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CLINICAL PATHOLOGIC REPORT OF A CASE OF PONS HEMORRHAGE (TYPE FOVILLE)*

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Hemorrhages in the pons are much less common than in the brain, such as in the region of the internal capsule, for instance. According to Millard (1) Rochaux found among 130 cases of intracranial hemorrhages only six instances in the pons, and Andral, according to the same authority, saw but nine cases among 386, while Dana (2) found among fifty cases only two in the pons.

The clinical picture of pontine hemorrhage is remarkable for its polymorphism resembling in some, though rare, cases that of ordinary capsular hemiplegia [Nothnagel (3), Dana (2)]. In the great majority of cases, however, it exhibits a characteristic clinical picture of so-called hemiplegia alternans, a term introduced by Gubler in 1856 (4).

The hemiplegia alternans, type Millard-Gubler, came to be known as hemiplegia alternans inferior, in distinction from hemiplegia alternans superior (Gubler-Weber type). In 1858, Foville described another pontine syndrome which Grasset (6) in 1900 proposed to call syndrome Foville. This represents the Millard-Gubler syndrome

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combined with paralysis of the external (VI nerve) and internal recti muscles (III nerve) of the eye (corresponding to the side of the lesion), strabismus and diplopia consequently being absent.

In 1901 (7) and more fully in 1903 (8) Raymond and Cestan described a pons syndrome in which hemiplegia was associated with contralateral involvement, usually very slight, of the V, VI, or VII nerves and sensory disturbances (loss of deep sensibility and hypalgesia in the V nerve area) combined with incoördination, tremor, athetosis, asynergy and other signs of cerebellar disturbances. The syndromes of Millard-Gubler, Foville, Raymond-Cestan, indicate not only a lesion of the pons, but also of a definite portion of the same. Thus, Millard-Gubler type denotes a lesion of the basal portion of the pons; the Foville type—the tegmental portion of the pons; the type Raymond-Cestan—likewise the tegmental portion, but its lateral portion including the cerebellar pathways.

The polymorphism of the clinical picture of pontine hemorrhages is thus due to the part of the pons involved, its extent and severity. The case to be reported is an instance of pontine hemorrhage, type Foville, which presents interesting clinical and pathologic features.

Report of Case. A fifty-two years old colored female, with good family history, entered the neurologic service of Cook County Hospital on October 18, 1920, complaining of swelling and numbness of the right half of the face, weakness and numbness of the entire right half of the body, buzzing in the right ear and difficulty in walking, speaking and swallowing.

On the day prior to admission, she suddenly became dizzy and felt numbness in the right half of the body. She had not lost consciousness, nor had convulsions. She denied venereal or any other infection and also miscarriages. Five years previously she had "kidney trouble."

Examination. The face was disfigured, its left half was flat, the left corner of the mouth drooping, the nasolabial fold on the same side obliterated, the forehead (on the left side) could not be wrinkled. The left eye was kept open and could not be closed, while the right eye showed a tendency to drooping—she could not keep it fully open for a certain length of time.

The pupils were equal and regular, but contracted and failed to react to light or in accommodation. The conjunctival and corneal reflexes were lost on the left and present on the right. Both eyes were turned toward the right, could be moved up and down and to the right, but not to the left. There was a slight horizontal nystagmus.

The movements of the jaws, head, tongue, soft palate and pharynx were normal, but the taste sense in the anterior part of the tongue, as well as the sensibility and the reflexes of the soft palate and pharynx were lost.

The right leg and arm were incompletely paralyzed, movements in both these extremities being possible but very weak. The tendon reflexes were present on both sides, without Babinski, Oppenheim or other pathologic phenomena. Brudzinsky and Kernig signs were absent, as were atrophies or trophic disorders.

Sensibility of the entire right half of the body, including the face, showed greatly diminished sensation for pain and touch, while the *temperature sense was preserved*. Stereognostic, localization, and position senses were lost on the right, the patient being unable to tell change of position in her right fingers, hand, forearm, leg and toes. The left side of the body showed no abnormalities whatever, as to the motor or sensory system.

The heart was enlarged, the second aortic sound accentuated, the first sound replaced by a murmur. The lungs, gastrointestinal,

Fig. 1: The hemorrhagic focus in the tegmental region causing marked protrusion of the floor of the fourth ventricle. (The tissues are somewhat disfigured as the brain was poorly preserved.) Photograph of an unstained specimen.

genitourinary organs, mentality were normal. Blood pressure was 210 systolic and 120 diastolic.

The urine showed albumin and a few granular casts. The spinal fluid: absence of globulin, eight lymphocytes per cubic millimeter, a negative Wassermann reaction which was also negative for the blood. The temperature was normal, the pulse 88, respiration varied from 24 to 36.

