PET and CSF amyloid in the diagnosis of Alzheimer's disease: a study from Gargano

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BACKGROUND

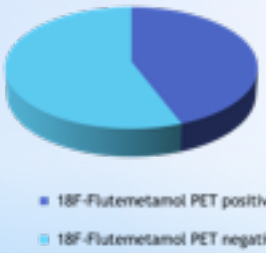
Alzheimer's disease (AD) is the most common cause of dementia. AD is thought to begin 20 years or more before symptoms arise, with small changes in the brain of affected patients. There currently is no effective treatment that delays the onset or slows the progression of AD.

This study is aimed at testing the already described [1] usefulness of liquor Aβ dosage, paired with amyloid imaging assessed by means of positron emission tomography (PET) and with genetics in the diagnosis of Alzheimer patients from Gargano (Apulia).

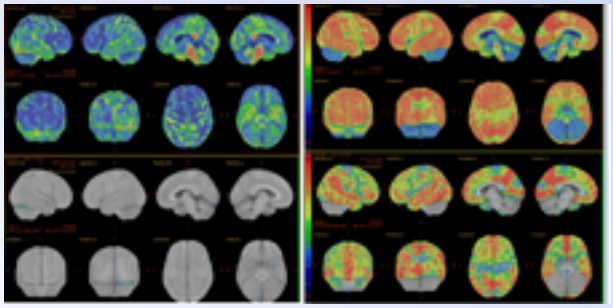
METHOD

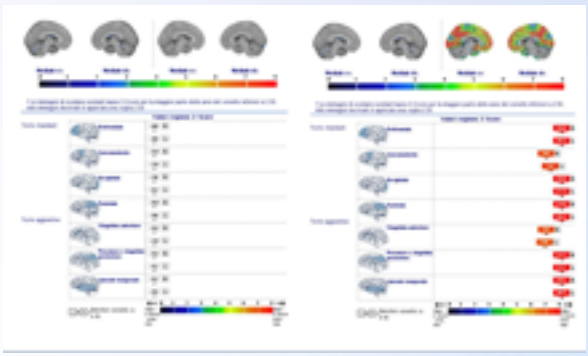
All the 68 patients admitted to Neurology of the IRCCS "Casa Sollievo della Sofferenza"(38 males, 30 females, mean age 63 +/- 5.1 SD) underwent both 18F-Flutemetamol PET and lumbar puncture for CSF. CSF samples were examined for the traditional CSF biomarkers $A\beta_{42}$ and quantified with commercially available ELISAs (EUROIMMUNE) according to the manufacturer's instructions. The recommended cut-off value for a normal test was $A\beta_{42}$ >650 pg/mL and T-tau <350 pg/mL [2].

RESULTS



18F-Flutemetamol PET positive	
n= 20	n= 10
Aß42 < 650 pg/mL	T-tau >350 pg/mL





Patient with 18F-Flutemetamol PET NEGATIVE (right) and with 18F-Flutemetamol PET POSITIVE (left)

DISCUSSION

The clinical diagnosis of disease can lead to a "probable" diagnosis and can only be definitively ascertained post-mortem by detective senile plaques in human brain.

The instrumental exams currently available are very useful, but not with absolute specificity. Diagnostic errors are not infrequent, especially in the early stages of the disease [3].

CONCLUSIONS

In this experience the paired CSF $A\beta_{42}$ dosage and PET assessment can strongly improve the differential diagnosis as accurate and early as possible between AD and other dementias. This represents, moreover, a crucial aspect, to set up a correct therapy for affected patients from AD, especially in relation to the possible introduction of etiological therapies specific to the AD.

REFERENCES

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