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PUNCTIFORM HEMORRHAGIC PIGMENTATION OF THE CEREBRAL PIA MATER¹

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In a careful search through the literature, including the well-known works of Mott, Nonne and Oppenheim, I have been unable to find any reference made to what is here described as punctiform hemorrhagic pigmentation of the pia mater.

Ameuille reported numerous minute hemorrhages in the white substance of the subcortex and the occurrence of small hemorrhagic foci in the pons was described recently by Rimbaud. In a syphilitic case Spiller found hemorrhages in the medulla oblongata and cord in chronic poliomyelitis, and similar extravasations occurred in the acute case reported by Mills.

Large and diffuse subarachnoidal or pial effusions have been mentioned as occurring secondarily to infectious diseases, such as anthrax and scarlet fever, in so-called purpura of the nervous system (Ameuille and Lenoble) and following traumatism or rupture of an aneurysm of one of the basal arteries.

An interesting case of such extravasation involving the pia of a large part of the brain was reported by Leopold. These conditions of diffuse subarachnoidal effusions of blood are all

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most likely due to a toxic weakening of the blood-vessel walls with subsequent large effusions, but owing to their large size, distribution and distinctly hemorrhagic nature they cannot be confused with the minute areas of pigment such as are present in the specimen here discussed.

Obersteiner refers to pigment cells found normally in the pia over the base of the brain and particularly on the anterior surface of the medulla oblongata but pathological extracellular pigment is not mentioned.

The gross specimen of the brain in this condition showed numerous minute brownish pigmented areas of pin-point size.

A microscopical examination of these punctiforme areas showed them to be clumps of granular pigment. They were mostly independent of the blood vessels but the presence in several places of similar pigment granules, though in much less number, immediately around as well as within the vessel walls suggested the probability of their hematogenous origin. Such granules were also scattered in very small numbers throughout the cortical substance but always in close proximity to the vessels. The pigment is for the most part free in the tissue, though some can be found to be intracellular.

The presence at one point in the pia of what looks like an aneurysmal enlargement forming a thickened connective tissue nodule with free pigment and blood cells extending from it into the cortical tissue also shows the blood to be the most evident origin of these areas of pigment.

The luetic infection in this case with its consequent arterial thickening and weakening would doubtless predispose to such condition.

While the pigment would react neither to the test for melanin nor to the one for hemosiderin, it is possible that the material was either too old or the preservative used not a proper one for the obtaining of these reactions. The presence, however, of a faint blue reaction in some of the blood-vessel walls, showing the presence of albuminate of iron, would lend contradiction to such a possibility. The pigment may be in the stage mentioned in the work of Claude and Loyez, in which the ferrocyanide test is not active.

By a careful comparison of the sections with specimens of a melanotic sarcoma, stained by similar methods, there seemed to be a distinct difference between the two pigments.

With the hemalum-fuchsin stain the granules were yellower than the melanin and with artificial light in addition to their being yellower, they showed, by oblique illumination, a glistening metallic, reddish copper colored appearance totally unlike the muddy black color of the melanin. This metallic luster is present, though much less distinct, in sections stained by Weigert's and Nissl's methods, while the melanin sections do not show this high refractive power by any stain. Nissl's stain showed the granules to be dark brownish, those of melanin black. Weigert's gives black color while the color of the melanin pigment is a lighter brownish black.

The general appearance of the pia is that of a case of syphilis. The round cell infiltration is very marked and plasma cells are present in great number. The vessels are thickened and in some of them the adventitial coats are thickened and infiltrated, while in others the overgrowth is confined mainly to the intima.

I am indebted to Dr. Wm. G. Spiller for the privilege of reporting the following case.

G. W., white, age 58, laborer. Admitted to the Philadelphia Hospital February 13, 1907. Family history and patient's early history negative. Present illness began eight days before admission, at which time patient fell from weakness while working but remained conscious. Further history was not obtainable owing to unreliability. The patient denies the use of alcohol, although the first thing he asked for upon awakening in the morning was a "five-cent whiskey." The patient's wife stated that on December 24 the patient was about to go to bed when he suddenly became nauseated, dizzy, and weak in the legs, particularly in the right leg. He fell to the floor, hitting his face, vomited freely and the bowels were evacuated. He was put to bed and remained there one week. Then he got up and seemed well except that at times he was mentally confused. He walked around the house but never returned to work. January 15 he seemed quite bright, but about ten o'clock he had an attack very much like the above and vomiting lasted all night, and till about noon the following day. The patient since then had been mentally confused, though at times he talked rationally, answering questions. He would sleep during most of the day but was very restless during the night, throwing the covers off, etc., but never making much noise. The patient had also been confused at times during the last year.

