse cerebral atrophy | Left frontal and temporal hypometab | FTD |
| 60  | M   | AD                    | 25         | Memory loss, anormal behaviour | Generalised cerebral atrophy | Right frontal and temporal hypometab | FTD |
| 68  | F   | AD                    | 26         | Forgetfulness, social withdrawal | Diffuse cerebral atrophy | Reduced tracer uptake in right frontal and temporal lobes | FTD |
| 60  | F   | FTD                   | 16         | Forgetfulness, Abnormal Behaviour, delusions | Diffuse cerebral atrophy | Reduced tracer uptake in Left fronto-parietal and temporal lobes, both basal ganglia | FTD |
| 26  | M   | FTD                   | 16         | Abnormal behaviour, forgetfulness | Normal | reduced tracer uptake in Right fronto-parietal temporal and both caudate nuclei | FTD |
| 61  | M   | FTD                   | 24         | Forgetfulness, disoriented | Diffuse cerebral atrophy | Reduced tracer uptake in left frontal temporal lobes | FTD |
| 60  | F   | FTD                   | 14         | Abnormal behaviour, irrelevant talking, forgetfulness | Diffuse cerebral atrophy | Right Fronto patieto temporal hypometabolism | FTD |

Average of MMSE Index 22.571
SD of MMSE Index 5.1398

Table 4: Clinical and diagnostic characteristics of DLBD subjects

| Age | Sex | Provisional diagnosis | MMSE Score | Symptoms | MRI | PET | Final diagnosis |
|-----|-----|-----------------------|------------|----------|-----|-----|----------------|
| 75  | M   | MSA                   | 19         | Cognitive decline, hallucinations | Diffuse cerebral atrophy | Reduced B/L Frontal Parietal and Occipital Cortex Hypometabolism | DLBD |
| 73  | M   | PD with dementia      | 21         | Cognitive decline, delusions | Diffuse cerebral atrophy | reduced tracer uptake in B/L fronto-parieto-temporo-occipital lobes | DLBD |
| 23  | M   | EPS with frontal lobe symptoms Deg Dementia | 18 | hallucinations, syncope | Frontal, caudate and cerebella atrophy | B/L Fronto-parieto-temporal occipital and both caudate hypometabolism | DLBD |
| 59  | F   | FTD                   | 23         | Forgetfulness, Cognitive decline | Diffuse cerebral atrophy | Reduced tracer uptake in Left parieto-temporal and occipital cortex | DLBD |
| 75  | F   | FTD                   | 18         | Forgetfulness, hallucinations, falls | Diffuse cerebral atrophy | B/L fronto-parieto-temporo occipital hypometabol | DLBD |
| 70  | F   | DLBD                  | 28         | Memory loss, cognitive decline, delusions | Diffuse cerebral atrophy | Reduced tracer uptake in Right parieto-temporal and occipital cortex | DLBD |
| 75  | M   | DLBD                  | 20         | Memory loss, cognitive decline, delusions | Diffuse cerebral atrophy | Globally reduced tracer uptake in B/L cortex and basal ganglia, thalamus, cerebellum | DLBD |
| 60  | F   | DLBD                  | 26         | Abnormal Behaviour, walking difficulty, tremors, visual hallucination | Diffuse cerebral atrophy | reduced tracer uptake in Right fronto-parietal-temporal and occipital | DLBD |
| 60  | F   | DLBD                  | 19         | Abnormal behaviour, forgetfulness, syncope | Diffuse cerebral atrophy | reduced tracer uptake in B/L fronto parietal temporal and occipital cortices | DLBD |
| 84  | M   | AD                    | 24         | Forgetfulness, cognitive decline | Diffuse cerebral atrophy | reduced tracer uptake in Right fronto parietal temporal and occipital cortices | DLBD |
| 76  | M   | DLBD                  | 21         | Abnormal behaviour, forgetfulness, loss of consciousness | Diffuse cerebral atrophy | B/L Fronto patieto temporal occipital and basal ganglia hypometabolism | DLBD |
| 75  | M   | DLBD                  | 18         | Abnormal behaviour, forgetfulness, hallucination, delusions | Diffuse cerebral atrophy | reduced tracer uptake in B/L fronto parietal temporal and occipital cortices | DLBD |
| 65  | F   | DLBD                  | 19         | Abnormal Behaviour, forgetfulness, hallucination | Diffuse cerebral atrophy | B/L reduced tracer uptake in fronto parietal temporal and occipital cortices | DLBD |

