

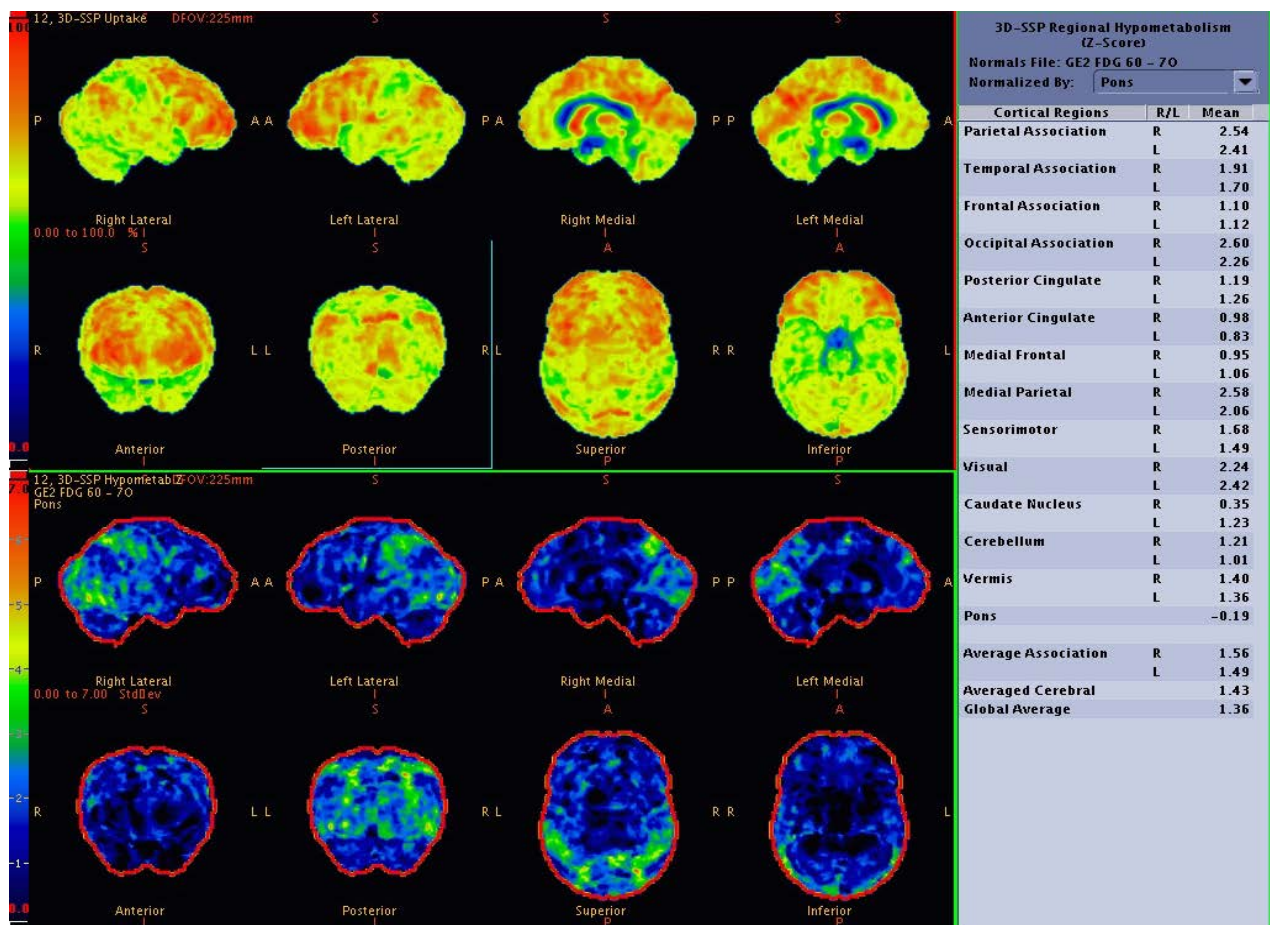
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F-18 FDG PET/CT in Lewy Body Dementia

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CLINICAL DETAILS

A 66y male patient presented with a history of repeated psychiatric admissions and episodes of memory loss. He was referred for an 18F FDG PET-CT Brain to establish underlying dementia.



Above: 18F FDG PET CT Brain. Top image series demonstrates regional hypometabolism (green) at the parietal, temporal and occipital lobes. The bottom image series is a parametric map that demonstrates concordant regions of significant hypometabolism compared with age matched healthy controls in the general population. Quantification data on the right confirms the degree of hypometabolism at the medial occipital lobes (2.6 standard deviations (SD) below the mean) is greater than the temporal lobes (1.9 SD below the mean). This is a recognized imaging feature for diagnosing Lewy Body Dementia.

SCAN FINDINGS

Reduced metabolism in the parietal lobes.
Reduced metabolism in the medial occipital lobes.
Reduced metabolism in the precuneus (R>L).
Mild reduction of metabolism in the lateral temporal lobes
Normal metabolism in the frontal lobes.
Normal metabolism in the posterior cingulate gyrus.

No significant structural abnormality on the non contrast CT brain. No significant structural hippocampal atrophy.

CONCLUSION: Evidence of an intrinsic neuro-degenerative disorder of the Lewy body type.

CONCLUSIONS

18F FDG PET is increasing in benefit and use in ascertaining the cause of dementia when there is clinical doubt.

Clinical, pathological and imaging overlap between Alzheimer's and Lewy Body Dementia can make diagnosis difficult.

Greater hypo metabolism at the medial occipital lobes relative to the hippocampi on 18F FDG PET-CT can discriminate DLB from AD

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DISCUSSION

Dementia with Lewy Bodies (DLB) is now the 2nd most frequent cause of age related neurodegenerative disease affecting over 100000 people in the UK. It belongs to a spectrum of Lewy Body diseases that include Parkinson's disease.¹

Patients typically present with symptoms of cognitive decline, and motor symptoms including slow movement and rigidity. Lewy Body Dementia is characterised by the accumulation of abnormal alpha synnuclein proteins in neurons throughout the brain that can only be confirmed on histology following post mortem.¹ These deposits can cause a loss of cholinergic acetyl production neurons and dopaminergic neurons that characterize Alzheimer's and Parkinson's respectively. This is thought to explain the overlap of presenting cognitive and movement symptoms that can potentially lead to clinical misdiagnosis.

Correct diagnosis is essential. While there is currently no definitive cure, the use of particular antipsychotic neuroleptic medications can cause a deterioration of symptoms that are potentially fatal.

Low dopamine transporter uptake in the basal ganglia on SPECT imaging (e.g. Ioflupane I-123 DaTSCAN) can be useful to establish the diagnosis in those with suspected dementia with DLB if the diagnosis is in doubt and is recommended by the NICE guidelines. In addition, the preservation of the medial temporal lobe structures on CT/MRI is supportive of DLB, however there can be overlap of areas of hippocampal atrophy between patient with Alzheimer's and DLB^{2,3}.

With 18F-FDG PET-CT, DLB is best characterized by hypo metabolism at the posterior cingulate gyrus, parietal association cortex, lateral temporal lobes and occipital lobes². These regions of reduced cortical metabolism are similar in Alzheimer's, however the relative hypo metabolism in DLB is most severe at the medial occipital lobes than the hippocampi with preservation of activity in the posterior cingulate gyrus, which helps to discriminate the diagnosis.⁴