Impacts of the Evolutionary Concepts of Cancer on the Study of Human Diseases

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OKUYAMA, S. Impacts of the Evolutionary Concepts of Cancer on the Study of Human Diseases. Tohoku J. Exp. Med., 1992, 168 (2), 445-448 — Evolutionary concepts of cancer (Okuyama and Mishina 1984; 1990) have already been found useful in understanding carcinogenesis and anti-cancer defense lines in the body and various cancer attributes. The concept of endosymbiotic disorders gave an impact upon the science of internal medicine leading to discovery of amitosis in Reed-Sternberg cells, a primitive eukaryotism (Okuyama 1991a). A novel concept of vasogenic necrosis was also developed for such diseases of Perthes' disease, cerebral vascular diseases and myocardial infarction: costs of bipedalitic evolution in man. ———— bipedality; cancer; evolution; Reed-Sternberg cell; vasogenic necrosis syndrome

The evolutionary concepts of cancer are important in describing fundamental changes: (1) devolution of cancer; (2) its evolutionary history; and (3) its age distribution (Okuyama and Mishina 1990). Their impacts are to be seen upon the study of Hodgkin's disease and diseases of vasogenic necrosis.

Hodgkin's disease is characterized by the presence of peculiar neoplastic cells called Hodgkin (H) and Reed-Sternberg (RS) cells. Their origin still remains unsettled. One of my suggestions is to follow Einstein's way of thinking: every category of cells so far claimed as probably candidate origin cells of H and RS cells (Okuyama 1991b). I hypothesize that all of those cells are potentially capable of undergoing that kind of specific cellular transformation under specific circumstances: uneven division and bizarre nuclear configurations. H cells represent those RS cells whose polyploid nuclei have not yet committed to discrete nuclear division. This amitosis may imply that these cells could have trespassed upon the domain of the primitive eukaryotism. The observation is remarkable because it was a morphological confirmation of our evolutionary concept of cancer. The presence of amitosis in a eukaryotic host implies a sort of mosaicism.

Bipedalitic burdens are especially heavy in the brain, heart and hip joint. I hypothesize that a category of diseases in these organs represent costs of bipedalitic evolution in man (Okuyama et al. 1991): vasogenic necrosis syndromes. Its prototype syndrome is Perthes' disease. The arterial circle of

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Hunter (HAC) is located in the epiphysis and metaphysis of a long bone. It receives 2 or 3 afferent arteries to nourish the bone. The hemodynamic load of bipedality amounts to 2.5 folds that of the quadrupedality. Growth and a concomitant defective HAC are both responsible. Thus, it is a pre-HAC disorder (Fig. 1A).

The vascular changes leading to generation of cerebral hemorrhage and infarction are different from each other: arterial microaneurysms (MA) for the former and primary atherosclerosis (AS) for the latter. Nonetheless, their initiating pathological process is an intimal damage in common. In such a heavy brain, the blood supply takes place from below in erect postures, and any specific cerebral functions have to be supported by acutely but on a supply-on-demand basis for the sake of economy and superoxide toxicity. The vasculature is so designed that ICA's blood flow is biased almost directly to MCA (Fig. 1B). The capacitance of Willis' circle is really not so great. Suwa's theorem of cerebral vasculature is also critically important: (a) to increase the blood flow rate; (2)
to lengthen the arterial branches of larger diameters and (c) to minimize BP fall (Suwa et al. 1963). AS in the proximal ICA is usually severe in the first 2 cm and arises in the posterior wall. Then, the aortic ejecta are twisted and exert shearing force upon ICA's posterior wall at its origin as well as cranialward impact. Therefore, cerebral infarction is primarily pre-WAC.

MA are another sequelae of intimal damage propagated and perpetuated through reactions relative to superoxide radical toxicity. Superoxide anions are produced because of aging-related uncoupling of blood flow and oxygen consumption in the basal ganglial territory (Yamaguchi et al. 1986), marring the post-WAC arterioles. Once should it take place, blood torrents as predicted by Suwas's theorem are also to amplify the damage to MA formation. This is one of the evolutionary loci minores.

Myocardial infarction (MI) is a necrotic sequela of coronary artery obstruction. AS plaques are formed principally in the proximal coronary arteries around 3 cm from the origin, and may embolize. Bipedalitic evolution demands an increased cardiac activity (Shannon et al. 1991). In the heart, a functional coronary arterial circle, CAC, is formed with the column of blood as soon as the aortic valve closes and the peripheral resistance to the cardiac outflow or back pressure establishes itself (Fig. 1C). CAC has an ample capacitance, and provides steady blood flow at reduced pressure, pulse pressure and velocity. This CAC-post CAC balance will be critically damaged through 2 mechanisms: (i) pre-CAC risk factors of civilization of excessive food intake including salt, alcoholism and cigarette smoking; and (ii) post-CAC AS formation. AS's primary predisposition to the proximal coronary arteries represents mechanical insults during the cardiac activity. Vibration at sites of branching emerging with uneven branching as in the case of air lift will foster formation, dislodgement and further embolic metastasis of AS plaques. Such post-CAC mechanical insults could have aggravated with the bipedalitic evolution as the heart is freely suspended in erect postures.

In this way, analysis of human diseases as viewed from an evolutionary point will probably lead us to better understanding them and expedite invention of more efficient and logical principles and devices of diagnosis and treatment. Thus, the importance of the evolutionary concepts cannot be overemphasized in the field of internal medicine, too.

References