Average of MMSE Index 21.07692
SD of MMSE Index 3.252218
Table 5: Clinical and diagnostic characteristics of miscellaneous subjects

| Reg. No. | Age | Sex | Provisional diagnosis | MMSE Score | Symptoms | MRI | PET | Diagnosis |
|----------|-----|-----|-----------------------|------------|---------|-----|-----|-----------|
| 193/10  | 60  | F   | FTD/VD                | 21         | Cognitive dysfunction, hemiparesis, bradykinesia, ataxia Forgetfulness, inability to walk, syncope | Gliosis and encephalomalacia | Right Fronto-temp and Rt. Thalamus Hypo metb | VD |
| 298/10  | 50  | M   | PSP/MCI               | 23         | Forgetfulness, frequent falls and walk slowly, tremors | Mild diffuse cerebral atrophy | FDG- reduced tracer uptake in B/L medial-frontal cortex and parietal cortex FDOPA- reduced tracer uptake in B/L globus pallidus and putamen | PD |
| 695/10  | 76  | M   | MCI/AD/VCI            | 22         | Forgetfulness, frequent falls and walk slowly, tremors | Chronic ischemic changes with cerebral atrophy | FDG- reduced tracer uptake in B/L frontal-parietal - occipital and caudate FDOPA- reduced tracer uptake in B/L basal ganglia | PD |
| 562/10  | 56  | F   | CJD                   | 24         | Memory loss, Personality Changes, Hallucinations | Increased signal in the parietal and occipital lobes. | Reduced tracer uptake in fronto-parietal - temporal lobes and B/L basal ganglia and thalamus | CJD |
| 231/10  | 60  | F   | PSP/PD                | 22         | Forgetfulness, Gait difficulty, tremors | Non specific Ischemic foci in B/L cerebral hemispheres Ischemic demyelination of brain | FDG- reduced tracer uptake in mesio-frontal cortex and Ant. Cerebral cortex FDOPA- reduced tracer uptake in B/L Putamens and globus pallidus | PD |
| 46/10   | 75  | F   | PSP                   | 23         | Abnormal behaviour, tremors, falls, slow gait | Diffuse Cerebral and cerebellar Atrophy | FDG- reduced tracer uptake in the frontal cortex, midbrain and pons FDOPA- reduced tracer uptake in B/L basal ganglia | PD |
| 295/10  | 76  | M   | PSP                   | 23         | Forgetfulness, abnormal behaviour, tremors, rigidity | Diffuse cerebral atrophy | FDG- reduced tracer uptake in B/L frontal and parietal cortex FDOPA- reduced tracer uptake in B/L basal ganglia | PD |
| 221/11  | 77  | M   | PD                    | 22         | Forgetfulness, tremors, ataxia, rigidity | Diffuse cerebral atrophy | FDG- reduced tracer uptake in B/L temporal cortex FDOPA- reduced tracer uptake in B/L basal ganglia | PD |
| 336/11  | 81  | M   | AD                    | 22         | Abnormal behaviour, Ataxia, slow gait | Diffuse cerebral atrophy | Reduced tracer uptake in temporal and occipital lobe markedly tracer uptake in B/L frontal-parietal basal ganglia and thalamus | VD |
| 378/10  | 61  | F   | CJD                   | 24         | Behavioral changes, involuntary body movements, inability to recognize people | Normal | Diffuse Cerebral Atrophy | FDG- reduced tracer uptake in the mesio-temporal cortex FDOPA- reduced tracer uptake in B/L Caudate nucleus | CJD: Creutzfeldt-Jakob disease |
| 932/09  | 62  | M   | Parkinsons Dementia   | 22         | Memory Loss, Bradykinesia, tremors | Diffuse Cerebral Atrophy | PD: Parkinson’s Dementia |

Average of MMSE Index 22.55
SD of MMSE Index 0.934

Figure 1: 71-year-old male presented with the complaints of forgetfulness. His MMSE score was 24. Arrows in F18-FDG PET images show bilateral Mesio-temporal hypometabolism diagnostic of MCI, in this patient
The miscellaneous group comprised of 7 patients of Parkinson’s dementia (PD) [Figure 5], 2 of vascular dementia [Figure 6] and 2 of Creutzfeldt-Jakob disease [Figure 7].

