Increased presence of the sphingolipid pathway in Alzheimer’s disease with capillary cerebral amyloid angiopathy

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Cerebral amyloid angiopathy (CAA) is frequently observed in Alzheimer’s disease (AD) and is marked by deposition of amyloid beta (Aβ) in leptomeningeal and cortical brain vasculature. In over 40% of AD cases, Aβ accumulates in cortical capillaries, a phenomenon referred to as capillary CAA (capCAA), which is associated with loss of tight junction proteins and a reduced function of P-glycoprotein, indicating impaired function of the blood brain barrier (BBB) and decreased transport of amyloid beta across the BBB. Increasing evidence suggests that an altered sphingolipid (SL) metabolism contributes to Alzheimer’s disease. However, to date it remains unknown if alteration of the SL pathway is involved in capCAA pathogenesis. In this study we set out to investigate the alterations of the different players of the SL pathway in capCAA. Expression and localization of ceramide, sphingosine-1-phosphate (S1P) receptors (S1P1, S1P3) and the enzyme involved in ceramide production, acid sphingomyelinase (ASM), were assessed using immunohistochemistry on post-mortem tissue from the occipital cortex of non-neurological controls, AD and severe capCAA cases. Increased immunoreactivity for ceramide, S1P3, S1P1 and ASM was observed in capCAA cases compared to non-neurological controls and AD cases. Immunoreactivity for ceramide and S1P3 was primarily observed in astrocytes, whereas immunoreactivity for S1P1 and ASM was observed in microglia. In capCAA, all SL markers showed high levels of immunoreactivity around amyloid-laden capillaries and correlated with the presence GFAP and HLA-DR as markers for glial activation. We find increased presence of SL pathway markers in AD cases with capCAA. The increased presence of these markers in glial cells associated amyloid-laden capillaries, suggests that the SL pathway is involved in the neuroinflammatory response in capCAA pathogenesis. Future studies are needed to address the functional role of the SL pathway in capCAA pathology.

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Anthracnose of lucky bamboo *Dracaena sanderiana* caused by the fungus *Colletotrichum dracaenophilum* in Egypt

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*Dracaena sanderiana*, of the family Liliaceae, is among the ornamental plants most frequently imported into Egypt. Typical anthracnose symptoms were observed on the stems of imported D. sanderiana samples. The pathogen was isolated, demonstrated to be pathogenic based on Koch’s rule and identified as *Colletotrichum dracaenophilum*. The optimum temperature for its growth ranges from 25 to 30 °C, maintained for 8 days. Kemazed 50% wettable powder (WP) was the most effective fungicide against the pathogen, as no fungal growth was observed over 100 ppm. The biocontrol agents *Trichoderma harzianum* and *T. viride* followed by *Bacillus subtilis* and *B. pumilus* caused the highest reduction in fungal growth. To the best of our knowledge, this report describes the first time that this pathogen was observed on D. sanderiana in Egypt.

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