The vestibular apparatus (by Drs. Novak and Profant) had been examined twice, on October 22 and 24, in the ear service of Cook County Hospital. The caloric tests revealed normal vertigo and past pointing from both right and left horizontal canals and a feeble nystagmus from the right horizontal while the left horizontal did not respond by nystagmus at all. The vertical canals gave a feeble response in both nystagmus and past pointing.

Summary. (1) Complete left sided facial paralysis (of the peripheral type); (2) paralysis of left lateral movements of the

eyes (persistent deviation to the right), that is, of the left external and right internal muscles; (3) contracted and fixed pupils; (4) right sided hemiparesis and hemianesthesia with involvement of sensation for pain, touch, position, stereognosis and localization, but with preservation of the temperature sense; (5) involvement of problematical fibers from both vertical labyrinthine canals and partial involvement of the fibers from horizontal canals, especially the left one.

Fig. 2: Arteries of the base; the larger one is the basilar artery which shows no evidences of syphilis (absence of cellular infiltrations of its layers), but shows marked arteriosclerotic changes (thickened and hyperplastic intima, splitting of the elastica and even changes in the media (hyaline-like mass between the internal and external elastica fibers). Weigert's Resorcin-fuchsin stain X 21.

The patient died from bronchopneumonia on the eleventh day after admission and the twelfth day of her illness.

Macroscopic and microscopic reports (Dr. Hassin). The post-mortem examination revealed a hemorrhagic focus (three-fourths of an inch by one inch in size), in the left tegmental region of the pons (Fig. 1) occupying an area containing the nuclei and roots of the

sixth and seventh nerves, the left posterior longitudinal and prae-dorsal bundles, the reticular formation, the main or mesial fillet and partly the fibers of the descending root of the fifth nerve.

The focus was confined strictly to the foregoing region and did not transgress the raphe from which it was separated by a narrow normal zone. It did not involve the fourth ventricle into which the floor was markedly protruding (Fig. 1). Nor did the hemorrhage transgress the annular or basal portion of the pons. In front it

Fig. 3: Choroid plexus (of the fourth ventricle) with fairly large globules of haemosyderin in the tuft-cells. Alzheimer-Mann stain X 300.

reached the region of the posterior corpora quadrigemina and did not involve the lateral lemniscus, the brachia conjunctiva or the spinocerebellar tracts. The horizontal extension of the hemorrhagic focus, near the artificial cut, corresponded approximately to the area occupied by the spinotectal and spinothalamic tracts which thus have been affected.

The basilar and vertebral arteries were markedly sclerosed (Fig. 2) and patent while the microscopic examination of the rest of the brain tissues showed no changes suggestive of syphilis or tuberculosis, nor any parenchymatous or inflammatory phenomena.

The temporal lobes and the cerebellum were excessively vascularized, the pia mater was thickened, containing, especially around the paracentral and frontal lobes, an abundance of fibroblasts, mesothelial cells and masses of granules of hemosyderin. They were mixed with a few lymphocytes, but no plasma cells could be found. Hemosyderin granules were likewise found in the perivascular adventitial spaces of smaller vessels and capillaries of the medulla, of the white brain substance (especially of the occipital lobe) and, in larger quantities, in the epithelial, tuft cells, of the choroid plexus (Fig. 3) which appeared grossly vacuolated.

The spinal cord and the abdominal organs could not be examined because of the restrictions placed on the postmortem examination. It was assumed that the principal pathologic lesion was in the nature of arteriosclerosis of the brain with subsequent rupture of the median branch of the basilar artery.

DISCUSSION

A typical Foville syndrome has been associated clinically with fixed and contracted pupils, dissociated touch and temperature sensations, slight hemiplegia, low temperature, interesting vestibular findings, and pathologically with the presence of blood pigment in the subarachnoid space and choroid plexus, and a well defined hemorrhagic focus in the left tegmental region of the pons. As the latter involved the nuclei and roots of the *left* sixth and seventh nerves, their paralysis is easy to understand. The simultaneous involvement of the *right* internal rectus was most probably due to involvement of the left posterior longitudinal bundle whose fibers connect the nucleus of the left sixth nerve with that of the fibers for the contralateral internal rectus muscle. The external rectus and the contralateral internal rectus muscles, though supplied by different nerves (sixth and third) are both dominated by one common center which according to Bechterew (9) is located in the second frontal convolution and is represented solely by the nucleus of the sixth nerve. Fibers from this center run through the centrum semiovale, internal capsule and the cerebral peduncles to the pons where they decussate before reaching the pontile nuclei of the sixth nerves. Lesion of the above fibers before their decussation produces a deviation of the eyes toward the cerebral lesion and away from the ocular paralysis, while their involvement after decussation, in the pones, will produce an ipsilateral paralysis of the nerves in the present case, on the left side. The result was that the eyes had to turn to the right because of the action of the contralateral nerve, and away from the lesion, the patient looking at the paralyzed side of the body. The

simultaneous involvement in our case of the right rectus internus muscle which gets its innervation from the third cranial nerve was due, not to the involvement of the latter which was found intact, but, as noted, to that of the posterior longitudinal bundle. Bechterew describes fibers running from the sixth nucleus into the area of the posterior longitudinal bundle of the same and opposite sides.

