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BRAIN PURPURA AND HAEMORRHAGIC LEUCO-ENCEPHALITIS

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Up to the turn of the century under the influence of Strümpell and Leichtenstern, the pathological process in the brain substance in which multiple petechiae seem to be an essential feature, was held to be of inflammatory origin and called haemorrhagic encephalitis. At the beginning of the present century M. B. Schmidt differentiated this picture from the inflammatory conditions and explained it as resulting from toxic vascular changes. In recent years, chiefly A. W. Hurst, and Henson and Russel contend that beside this noninflammatory brain purpura, there also exists an inflammatory form, the so-called haemorrhagic leuco-encephalitis. H. Jacob goes one step further, placing the cases of purpura so far held to be toxic of origin, into the same class with the seroushaemorrhagic inflammations of the nervous system. According to Mc Ardle, van Bogaert, and Lhermitte, in cases with simultaneous inflammation and destructive processes it is difficult to differentiate between primary encephalitis and secondary inflammation, since various processes (infections, intoxications, mechanical circulatory disorders) are capable of producing an identical morphological picture, at least in certain phases of the disease.

The following four cases may contribute to the clarification of the morphological similarities and differences existing between brain purpura and haemorrhagic leuco-encephalitis.

Case No. 1. B. T., a boy, aged 2 1/4 was observed in the Paediatric Department of the University of Pécs (Prof. Kerpel-Fronius), he showed elevated temperature and general weakness a week before his death, and had developed jaundice the last two days, vomiting repeatedly. On admission, the case looked one of grave septic jaundice, with the child on the verge of unconsciousness, having a set, fixed, unsteady look, a rigid neck, and Babinski's and Kernig's signs were slightly positive. The temperature was at first normal, but after a few hours it rose to above 39° C. Red blood count, 1.400.000 ; white blood count, 36.000. Reticulocytes, 72 per million. Thrombocytes, 1.000.000. Differential count, Jg. 2 ; St, 5 ; Sg, 68 ; Eo, 1 ; Ba, 0 ; Mo, 2 ; Ly, 22. Serum bilirubin 2,6 mg per 100 ml. After a hospital stay of half a day the child died.

Autopsy (Department of Pathological Anatomy, Medical University of Pécs, Prof. Entz). Pneumonia in both lower lobes ; fibrous purulent pleurisy.

Brain. On the surface of the brain no pathological changes were visible. Coronal sections revealed innumerable tiny punctate petechiae, confined to the white matter and in greatest number in the rostrum of the corporis callosum (Fig. 1.). They were situated symmetrically and subcortically with a sharp line of demarcation immediately beneath the U fibres but, as a rule, without reaching them. Towards the ventricles they became scanty, almost to disappear

in the centrum semiovale. There were no petechiae in the cerebellum, in the pons, and in the bulb. The walls of the ventricles, and the choroid plexuses were normal.

In all four cases under review, several petechiae-containing and haemorrhage-free areas of the white matter, and portions of the centres and brain stem were subjected to microscopical examination, employing Nissl's, haematoxylin-eosin, van Gieson's, Spielmeyer's, Sudan's, Bielschowsky's, Masson's, Mallory's method for glia, and for connective tissue.

Microscopical examination (Fig. 2.). Chiefly ring haemorrhages are present in the form of narrow, homogeneous zones mostly around vessels, surrounded by glia and enclosed, beltlike, by red blood corpuscles. In the centre of many ring haemorrhages, and, in some instances, also in the vessels around them, hyaline thrombi, staining yellow according to van Gieson, are detectable. The central vessel was always found to be a capillary. Its wall is often homogeneously thickened, in other instances decomposed into fine fibres and more than once unrecognizable, having presumably melted into the surrounding necrotic tissue. Around vessels of precapillary or postcapillary character no haemorrhages are visible but effusion of plasma is not unfrequent around them. No inflammatory elements are demonstrable. No demyelination around the vessels, or in any other place. Generally, the myelinated fibres are only pushed apart, but occasionally the axis cylinders are fragmented and their ends swollen.

The histological picture is characterized by ring haemorrhages and by capillary thrombosis.