Examination.—The right lower face seemed paretic—speech was slow and thickened, though the patient said that he had always lisped a little.

The pupils were large and equal and reacted promptly to light

and accommodation. There was some ptosis and the left palpebral fissure was wider than the right.

Extraocular movements were normal except upward associated movement which was somewhat impaired.

Tongue deviated slightly to the right on protrusion, and showed some coarse tremor.

Upper extremities were normal in power and movement—no atrophy. Some ataxia, however, was seen in the finger to nose test.

Lower limbs were normal in size and strength, though there was some ataxia by heel to knee test. Knee jerks and Achilles' tendon reflexes exaggerated. A suggestion of ankle clonus was present on the right side. No Babinski on either side.

No vesical disturbance but occasional loss of control of bowel.

Patient's mind was not clear but confused and semi-stuporous. He had to be restrained in bed during the night. He presented the picture of senile confusion with alcoholism.

Dr. Spiller examined him and the notes are as follows:

Patient was decidedly stuporous but he could be partially aroused. Would not do anything he was commanded to do.

The right side of the face was not moved so much as the left during his mumbling. Pin prick on the right side caused him great discomfort and he raised his head and moved the left arm very feebly. The right arm was not moved at all. The biceps and triceps tendon reflexes were normal. He was not aphasic. The left lower limb was moved freely when stuck with a pin; the right only feebly and only at the hip. The patellar reflexes were slightly exaggerated—the Achilles' tendon reflexes were very prompt. The Babinski sign was not obtained on either side.

February 15, '07. The patient had become more stuporous and more restless when awake—struggling a great deal. His nose became reddened and swollen, this swelling extending to the left side of the face.

February 16, '07. Patient had been doing very well—temperature had fallen but in the morning suddenly became very high. Respiration became short, jerky and rapid. There was dullness over the right lung posteriorly, extending anteriorly to anterior axillary line. Numerous râles could be heard over this area. The general condition became gradually weaker in spite of all stimulation, and the patient died at 9 p. m.

For microscopical examination sections were made from the medulla oblongata, and from the cervical, thoracic and lumbar regions of the cord. Hemalum-fuchsin, Nissl's and Weigert's stains were used.

The examination of the cortex shows the pia thickened and intensely infiltrated with round cells and a large number of plasma cells. The vessels are sclerosed, thickened and congested. The ganglion cells are very weakly stained, numbers of them degenerated and the glia may be somewhat increased. Clumps of yellowish pigment as previously described are beneath the pia.

In the medulla oblongata the round cell infiltration of the meninges is very marked and the vessels are congested, some of them being so distended and their walls so indistinct that the appearance is that of an intrapial hemorrhage.

Streaks of yellow pigment of irregular sizes and shapes are around some of the blood vessels (hemalum-fuchsin stain).

The cells for the most part are very weakly stained and with a few exceptions, probably degenerated.

In the cervical region the meninges appear thickened, congested and infiltrated and considerable fibrinous thickening is present.

The vessel walls are thickened and many of them hyaline in appearance. Surrounding or close to them are stringy looking masses stained bluish by the Weigert method. The fibers of the anterior and posterior roots are beaded and to a great degree degenerated. In the cord medulla the neuroglia is slightly increased and some of the vessels are congested, but there does not appear to be any degeneration of fibers or tracts of fibers. The ganglion cells are irregularly affected, in some instances staining very poorly, and in many the nuclei are partially disintegrated or entirely gone.

Amyloid bodies are very numerous and show a tendency to linear arrangement along the septæ or the vessels and are particularly thick along the posterior septum. The central canal is large and patulous and the ependymal wall is broken down.

In the sections from the thoracic region the changes are almost the same as in the foregoing. Some of the pial vessels have their walls so disintegrated that the different coats can not be distinguished from the surrounding infiltrated tissue and cells. With the exception of a few fibers to the cells of Clarke's columns none can be found at the entering root zones. The cells of Clarke's columns are degenerated, very few of them having any nuclei and these only indistinct and poorly staining ones. The anterior horn cells are not so numerous as in the normal and some of them are degenerated. In those apparently normal there is an unusual prominence of the protoplasm granules by the acid fuchsin stain.

The central canal is obliterated and the group of ependymal cells seems abnormally large.

In the lumbar segments the findings are about the same with the exception of the presence of a more intense congestion of the capillaries of the gray substance of the cord and a more definite and pronounced overgrowth of the intima in the blood vessels of the pia.

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