Fluid dynamics of venous flow in multiple sclerosis

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Background

Venous insufficiency in multiple sclerosis (MS) has often been associated with immobile valves in the tributary veins of the brachiocephalic veins (BVs) and the superior vena cava (SVC). Immobile valves have, in turn, been associated with reflux pulse flow in the tributary internal jugular (IJV), azygous (AV) and vertebral veins (VVs), causing breakdown of the blood-brain barrier (BBB) and extravasation of lymphocytes into the cerebral parenchyma.1,2

Methods

The physics of fluid dynamics is used to analyze the transition from laminar to localized vortex generation with periodic reversing flow and points of flow stagnation at curvatures, at tributary flow confluences with the BVs and SVC and at valve leaflets proximal to the confluences. Chaotic flow is associated with the development of venosclerosis plaques at such points. This analysis predicts plaque formation and valve immobility most prominently in the left IJV, secondarily the right IJV and tertiarly in the AV and VVs as observed in clinical trials. Vortex generation is also associated with valve leaflet elongation and the growth of flaps and septa as observed clinically.3 Standing wave patterns in the IJV, AV and VVs, which are dependent on venous compliances, are predicted to cause stagnant and periodic reversing flow in the DCVs, resulting in disruption of the blood-brain barrier, also as observed clinically.4

Results

For typical venous structures of the BVs, SVC, IJVs, AV and VVs, fluid dynamics predicts localized vortex generation with periodic reversing flow and points of flow stagnation at curvatures, at tributary flow confluences with the BVs and SVC and at valve leaflets proximal to the confluences. Chaotic flow is associated with the development of venosclerosis plaques at such points. This analysis predicts plaque formation and valve immobility most prominently in the left IJV, secondarily the right IJV and tertiarly in the AV and VVs as observed in clinical trials. Vortex generation is also associated with valve leaflet elongation and the growth of flaps and septa as observed clinically.3 Standing wave patterns in the IJV, AV and VVs, which are dependent on venous compliances, are predicted to cause stagnant and periodic reversing flow in the DCVs, resulting in disruption of the blood-brain barrier, also as observed clinically.4

Conclusions

Applying fluid dynamics to flow transition from laminar to chaotic in compliant tubes with curves and confluences provides substantial insight into the causality of vein scleroses and subsequent disruption of the BBB in MS.

References