

## **APomorphine infusion and aMYLoid deposition in Parkinson's disease (APOMYL): preliminary clinical and amyloid imaging data**

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**Objective:** to explore the potential effects of chronic Apomorphine infusion treatment on reducing brain Amyloid- $\beta$  (A $\beta$ ) deposition in Parkinson's disease (PD).

**Background:** a majority of PD patients will develop functionally significant cognitive decline and A $\beta$  plaque deposits have been implicated as a major contributor. Transgenic Alzheimer disease mice treated with Apomorphine show a decrease of intraneuronal A $\beta$  and an improvement of memory function. In cognitively normal PD patients, a neuropathological study has shown that ante-mortem Apomorphine exposure significantly reduces A $\beta$  deposition in the brain.

### **Design/Methods:**

preliminary descriptive cross-sectional open label analysis of seven PD patients on chronic continuous Apomorphine infusion treatment who underwent [<sup>18</sup>F] florbetaben PET/CT scan, as surrogate marker for brain amyloid deposition, as part of the APOMYL study at King's College Hospital (London UK). Amyloid was scored as present or absent. Clinical data was collected from medical records and Non-motor Longitudinal International Study (NILS; UKCRN No: 10084). Data is presented as mean (range).

**Results:** seven PD patients (mean age 70.6 (55.6-80.6) years, disease duration 14.1 (5-23) years, median Hoehn-and-Yahr stage 4 (2-5) had undergone amyloid PET/CT scans. The patients were on continuous Apomorphine infusion (daily dose 61.9 (45-96) mg) for 51.1 (10-180) months. Mean Mini Mental State Examination score was 27.2 (24-29), Non-motor symptom (NMS) questionnaire score 11.9 (6-15), NMS scale total score 65.0 (50-88) and SCOPA scores 20 (14-28) for part A, 14 (6-18) for part B, and 5 (1-6) for part C. None of the patients showed abnormal Ab accumulation on PET/CT.

### **Conclusions:**

Although open label, this study provides the world-first real-life evidence that Apomorphine infusion may be protective against cerebral A $\beta$  accumulation in PD. Control data is required to confirm this finding and studies are in progress.