Case No. 2. Sz. S., a girl, aged 12 (Paediatric Department of Baja Hospital, Head, Dr. Ujsághy). History revealed that because of fever for two weeks, she had been given Pyramidon tablets at home. Repeated bleedings of the nose, difficult to stop. On admission, a medium-developed and average-nourished patient was seen in a grave state ; with reduced turgor, and pallid skin On the dorsal surface of both thighs bluish discolourations with distinct borders and the size of a thumbprint. In the right corner of the mouth a crust-covered defect of about fingerprint size. There was foetor ex ore. Continous bleeding from nose and mouth. Liver and spleen not palpable. Cardiac sounds faintly audible ; pulse 140. Swollen lymph nodes were palpable in several regions. The patient was incontinent. Sensorium clouded ; no answers to questions. Temperature around 39° C. Death occurred a week after admission. Red blood count around 1.000.000, notwithstanding repeated transfusions. White blood count, 1200, later 600 Hgb, 20 per cent. Differential count, Seg, 2% Ly, 98%. Sedimentation rate, 180/hour. Number of thrombocytes and reticulocytes practically zero. No medullar elements in the sternal punctate, only a few red corpuscles and ripe lymphocytes.

Autopsy (Pathologist, Dr Cseh, Baja). Tonsils in state of gangreneous decomposition. In the mucous membranes of the ileum and the colon crust-covered circumscribed necroses. Red infarct in the lower lobe of right lung. Bonemarrow of the sternum atrophied, that of the femur fatty. Siderosis in the parenchymal organs. Diagnosis : panmyelophthisis.

Brain : In the white matter of the brain, in the cerebellum and the pons, there are innumerable, tiny, pinpoint petechiae, with a faint spot in their centre almost invisible to the naked eye. They are situated subcortically and simmetrically, becoming scanty towards the ventricles.

Microscopical examination. Only one section was available from each the cerebrum and the cerebellum. In these, ring haemorrhages were visible mostly without a vessel in their centre. In the erythrocytic zone, too, blood cells were only recognisable in restricted numbers. The principal characteristic of the petechiae is a broad glia wall (Fig. 3). There are no signs of inflammatory reaction. With Mallory's stain the white matter seems diffusely loosened.

Brain purpura is known to accompany diseases of the haemopoietic system. Its association with panmyelophthisis gives our observation a special interest.

Case No. 3. Zs. J., a woman, aged 50. Six months previous to her death she had undergone tonsillectomy without complication, on account of recurrent polyarthritis, but otherwise had no disease worth mentioning. She had been apparently well, occupied with housework, when she suddenly collapsed, became dazed and confused, later unconsious. Next day admission to hospital in a dying state. Temperature 40° C. Pupils miotic ; deep reflexes of the lower extremities absent. Patient died 60 hours after onset of disease. The lues reactions were negative in the blood.

