cells themselves were almost of normal size but the nuclei showed some atypism and decreased chromatin. In the molecular layer, there were a lot of myelinated fibers forming a network. Between the granular layer and the molecular layer, corresponding to the Purkinje cell layer, there were collections of abnormal nerve cells which were sending the myelinated fibers into the molecular layer and partly into the granular layer. These cells contained Nissle substance and some of the cells had PAS-positive granules. These were calcareous deposits in the molecular layer and a small angiomata was found on the surface of the cerebellar cortex.

The second and the third cases were of some similar clinical and histological characteristics as the case one.

According to Christensen (1937) and Courville (1958, 1960), the origin of the abnormal nerve cells of this disease was thought to be Purkinje cell and they called this disease "Purkinjeoma". However, other authors insisted that the abnormal nerve cell came from granular cell. According to the histological evidence of our Case 1., the origin of the myelinated fibers in the molecular layer was the medium sized nerve cells between the molecular layer and the granular layer and this nerve cells had the characteristics quite different from that of both Purkinje and granular cells from the point of their shape and the pattern of their fibers. These abnormal nerve cells were mainly collected along the Purkinje cell layer but in places, they were also scattered diffusely in the molecular layer. However, there was no histological characteristics indicating these cell were of neoplastic nature.

The most appropriate explanation of the origin of this disease was a malformation delivered from some disturbance of the morphogenesis of the three layers of the cerebellar cortex.

69. Extraneural Metastasis of Intracranial Tumors

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