While some plausible explanation can be given for the associated ocular paralysis it is much more difficult to explain the cause of the rigid and myotic pupils. That the pupils in pons lesions may be contracted is a well known fact, but rigid or sluggish pupils are rather an uncommon occurrence in pontile hemorrhage. Nothnagel (3), Wernicke (10) and Gee-Tooth (11) mention the pathologic pupillary reaction in such cases but the majority of authors have found the light reaction to be prompt. The most frequent cause of abnormal pupillary reaction, such as syphilis, was absent in our case, and the explanation therefore must be looked for elsewhere. Some authorities, Dana (2) for instance, explain pupillary changes in pons lesion, by assuming compression or irritation of the third nerve nuclei, or by probable coincident palsy of the sympathetic nerve. The experimental studies of Levinsohn (12) and others showed that irritation of the fifth nerve nuclei or of the descending root of this nerve may produce a pupillary contraction. Such an explanation, however, in our case is altogether unsatisfactory for it would not explain the *bilateral* involvement of the pupils from a unilateral pontile lesion. Lutz (13) points out that a marked myosis may obtain from an interruption of the descending fibers of the posterior longitudinal bundle which goes from the anterior corpora quadrigemina to the centrum cilio-spinale. If the lesion, he says, destroys Meynert's fountain-like crossing in the median line, it produces *bilateral* myosis; if it acts only on one side, unilateral myosis follows.

As Meynert's crossing in our case was not involved, the lesion having stopped at the level of the posterior-corpora quadrigemina, we must assume that these fibers or some other structures such as the sympathetic nerve fibers, have been either compressed by the neighboring hemorrhage or in some other way damaged and thus produced the pupillary contraction.

A better anatomic explanation might be given for the rigid pupils. They were of course due to a lesion of some portion of the pupillary reflex arc, pupillary optic fibers, corpora quadrigemina and oculomotor nerve. Here should be included the praedorsal longitudinal bundle, also called fasciculus tecto-bulbaris praedorsalis [Pavlow (14)] which according to Van Gehuchten (15) and his school

contains fibers transmitting the light reflex from the pupillary fibers and their center in the anterior corpora quadrigemina to the third nerve nucleus. As this praedorsal bundle was destroyed in our case and as it contains crossed fibers, it is highly probable that its lesion was instrumental in the causation of the frozen pupils.

It is much easier to explain the motor as well as sensory phenomena. The former were due to pressure on the pyramidal fibers, which were not destroyed, nor markedly damaged. This would account for the absence of the Babinski and Oppenheim phenomena as well as for the exaggerated tendon reflexes.

The total destruction of the left mesial or main lemniscus explains the loss of the sense of touch and deep sensibility (position and stereognostic senses); the involvement of the spinotectal and spinothalamic tracts—that of pain, while the involvement of formation reticularis with its ventral tectal pathways which represent, according to Spitzer (16), the sensory pathways of the fifth nerve, will explain the sensory disturbances in the areas of this nerve (face). Preservation of the temperature sense is due to the intactness of the corresponding fibers which occupy a territory not affected by the hemorrhage. In short, this case demonstrates that the sensory pathways in the pons are dissociated just as they are in the spinal cord and that there are different fibers for the conduction of temperature sense.

The vestibular findings, absence of nystagmus, upon irritation of both the vertical and horizontal canals can be explained by the involvement of the posterior longitudinal bundle. Whether this case speaks for or against the assumption by some authors, Randall (17), Fischer (18), Jones (17), of separate pathways from the horizontal and vertical canals to the cerebellum, it is rather hard to tell, as the patient's condition did not make possible repeated or extensive examinations along this line.

Finally, a few words may be added as to the histologic features, the presence of blood pigment in the subarachnoid space and the choroid plexus. That it came to these structures from the hemorrhagic focus cannot be questioned. The pathways by which the blood pigment traveled are indicated by the presence of the pigment in the perivascular spaces by which it was carried from the brain to the subarachnoid space, just as any other foreign, useless substance is removed from the brain tissues. This problem has been discussed elsewhere (19) and here I just wish to direct attention to an additional fact which speaks in favor of the view that the unloading of

the useless cerebral substances, in this case blood pigment, takes place into the subarachnoid space.