DISCUSSION

The importance of PET imaging in the management of the dementia patient is to help in the early diagnosis of the dementia disease process. Early detection of patients of MCI is essential as one third of MCI patients proceed to manifest DAT while more than half do not show progression to dementia. These results confirm that MCI patients even when selected carefully...
after clear cut inclusion criteria, represent a very heterogeneous patient population with regard to prognosis. Our study group comprised of 39 patients referred to our institute after being diagnosed to have MCI on clinical and MMSE test examination. Out of these MCI patients 13 were found to be normal. This itself highlights the importance of PET examination in patients having a neurocognitive disorder. Mosconi et al., reported that out of 37 patients screened for MCI, 12 were found to be normal on PET study.[28] Our study findings also show that F18-FDG PET can differentiate MCI from normal patients quite efficiently making it an effective tool to distinguish between these two groups which cause a lot of diagnostic issues as clinically it is quite difficult to distinguish age related cognitive deterioration from MCI which is a diagnostic dilemma for the clinician. Various F18-FDG PET profiles have been reported in the MCI group of patients which is due to the spectrum of cognitive deficits reported in this patient group.[29] In the present PET study AD PET pattern was found in 4 subjects and FTD pattern was noted in 2 patients. In previously reported PET studies 22-41% of the MCI patients with an AD PET pattern eventually converted to AD within a time period of 1-3 years.[29-34]

F18-FDG PET scan has played an important role in the diagnosis of AD. Patients of AD present with parieto-temporal defects in the cortices when compared to their age equivalent healthy subjects.[35] It has already been highlighted that patients of MCI are at high risk of developing AD in future.[36] Diagnosis can be made in patients of AD when clinical symptoms are not being fully expressed by the patient, in very early stage of AD. There is definitive evidence to show that generalized atrophy of brain is present in elderly patients, years before they actually develop AD.[37-41]

In our study, 42.5% of the patients had a mild AD while the rest had a severe form of the disease. The patient with the milder form of the disease had temporo-parietal defects while those with severe form the disease also had defects in the frontal region. In contrast, the western literature has reported that 99% of the patients had mild form of disease while 1 had severe AD pattern.[42] Thus the Indian population in contrast appears to be affected from a more severe form of the disease. On further analysis it was found that 65% population had bilaterally symmetrical defects, while 15% had right dominant pattern and 20% had a left dominant hypometabolism. None of the AD patients were found to have occipital lesions on FDG PET scans.

FTD is one of the most common forms of cortical dementia, accounting for about 20% of presenile dementia. Diagnosis of FTD is difficult as these groups of patients have a variegated clinical and pathological picture.[43,44] Patients suffering from this form of dementia have been reported to suffer from forgetfulness and a variety of behavioral disorders and hence are difficult to separate out from patients of AD, vascular dementia and psychiatric illnesses. It has been reported earlier that FTD results in finding of cerebral atrophy on CT and MR studies and hypo-perfusion in PET studies in frontal and temporal regions of the brain.[45,46] We found additional hypometabolism in the basal ganglia region which are known to be involved...
Thus in the majority of AD patients temporo-parietal defects were noted, in FTD patients more prominent hypometabolism in frontal and temporal cortex was noted and in DLBD patients though a global hypo-perfusion in the cortex was noted, the hypometabolism was most profound in the occipital cortex, which corresponds to the results of earlier workers. This characteristic pattern of cortical hypometabolism including the occipital areas could be a result of diaschisis due to disruption of intracortical connections. Diaschisis is defined as depression of regional neuronal metabolism and cerebral blood flow caused by dysfunction in anatomically separate but functionally related neuronal regions. Typically, sparing of primary sensorimotor cortex was noted. All the patients from this group showed prominent hypometabolism in the occipital region as reported in previous reports. Thus, it is concluded that the most common cause of dementia in Indian population is AD followed by MCI. FTD and DLBD had almost same incidence patterns.

CONCLUSION

The present study illustrates the utility of F18-FDG PET in the diagnosis and characterization of neurocognitive dysfunction. AD has been found to be the most prevalent form of dementia in the Indian subcontinent, which is in conformity with the global trend. A significantly higher proportion of frontal lobe involvement was noted in the Indian population, as compared to that documented in the world-wide literature. These new findings, and epidemiological and genetic studies, are in conformity with the previous reports. F18 FDG PET scans provide an objective and sensitive support to the diagnosis of early dementia.

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