for Depression were found in all of 51 investigated patients (100%), but with inverse correlation. As less was cognitive impairment and memory deficit as higher were scores in depression Scales, and as more profound was dementia, patients were less depressed. 33 patients were "naive", 18 patients had previous uneffective treatment with so-called neuroprotectors and vitamins. 6 patients were treated with MAO inhibitor (amitriptyline) but discontinue their treatment because of memory detoriation and worsening of the activities of daily living. 16 patients with MCI successfully overcome their depressive episode when started effective treatment for their memory impairment. **Conclusions:** Both depression and dementia are existing co-morbidities especially in elderly, and need in-time and precise evaluation. Early and correct diagnosis of both conditions must serve as basis for further effective management. In MCI effective treatment of memory impairment could be paramount for general improvement of the mood and reducing of the stress, treatment of patients with Dementia with MAO is usually complicated because of drowsiness and additional reduction of daily activities. Every patients with dementia must be evaluated for depression and treat accordingly the neurological and cognitive deficit.

P1-160 CLINICAL PHENOTYPIC VARIABILITY IN AN ITALIAN FAMILY BEARING THE IVS6+5_8DELGTGA MUTATION IN PGRN GENE

Gabriella Marcon[1], Fabrizio Tagliavini[2], Giacomina Rossi[3], Giorgio Giaccone[3], Marina Grisoli[4], Elena Piccoli[5], Onelio Geatti[6], Sergio Zanini[7], *[1]University of Udine; Fondazione IRCCS Istituto Neurologico Besta (Milan); [2]Fondazione IRCCS Istituto Neurologico Carlo Besta, Milano; [3]Fondazione IRCCS Istituto Neurologico Carlo Besta, Milano; [4]Fondazione IRCCS Istituto Neurologico Carlo Besta, Milano; [5]Fondazione IRCCS Istituto Neurologico Carlo Besta, Milano; [6]Medicina Nucleare, Udine; [7]Istituto E. Medea, Pasian di Prato (UD).*

Background: Frontotemporal dementia (FTD) is a complex presenile disorder characterized by behavioural changes and executive functions, expression of fronto-temporal degeneration. Hereditary FTD accounts for 20-30% of cases and, in the past decade, mutations in the microtubule associated protein tau (*MAPT*)gene were identified as a main genetic causes of familial FTD. In 2006, mutations in the gene encoding progranulin (*PGRN*) were reported, to account for a wide part of the familial FTD cases. Clinically, an high phenotypic variability within and among the kindreds is reported in the familial FTD associated with *PGRN* mutations and occasionally the memory deficits are the first symptoms, resembling Alzheimer's disease (AD). We report an Italian family with dementia associated with a *PGRN* mutation characterized by a deletion of 4 base pairs inside the intron 6 of the gene, leading to haploin sufficiency In our kindred, all three affected patients carried the mutation, but presented very different clinical phenotypes, evoking FTD, AD and rapidly-progressive dementia mimicking prion disease. **Methods:** Informations on the members of the first, second and third generations were obtained conducting interviews with relatives, while for the three patients studied, the clinical evidence of dementia symptoms and their characterization was documented directly with sequential neurological examinations, cognitive assessments and neuroimaging. Blood sample collection and DNA extraction from peripheral blood lymphocytes for genetic analysis were performed after written informed consent of the patients. **Results:** In our pedigree, the *PGRN* mutated patients are affected by dementia with three different clinical pictures: FTD, AD and rapidly progressive dementia mimicking prion disease. Neuropsychological examinations supported these diagnoses, documenting generalized deficits of cortical functions in AD patient and deficits in executive functions and in language in FTD patient. Regarding neuroimaging, in the same two cases MRI results do not correspond to the clinical diagnosis. **Conclusions:** These findings confirms the marked heterogeneity of the clinico-radiological features in patients with *PGNR* mutations and underline the need of considering mutations of this gene as causes of familial dementing diseases with atypical or uncommon features or discrepancies between the clinical and the neuroimaging findings.

