ANDROGEN INTERVENTION THERAPY IN A PATIENT WITH FAMILIAL ALZHEIMER'S DISEASE

V.B. Gupta1, H. Sohrabi1, R. Clarnette2, A. Zentner3, A. Paton2, K. Taddei1, R. Martins1

1Edith Cowan University, 2University of Western Australia, 3Independent Practitioner Network, Perth, WA, Australia

Introduction: Mutation in the presenilin-1 (PS-1) or amyloid precursor protein (APP) genes results in autosomal dominant Familial Alzheimer’s disease (FAD), in which the onset of the disease occurs at an unusually early age (onset under the age of 60). Previously, androgen modulating therapy for AD has been proposed by our group based on published animal and clinical studies.

Aim: We now report on a symptomatic mutation carrier of FAD prior to and following testosterone intervention therapy.

Methods: We investigated clinical and blood biomarker correlates in this female aged 32 years. We monitored blood plasma levels of Amyloid beta 1-42, amyloid beta 1-40, clusterin, total apoE and TNF-alpha during the course of this treatment.

Results: Neuropsychological testing revealed a MMSE of 17. The plasma biomarker levels before testosterone implant at two different time points (3 weeks apart) showed amyloid beta 42 (97.52 pg/ml; 115.43 pg/ml) amyloid beta 40 (148.87 pg/ml; 136.17 pg/ml) total apoE (18.5 mg/dl; 11.5 mg/dl), clusterin (450.21 µg/ml; 453.10 µg/ml), TNF-alpha (28.6 pg/ml; 38.08 pg/ml). A repeated analysis of the same panel of biomarkers was then carried out one month after treatment with 200mg of testosterone pellet implant. Testing revealed a MMSE of 18 and plasma biomarkers showed amyloid beta 42 (87.13 pg/ml), amyloid beta 40 (155.46 pg/ml), total apoE (12.5 mg/dl), clusterin (420.34 µg/ml), TNF-alpha (10.86 pg/ml).

Conclusion: The amyloid beta 42/40 ratio, clusterin and TNF-alpha levels have significantly decreased and total apoE levels showed an increase (p< 0.05) after one month of testosterone treatment (P< 0.01).