The presence of pigment in the choroid plexus seems also to favor the view that the spinal fluid, derived wholly from the brain tissues, before being absorbed by the various channels becomes purified by the cells of the choroid plexus (20). The presence of the blood pigment in the spinal fluid is not only of academic, but also of great practical interest. For it can be demonstrated *intravital* by means of spinal puncture and the benzidin test (for blood). In hemorrhages of the brain the spinal fluid always gives a positive benzidin test, while in embolism and thrombosis it is always negative. In my service, at the Cook County Hospital, such examinations have been carried on for the last eighteen months, and the results uniformly supported the views here expressed.

BIBLIOGRAPHY

1. Millard. *Gaz. hebdomaire de méd. et de chir.*, 3, 1856, 816. *Bulletin de la Soc. Anat. (Paris)* 1856, May and June, pp. 206, 217.
2. Dana, Ch. Acute Bulbar Paralysis due to Hemorrhage and Softening of the Pons and Medulla. *Med. Record*, 64, 1903, 361.
3. Nothnagel, H. *Ziemssen's Cyclopaedia of Practice of Medicine*. Am. Ed. New York, 12, 1877, 139.
4. Gubler, A. De l'hémiplégie alterne envisagée comme signe de lésion de la protubérance annulaire et comme preuve de la decussation des nerfs faciaux. *Gaz. hebdom. de méd. et de chir.*, 3, 1856, 749, 789, 811.
——— *Mémoire sur les paralysies alternes en general, et particulièrement sur l'hémiplégie alterne, avec lésion de la protubérance annulaire*. *Gaz. hebdom. de med. and chir.*, 5, 1858, 721, 765, 801, 835.
5. Foville, A. Note sur une paralysie peu connue de certains muscles de l'oeil, et sa liaison avec quelques points de l'anatomie et la physiologie de la protubérance annulaire. *Bull. Soc. Anat. de Paris*, 33, 1858, 393.
6. Grasset. Un type spécial de paralysie alterne motrice (type Foville). *Rev. Neurol.* 8, 1900, 586.
7. Raymond, F. and Cestan, R. Trois observations de paralysie des mouvements associés des globes oculaires. *Rev. Neur.*, 9, 190, 70.
8. Raymond, F. and Cestan, R. Le Syndrome protubérantiel supérieur. *Gaz. des hopitaux, Lançette Française* 76: 1903, 829.
9. Bechterew, W. Ueber eine Affection der Varolschen Brücke mit bilateraler Lähmung der willkürlichen Augenbewegungen. *Deut. Ztschr. f. Nervenhe.*, 17, 1900, 221.
10. Wernicke, C. *Lehrb. der Gehirnkrankheiten*. 2, 94, 1881 (Case of Crichton Brown, *Lancet*, 1875, Feb. 6).
11. Gee, S. and Tooth, H. Hemorrhage into pons, secondary lesions of lemniscus, post. long, fasciculi and flocculus cerebelli, Brain. 21, 1898, 1.
12. Levinsohn, G. Beiträge zur Physiologie des Pupillenreflexes. *Arch. für Ophth.* 59, 1904, 191.
13. Lutz, A. The light-pupillary Reflex. *Arch. of Ophthalm.* 47, 1918, 370.
——— A Case of Alternating Hemiplegia (Millard-Gubler) with Remarks on the paths of the Pupillo-dilator and vestibulo-ocular fibers in the brain stem. *Am. Arch. of Neur. and P.* 5, 1921, 166.
14. Pavlow. Le faisceau long. predorsal ou faisceau tecto-bulbaire. *Le Névra.* 1, 1900, 59.

15. Van Gehuchten. Anatomie du Système Nerveux. Louvaine, 1906, p. 936.
16. Spitzer, A. Ein Fall von Tumor am Boden der Rautengrube. *Jahrb. f. Psych.* 18, 1899, 1.
See also, Goldstein, Kurt. Ueber die aufsteigende Degeneration nach Querschnittsunterbrechung des Rückenmarks. *Neurol. Centralbl.* 29, 1910, 898.
17. Randall, A. and Jones, I. The Ear Tests of Barany in Locating Cerebellar or other Encephalic Lesions. *The Amer. Journal of the Med. Sc.* 151, 1916, 515.
18. Fischer, L. *Penn. Med. J.*, 1916, Dec.
19. Hassin, G. B. Histopathology of Brain Abscess. *Am. Arch. Neur. and Psych.* 3, 1920, 616.
20. Hassin, G. B. Histop. Findings in a Case of Superior and Inferior Policephalitis with Remarks on the Cerebrospinal Fluid. *Am. Arch. Neur. and Psych.* 4, 1921, 552 (May).