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Letter to the Editor

Binswanger disease may benefit from omental arteries

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Dear Sir,
It is rare that a limited medical article written years earlier will have potential clinical significance a quarter of a century later. However, this situation could prove possible in the future treatment of Binswanger disease, a form of dementia that is considered progressive with no cure.

In preparing publications on Alzheimer's disease (AD),[1-7] a form of dementia was encountered that was unknown to the author, namely, Binswanger disease. It became evident that Binswanger disease and AD can both lead eventually to dementia, however, the diseases are completely different in their pathological conditions involving the deliverance of cerebral blood flow (CBF) to brain tissue.

The decrease in CBF in AD has been believed over the years to have been caused by the death of cerebral neurons which no longer require significant CBF. However, there is presently increasing consideration that in AD, it is not the death of neurons that cause a decrease in the level of CBF, but it is actually the decrease in CBF that slowly leads to the death of critical cerebral neurons.

Binswanger disease is completely different from AD, in that it is not a decrease in CBF that eventually leads to dementia, but it is excessive arteriosclerosis in cerebral arteries that narrows the lumen of the arteries causing a restriction of CBF flowing through the vessels supplying the subcortical areas of the brain.

Otto Binswanger lectured for the first time regarding the disease, eventually named after him, at the annual meeting of the German Lunatic-Physicians in Dresden, Germany in 1894.[12] He claimed that he had followed eight cases for 11 years, and that there was "a strong atheromatosis of brain arteries," which he explained lead to white matter atrophy as a result of chronic diffuse nutritional disturbance. Because of the increasing use today of improved magnetic resonance imaging (MRI) techniques, there is a greater opportunity to observe abnormal arteries in the brain that may lead to an increase in the diagnosis of Binswanger disease that previously would have been classified as AD.[11]

The arterial pathology involved in Binswanger disease brings to mind a personal experience involving the omentum that occurred in the autopsy laboratory in the 1960–1970s. The author became interested in the use of the absorptive properties of the omentum for the treatment of the lymphedema that developed in breast cancer patients following radical mastectomy and radiation therapy.[8] Much time was spent in the autopsy facility observing the omentum in cadavers to learn the arterial system within the omental apron and to conceptualize various surgical techniques by which the omentum could be mobilized to distant areas of the body with its blood supply remaining intact.

While studying arteries in cadavers, which included the aorta, renal and iliac vessels, there was clear evidence of severe arteriosclerosis especially in older individuals. In contrast, omental arteries seen in cadavers were consistently found to be soft and pliable with no evidence

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of arteriosclerosis. When anatomical pathologists were asked if they were aware of hardening of omental arteries, they reported that they had never observed that finding.

I became increasingly interested in the lack of arteriosclerosis in human omental arteries. Based on observations that had been made in human subjects, a study was carried out in monkeys to confirm what had been observed in humans. Twenty cynomolgus monkeys (Macaca Fasicularis) were fed an atherogenic hypercholesterolemic diet for 12 months consisting of 2% cholesterol and 10% butter by weight. In five of the monkeys the experimentally induced atherosclerosis was accelerated by the surgical production of hypertension produced by coarcting the thoracic aorta.

After consuming this atherogenic diet for one year, the animals were sacrificed and autopsied. Segments of aortic, coronary, carotid, cerebral, renal, iliac, and omental arteries were removed and fixed in 10% formalin and subsequently imbedded in carbowax for histologic sectioning. Sequential histologic sections were cut and stained with hematoxylin and eosin (H and E), oil red-O for lipid, van Gieson for collagen fibers, Verhoeff or Weigert for elastic tissue and Toluidine blue for glycosaminoglycans. The quantitative estimation of the area of the lumen of the excised vessel was obtained from sections stained with Verhoeffs–Van Geison.

All 20 monkeys had developed severe and extensive atherosclerosis of the aorta, coronary, cerebral, and peripheral arteries. In the five monkeys with hypertension, the disease was especially advanced in the coronary, carotid, and cerebral vessels. In contrast to these findings, omental arteries were observed to be normal in appearance. Upon transection, radiological and histological examination of the monkey's arteries confirmed the absence of atherosclerosis.

The observation that omental arteries in humans and monkeys were devoid of atherosclerosis became the basis for a short Letter to the Editor of *The Lancet*.[9]

The article was published in 1990 under the title, "Lack of Atherosclerosis in Omental Arteries" [Figure 1]. It was sent to the *The Lancet* simply as an interesting observation with no expectation of any clinical significance.

In Binswanger disease, the pathological change that develops within cerebral arteries limits CBF through the narrowed lumen of the involved arteries and results in decreased CBF to subcortical areas of the brain. Since it has been shown that omental arteries are devoid of atherosclerosis and that placing an intact vascularized omental pedicle on AD brains has lead to cognitive improvement,[7,13,14] Binswanger disease might be treatable by placing an intact vascularized omental pedicle directly on a Binswanger brain which would increase CBF[10] through omental arteries that are known to be free of atherosclerosis. This finding would compensate for the decreased CBF flowing through the narrowed lumens of the pathological arteries found in Binswanger disease.

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Lack of atherosclerosis in omental arteries

SIR,—I have studied the omentum over the past 25 years in man and in animals, and I have consistently observed the absence of atherosclerosis in omental arteries. This anti-atherosclerotic capacity has been noted in small omental arteries as well as in the large gastroepiploic vessels sited within the omental apron. This sparing of omental arteries has even been observed in the presence of generalised and severe atherosclerosis. I have asked many pathologists over the years if they had ever noticed atherosclerosis in omental vessels and I have yet to hear an affirmative answer. The long-term administration of a high-lipid diet in monkeys also fails to affect omental arteries despite widespread atherosclerosis throughout the vascular system.

Omental tissue has proved to contain angiogenic factors,[1] neurotransmitters,[2,3] and neurotropic substances.[4] Clinicians have long appreciated the anti-infectious and antineoplastic characteristics of the omentum. Does the omentum also have a detectable anti-atherosclerotic factor? If such a substance could be identified, it might have clinical potential.

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Figure 1: A short article published in *The Lancet* a quarter of a century ago

CONCLUSION

The diagnosis of AD is made radiologically by observing changes in the hippocampus and deposition of amyloid plaque. These findings have warranted the diagnosis of AD. If, however, atherosclerotic arteries are radiologically observed in the brain by MRI, the diagnosis of Binswanger disease should be entertained. If blood flow to the subcortical areas of the brain in Binswanger patients could be increased by omental placement on the brain, it seems reasonable that a small carefully controlled study should be undertaken.

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