P1-161 A COMPARISON OF THE DIAGNOSTIC SENSITIVITY FOR ALZHEIMER'S DISEASE AMONG MRI, ECD-SPECT, FDG-PET, AND CEREBROSPINAL FLUID BIOMARKERS IN A MEMORY CLINIC

Akiyoshi Morinaga[1], Kenjiro Ono[1], Masahito Yamada[1], Tokuhei Ikeda[1], Moeko Shinohara[1], Mitsuhiro Yoshita[1], Yoshihisa Ikeda[1], Keisuke Shima[1], Miharu Samuraki[1], Daisuke Yanase[1], Kazuo Iwasa[2], Ichiro Matsunari[1], *[1]Kanazawa University Graduate School of Medical Science, Kanazawa; [2]The Medical and Pharmacological Research Center Foundation, Hakui.*

Background: Magnetic resonance imaging (MRI), cerebral blood flow single photon emission computed tomography (CBF-SPECT), fluorodeoxyglucose-positron emission tomography (FDG-PET), and cerebrospinal fluid (CSF) biomarkers are used for the diagnosis of Alzheimer's disease (AD). The purpose of our study was to reveal the relative usefulness of these diagnostic tools in clinical practice in a memory clinic. **Methods:** In 207 patients with probable AD in our memory clinic, medial temporal lobe atrophy on MRI, hypoperfusion/hypometabolism of the parieto-temporal lobe and posterior cingulate gyrus in ethylcysteinate dimmer-CBF-SPECT/FDG-PET, and abnormalities of CSF amyloid beta-protein 1-42, total tau, and phosphorylated tau were evaluated as findings characteristic of AD. **Results:** The findings characteristic of AD were found in 77.4% of all AD patients with MRI, 81.6% with CBF-SPECT, 93.1% with FDG-PET, and 90.0% with CSF biomarkers. At the stage of Clinical Dementia Rating (CDR) 0.5, CSF biomarkers were the most sensitive (90.0%); at the stage of CDR 1, FDG-PET (96.7%) and CSF biomarkers (95.5%) were highly sensitive. At the stage of CDR 2, all tools showed high positive percentages. **Conclusions:** The diagnosis of AD was most often supported by CSF biomarkers and FDG-PET at the early stage of dementia (CDR 1) and by CSF biomarkers at the earlier stage (CDR 0.5).

P1-163 COMPARING FACIAL EMOTION PROCESSING IN PROGRESSIVE NONFLUENT APHASIA, LOGOPENIC PROGRESSIVE APHASIA AND ALZHEIMER'S DISEASE

Olivier Piguet[1], Liam Gleeson[2], Suncica Lah[3], Laurie Miller[4], John Hodges[5], *[1]NeuRA, Randwick; [2]University of New South Wales, Sydney; [3]University of Sydney, Sydney; [4]Prince of Wales Hospital, Sydney; [5]Neuroscience Research Australia, Sydney.*

Background: Nonfluent primary progressive aphasias are generally divided into primary nonfluent aphasia (PNFA) and logopenic progressive aphasia (LPA), with distinct underlying pathology: PNFA belongs to the frontotemporal dementia spectrum whereas LPA is predominantly associated with Alzheimer pathology. Clinically, PNFA and LPA present with similar expressive language deficits and are difficult to differentiate in life. Facial emotion recognition is typically impaired in patients with Alzheimer's disease (AD). Whether performance on facial emotion recognition tasks differs in PNFA and LPA patients and whether LPA patients experience deficits that are similar to those observed in AD is unknown. **Methods:** Fourteen PNFA, 12 LPA and 18 AD patients were recruited along with 38 age- and education-matched healthy controls. Participants were administered four tasks measuring different aspects of emotion recognition: face perception, identity matching, emotion matching and emotion selection, as well as the Ekman 60 face recognition task. **Results:** Compared to the healthy controls, all three patient groups were significantly impaired on the Ekman 60 face recognition task. In contrast, patient groups exhibited