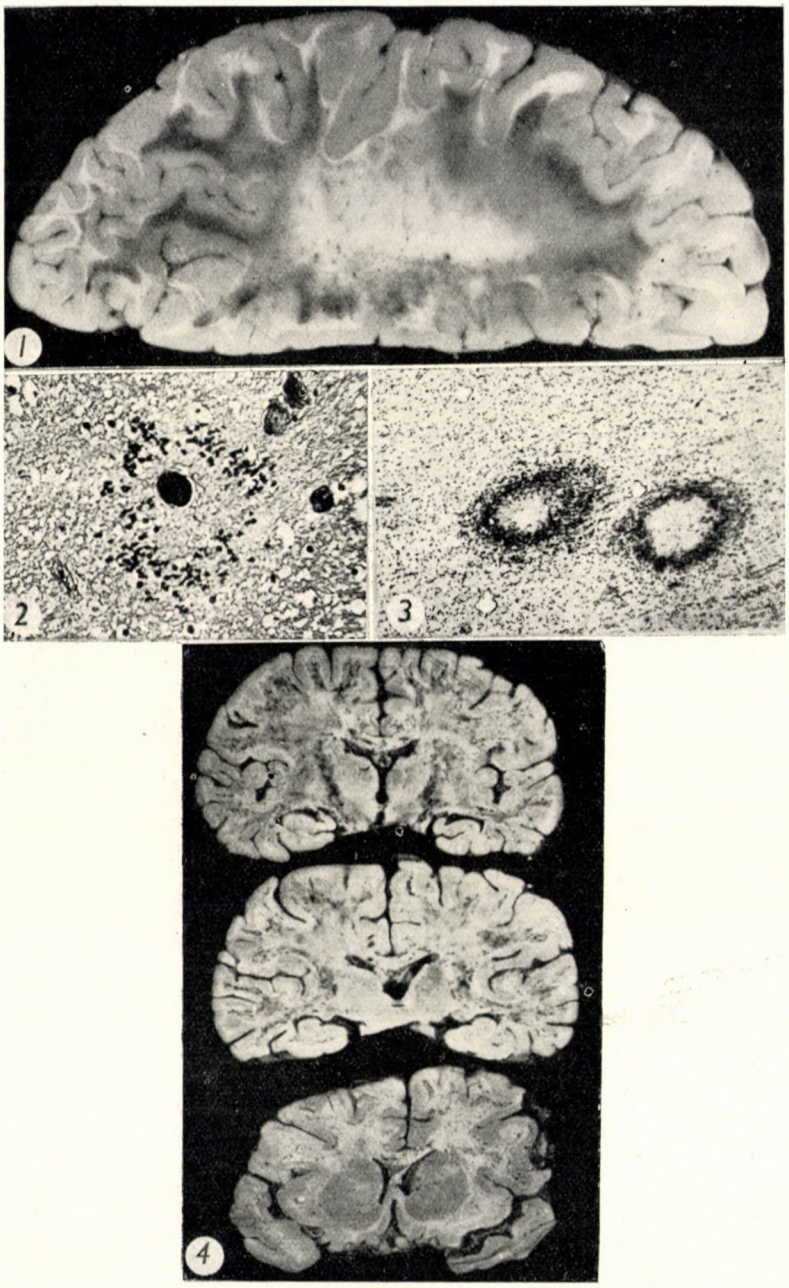

Fig. 1. Numerous punctate haemorrhages in the subcortex barring the U fibres
Fig. 2. Ring haemorrhage. Homogeneous perivascular area surrounded by red blood corpuscles (black in the picture). Thrombus in the vessel lumen. Masson's stain
Fig. 3. Broad perinecrotic glial proliferation. Nissl's stain
Fig. 4. Symmetrically located subcortical petechiae, becoming fewer in the centrum ovale but numerous again in the internal capsule and the corpus callosum

Autopsy revealed oedema of the lungs, and confluent subpleural and subpericardial haemorrhages.

Brain. Weight 1195. g. Leptomeninges markedly hyperaemic, convolutions slightly flattened. Transversal cut surfaces showed innumerable petechiae of poppy-seed or even millet-seed size, situated close to each other; sharply demarcated groups of them were dispersed practically over the whole of the white matter, fairly simmetrically in both hemispheres. They were present in the greatest numbers in the convolutioned white matter, scantier in the centrum semiovale, but all the more numerous again in the corpus callosum and the capsula interna (Fig. 4). Petechiae in large numbers were visible also in the white matter of the cerebellum, the pons, and spinal cord, in which the process wass less severe in the lumbar segments than in the cervico-thoracic part.

Microscopical examination. Compact ball haemorrhages without any particular glial reaction. They are sharply circumscribed. In smaller numbers, there are also some ring haemorrhages to be found. In their centre there is a homogeneous mass, evidently a necrotised vessel wall which stains a bluish red with trichrome and is surrounded by a narrow necrotic zone outside of which a broad zone built up by erythrocytes is lying. There are also foci in which only a few red corpuscles can be seen evenly scattered around the vessel whereas the glial tissue is loosened by extravasation of blood plasma. On myelin staining, demyelinated areas of pinhead size are visible to the naked eye. Under the microscope, they show sharply defined borders (Fig. 5.), and against an unstained background many normally stained and shaped myelin sheaths can be recognised, in which elements are in about the usual number present and only a few erythrocytes are scattered. Wherever a vessel can be seen in the centre, it is a capillary around which a tissue detritus in lying. In ball haemorrhages no demyelination is observable, yet they contrast with the enviromental medullary substance due to their massive content of blood cells. There is no fatty disintegration. Around the central capillary of some of the larger petechiae, scattered polynuclear leucocytes can be recognized. With silver impregnation for axis cylinders a similar picture was obtained to that described in case 1. Apart from a hyperemia, the meninges show no pathological lesions. The intracerebral vessels are also hyperaemic, but the individual erythrocytes can readily be identified, and there are no thrombi.

Case No. 4. K. T., a woman, aged 44, admitted on August 22, 1944. The history contained nothing worth mentioning. Her illness had begun three days before admission, with sudden severe headache, nausea, uneasy sleep, and anorexia. One day before admission, she lost her speech without unconsciousness, became confused, trismus and urine retention developed. On admission, blood pressure 165/100 mm mercury, pulse 66/min. Physical and radiological examinations showed no pathological changes in the internal organs. The patient was entirely uncooperative. High degree of sensitiveness in left frontotemporal region and slight signs of a lesion of the pyramidal tract on the right side. There was slight nuchal rigidity. The cerebrospinal fluid was slightly yellowish, its proteincontent 0,60 mg per 1000 ml., cell count, 30/3; complement fixation test for syphilis, negative; bic. mastix reaction 0,01, 2, 3, 4, 3, 2, 1, 0,0. The temperature keept gradually rising, the general state deteriorated until death occured 11 days after the onset of the illness.

