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A case of juvenile idiopathic arthritis plus dermatitis similar in two siblings treated by different remedies and how homeopathy applies to the healing process

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Introduction: A paradigmatic familial case demonstrating the rules of homeopathy in curing illnesses.

Case description: A two year old girl suffering from a juvenile idiopathic arthritis and her eight year old brother affected by a serious genital and retroauricular hemorrhagic keratotic dermatitis were both cured exclusively with homeopathic remedies. Interestingly the girl's joint disease has improved with Bryonia remedy together with the worsening and spreading of her skin eruption (typical evolution from internal to external and from head, peripherally), which partly resembled the hallmarks of her brother's manifestations. Particularly the girl's skin eruption, its timing and site of appearance has given the key for choosing between two different homeopathic remedies (Medorrhinum alternate with Sulphuriociatum), necessary for the disappearance of the symptoms in one year time.

Discussion: These two cases could give the clue of a profound analysis regarding the practice of the complementary medicine in serious chronic complicated cases but homeopathy can specially contribute to the understanding and treatment of a chronic disease, beginning from the concept of illness as an individually specific dynamic process. The homeopathic treatment can be seen as an individually specific epigenetic reprogramming, aimed to the eradication of the chronic disease, the epigenetic programming is indeed a progressive and constant process of cellular and tissue genomic adaptation to the local and systemic environment. The homeopathic remedy would give coherent information, able to directly modify the morphology of the system's electromagnetic field.

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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 $(A\beta)$ in leptomeningeal and cortical brain vasculature. In over 40% of AD cases, $A\beta$ 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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