Blue-green algae or cyanobacteria in the intestinal micro-flora may produce neurotoxins such as Beta-N-Methylamino-L-Alanine (BMAA) which may be related to development of amyotrophic lateral sclerosis, Alzheimer’s disease and Parkinson-Dementia-Complex in humans and Equine Motor Neuron Disease in Horses

Blue-green algae, or cyanobacteria (CB) are the source of Beta-N-Methylamino-L-Alanine (BMAA), a neurotoxin found in the brains of patients with Amyotrophic Lateral Sclerosis (ALS), Alzheimer Disease (AD), and Parkinson-Dementia-Complex (PDC) [1].

BMAA acts through multiple mechanisms including the N-Methyl-D-Asparate (NMDA) receptor, Glutamate 5 receptor and oxidative stress, in the nervous system [1].

A similar disease to ALS occurs in horses, Equine Motor Neuron Disease (EMND), when they are deficient in Vitamin E and restricted from fresh pasturage [2].

The source of CB associated BMAA has been considered environmental, however CB are in the intestinal micro-flora of both men [3] and horses [4].

Usually CB are a minor component of the intestinal micro-flora, however disease or malnutrition may alter the proportion between pathogens and the normal bacterial micro-flora, enabling overgrowth of CB with production of neurotoxins such as BMAA, with subsequent development of neurodegenerative diseases, in both horses and men.

Assessing intestinal contents for the presence of CB, BMAA and other CB neurotoxins such as saxitoxin and anatoxin-a, could be helpful in determining the cause of ALS, AD and PDC in humans and EMND in horses.

If CB in the intestinal micro-flora produce neurotoxins, it may be possible to control them through diet or medications, in the treatment of such diseases of the nervous system.

References


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doi:http://dx.doi.org/10.1016/j.mehy.2012.10.010

How Doppler effect occurs in absence of intracranial blood flow in brain death?

Lack of intracranial arterial and venous circulation (cerebral circulatory arrest) is major confirmatory criterion of brain death diagnosis. Circulatory arrest can be demonstrated by angiography, single photon emission computed tomography or transcranial Doppler ultrasound (TCD). TCD is more practical mainly due to its bed-side applicability [1]. TCD sonogram patterns compatible with brain death are to-and-fro pattern (TaFP, complete diastolic flow reversal), systolic spikes (SS) and flow absence within internal carotid arteries (ICA) and vertebral arteries, bilaterally [2,3]. Flow absence cannot be used for confirmation unless there is a loss of definite flow signals previously recorded through the same bone window and hemodynamic condition.

The pathophysiology underlying these sonogram patterns (sometimes erroneously referred as flow patterns) are not fully evaluated. In brain death, intracranial pressure is higher than systolic blood pressure preventing blood entry into the intracranial cavity. Accordingly, these sonographic patterns are significantly correlated with the level of flow arrest in angiography. The TaFP signal corresponds to contrast blockage at the supraclinoid segment level, the SS at the petrous segment level and flow absence at the cervical segment level [4]. This observation in turn raises the question of how these TCD signals are generated in absence of any blood flow within intracranial cerebral arteries. The usual explanation has been the detection of flow reversal or just systolic entry (in case of SS) into the rostral extracranial ICA by TCD sample volume side lobes. We, however, propose another mechanism.

Given absence of any angiographic flow in cerebral vasculature in brain dead cases, the cause of the Doppler effect observed cannot be a realflow at all. On the other hand, the stagnant blood column in cerebral arteries is moved back-and-forth due to the percussive effect of every heart beat at its proximal tip. In the early stages of brain death, the forward push and then backward movement of this blood column leads to a TaFP pattern on TCD. With further deterioration, the clot retracts and becomes stickier to push, thereby eliminating any movement in the blood column; at this stage the sonogram turns into SS pattern probably only reflecting the percussions of the heart beat. Change in SS amplitude by the phase of respiration, observance of increase in SS amplitude or sometimes reappearance of TaFP pattern after blood pressure augmentation in a case with SS supports this idea further. Another explanation could be that the signals might reflect heart beat related alternative compression and relaxation within the blood column, rather than en-bloc back and forth movements. But, the amplitude of the signal should be very small if this mechanism plays a role.

Therefore, we propose to refrain from using the word ‘flow’ while describing TCD patterns observed in brain dead cases, until the nature of these sonographic signals are better understood.

Conflict of interest statement

None declared.

References


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doi:http://dx.doi.org/10.1016/j.mehy.2012.11.002