Autopsy: Disseminated bronchopneumonia in the inferior lobe of the left lung: serofibrinous pleurisy, chronic endocarditis, and sclerosis of the aorta. Pneumonia and pleuritis were secondary developments shortly before death. The endocarditis was of several years' duration and compensated.

Brain. Walls of basal arteries appeared normal. Leptomeningeal vessels were congested and the leptomeninges very slightly blurred and pinky. Convolutions normal. Transversal cut surfaces presented hyperaemia of the white matter. In both hemispheres, largely simmetrically situated punctate haemorrhages were visible in some regions showing a tendency to confluence. The haemorrhages were localised chiefly in the anterior and posterior part of the internal capsule, in the lateral parts of the corpus callosum and the cerebral peduncle, in the brachia conjunctiva, and in the radiation of the splenium corporis callosi over the posterior horns. In the last-mentioned region the haemorrhages coallesced and in the medullary substance overlying the posterior horn softened. No haemorrhages were present in the bulb in which occasionally a larger vessel seemed dilated. The grey matter was everywhere free from haemorrhages.

Microscopical examination. The connective tissue of the leptomeninges seem loosened, the veins are dilated and filled with red blood corpuscles. Around the vessels there are occasionally loose round cell infiltrations. The vessels of the white and grey matter are similarly dilated and more or less filled with blood; the perivascular spaces are also distended. In the corpus callosum, the internal capsule, and the wall of the posterior horns the majority of the haemorrhages has the appearance of ball haemorrhages varying in size and compactness. The ner-

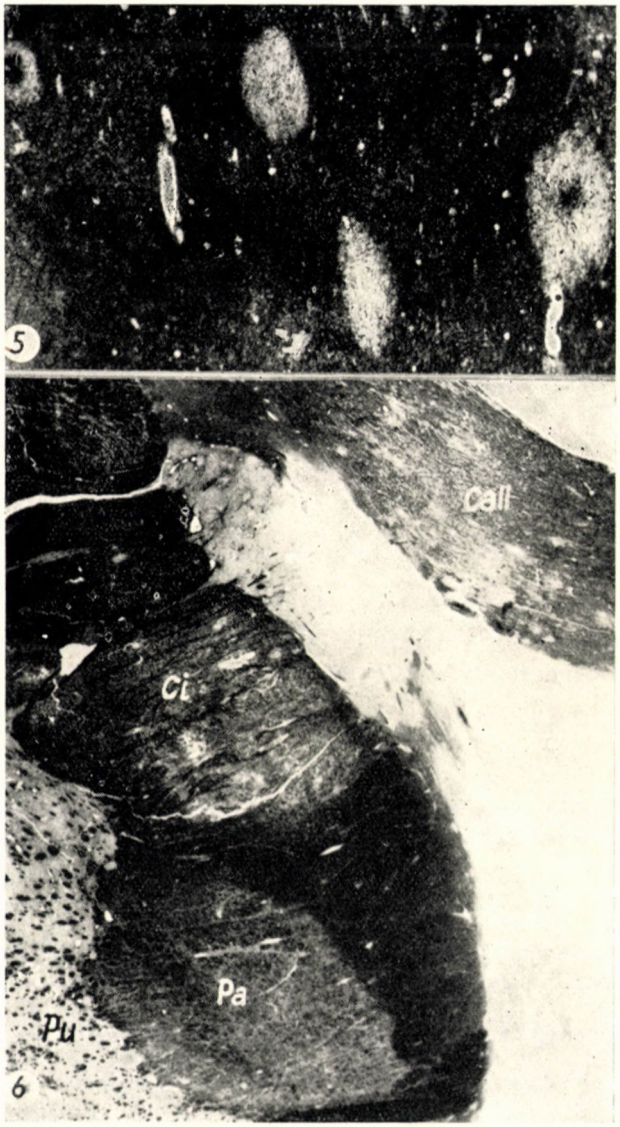

Fig. 5. Perivascular foci of demyelination. Spielmeyer's stain

Fig. 6. Circumscribed demyelination and diffuse discolouration in the corpus callosum. Diffuse discolouration in the anterior stem of the internal capsule. Spielmeyer's stain

vous tissue is often loosened in the internal capsule, the corpus callosum, and the walls of the lateral ventricle. On the margins of some bleedings and in the area between them glial proliferation with large scavenger cells is visible. In these areas, the myelin sheaths stain but poorly (Fig. 6). In the marginal parts of the glial nodules swollen and fragmented myelin sheaths and myelin globules are visible. At some places, signs of fatty disintegration. The axis cylinders are swollen and tortuous.

