



CCSVI – Some More Thoughts

Ashton Embry

Last month I described the basics of the concept of chronic cerebrospinal venous insufficiency (CCSVI) which undoubtedly marks the start of a new era in understanding and treating MS. In this issue, Ian Cook has followed up on CCSVI and has focused on currently-available treatments for relieving CSSVI as well as a major research project which will be done at the University of Buffalo over the next few years. In this column I want to address a few issues that have arisen with the advent of the CCSVI concept. These include the role of CCSVI in MS, the historical roots of the concept, the current response of the main elements of the MS community to CCSVI, and what might be done to get treatment of CCSVI available as soon as possible.

The idea that CCSVI is the cause of MS is already being bantered about. I can certainly understand this but to me such a way of thinking is not productive. What need to be answered in this regard are questions such as “Does CCSVI play a major role in the MS disease process?” and “Does CCSVI come before or after the onset of CNS autoimmunity?” To me I think we can answer the first question with, “Given the current data it is almost a certainty that CCSVI is a key part of the MS story”. I say this because 1) almost everyone with MS tested at four different research centres in three different countries exhibited CCSVI, 2) CCSVI explains a number of previously puzzling characteristics of MS lesions such the association of iron and the venocentricity, and 3) CCSVI is theoretically compatible with all we know about MS. Notably, inclusion of CCSVI as an important part of the MS disease process provides a much improved explanation of all the features of MS.

It is much harder to answer the second question. Currently, there is no evidence or theoretical reasoning that supports a model that has CCSVI following the onset of autoimmunity. It is much more reasonable to assume CCSVI precedes autoimmunity because such a model is theoretically sensible and has been sporadically postulated by MS researchers over the past 150 years. In regards to CCSVI coming first, some researchers have suggested that the venous blockages are congenital and are in place at birth. This is doubtful given that adequate vitamin D can prevent MS but right now our ignorance of how and when CCSVI is generated is profound.

The concept that MS is primarily a vascular disease can be traced back to 1863 when a researcher named Eduard Rindfleisch proposed that “the primary cause of MS is an alteration of individual blood vessels”. He based this interpretation on his consistent observations that a vein was present in the centre of each lesion. Over the next 100 years, the vascular basis for MS resurfaced every so often based on the close association of lesions with veins but never really took off due to a lack of any convincing evidence. The last main champion of the concept was Dr Roy Swank who thought venous blockages and resultant breaches in the blood-brain barrier were due to fat globules. This led him to advocate for his well known dietary therapy for relieving the fat-induced, vascular problems.

Over the last 60 years, the vascular hypothesis was completely overshadowed by the autoimmune theory for MS which came into vogue in the 1930s and which has become steadily more popular since that time. It must be emphasized that the vascular hypothesis has never been disproved; it has been simply ignored because of the complete focus on immunological aspects of MS.

Of course, with the discovery of CCSVI in virtually every person with MS, we finally have that elusive, convincing evidence that vascular problems are indeed a major part of MS. CCSVI marries all the vascular-related data gleaned from detailed studies of MS lesions with the huge immunological data base which has accumulated over the past 50 years. The two different data sets now fit very well together in a model in which impaired venous drainage causes breaches in the blood-brain barrier (vascular hypothesis) which then leads to autoimmune reactions (autoimmune hypothesis).

With the recognition of CCSVI and the resultant much better understanding of the MS disease process, one might expect that MS researchers and clinicians would be falling over themselves to redirect their efforts to take into account this major discovery. Unfortunately this is not the case and the main reaction from the clinicians is, at best, one of wait and see or, at worst, total rejection. One person with MS I know recently asked their neurologist about CCSVI and how they might get it treated. The neurologist became agitated and stated that CCSVI was nonsense and there was no evidence for it. I expect such an uninformed reaction is commonplace. MS clinicians are content to simply prescribe an MS drug and they don't want to think about a completely new way of treating MS until they have to.

The MS research community has exhibited a similar, highly skeptical reaction to CCSVI and I expect they will not even consider incorporating CCSVI into their research plans until the data become so overwhelming they will have little choice but to do so. So where will this overwhelming evidence come from? The good news is that there are a few far-sighted researchers who have seen the obvious “paradigm shift” nature of CCSVI and are starting to undertake significant CCSVI studies. The most progressive of these are Drs Bianca Weinstock-Guttman and Robert Zivadinov of the University of Buffalo whose planned research is discussed by Ian Cook in this issue. I believe once the results of the Buffalo study are announced (2012?), CCSVI will experience a tipping point and everyone from researchers, to clinicians, to the main MS charities, will get on the CCSVI bandwagon.

Given the data we have, it is not a stretch to say that all persons with MS have impaired venous drainage which is actively, or potentially, contributing to their MS disease progression. Given this, it is not surprising that every person with MS who understands the implications of CCSVI wants to have their venous problems resolved by vascular surgery as soon as possible. Unfortunately very few medical centres are doing vascular procedures to relieve CCSVI and thus it is basically impossible for almost all persons with MS to get the important vascular treatments they need.

It is hard to know how the MS patient community can improve this appalling situation and get relief for both CCSVI and the great frustration many are feeling. It seems to me a patient revolt is needed and it is also essential that this unacceptable situation be widely exposed through the media. Perhaps persons with MS should follow the advice given in the 1976 movie, Network, and go to the window and shout “I am as mad as hell and I am not going to take this anymore”

(<http://www.youtube.com/watch?v=WINDt1PXmmE&feature=related>).

Finally, another obvious question which needs to be answered is what nutritional strategies can be adopted to offset the effects of CCSVI. I will address this in my next column.