Discussion

Minute pericapillary haemorrhages represent the common feature of our cases. No pronounced infiltrations are found in any of them. The extravasation of serum is very substantial in Case 1, demonstrable in Cases 2, and 4, but doubtful in Case 3. While in Case 1 the myelinated fiberpattern shows only some loosening, if any change at all, in Case 3 sharply circumscribed foci of dissociated fibres are present around small perivascular necrotic zones. In Case 4, there are small indistinct foci of demyelination, and larger areas in which the myelin sheaths gained diffusely a faint colouration due to their disintegration. In the absence of sufficient material, Case 2 could not be examined satisfactorily on this point. The exudation of serum seemed to be independent of the haemorrhages. It was shown particularly in Case 1 that wile the haemorrhages had occurred around capillaries, serum effusion had taken place from veins and presumably from arterioles. In Cases 3 and 4 the nerve fibres suffered also independently of the haemorrhages.

The pathohistological analysis leads thus to the following conclusions. Case 1 must be regarded as a case of purpura, associated with extravasation of serum. In cases 3 and 4 haemorrhages and demyelination are present independently of one another. Therefore they may be considered as haemorrhagic leuco-encephalitis, in spite of the absence of inflammatory reaction.

Cases of brain purpura were described in connection with various infectious and toxic diseases, such as sepsis, influenza, nephritis, jaundice, malaria, poisoning by phosphorus, lead, arsenic, cyanide, veronal, CO, fat embolism, anaemia, leukaemia, etc. Aranovich in 1939, and Hurst in 1941, described as primary inflammatory disease the haemorrhagic leuco-encephalitis. Similar cases were reported later by Henson and Russel, and by H. Jacob. Into the same class seem to fall the cases described by Adams, Cammermayer, and Denny-Brown as »acute necrotizing haemorrhagic encephalopathy«. Baker described 20 cases in 1935, but in our opinion their place is much rather in the group of secondary purpurae than in that of primary haemorrhagic leuco-encephalitides. The 10 cases reported by Alpers as »brain purpura or haemorrhagic encephalitis« developed as sequelae of nephritis, sepsis, pneumonia, etc.

Clinically, the haemorrhagic leuco-encephalitis appears with a sudden onset amidst perfect wellbeing, in the form of acute cerebral disease. The symptoms are fever, headache, nausea, vomiting, mental confusion gradually leading

to a comatose state, signs of meningeal irritation, increased reflexirritability, pathological reflexes, often epileptiform seizures and focal symptoms. The disease lasts from a few hours to twelve days. The cerebrospinal fluid is clear or slightly yellowish-pink, sometimes showing moderate leukocytosis and containing an increased amount of protein.

Haemorrhagic encephalitis cannot be differentiated from brain purpura by changes visible to the naked eye. There are numerous tiny, punctate petechiae in the white mattter, mainly in the subcortical medulla, the internal capsule, and the corpus callosum. Their distribution is usually fairly symmetrical. In all four cases under review, the elective involvement of the white matter is distinct, not only the macroscopic but also the microscopic changes being entirely restricted to it.

As regards microscopic changes, the essential feature of brain purpura is the ring haemorrhage around a capillary. The vessels are usually dilated with stasis, or sometimes a clot, in their lumen. In some instances their walls are only attenuated, in some others broken up into fine streaks of fibres running largely parallel with each other, and yet in others irregularly thickened. These last show a homogeneous staining, as is usual in fibrinoid degeneration and melt gradually into the surrounding necrotic tissue. There are no signs of inflammation. Around the haemorrhages the myelin sheaths are distended and occasionally thickened, but there is no demyelination. Consequently we have to assume that the necrosis in the centre of petechiae is due to a fibrinoid degeneration of the vascular wall. Sometimes a broad glial layer forms around the homogeneous lumenless substance (Fig. 1 and 3).

The classical concept permits the diagnosis of haemorrhagic encephalitis only if the presence of petechiae, in contrast with the picture dealt with above, is associated with that of inflammatory cells and demyelination. Here, too, the haemorrhages are ball or ring type. The pathological changes in the vascular wall are generally the same as in purpura. The inflammatory process is characterized, apart from serum exudation, by extravasation of polymorphonuclear leucocytes, to a lesser extent by the presence of lymphocytes, and by perivascular proliferation of the microglia. The great number of polymorphonuclear leucocytes reminds one of a purulent process, as it is true for the observations of H. Jacob, Mc Ardle, Van Bogaert and Lhermitte, and Adams, Cammermeyer, and Denny-Brown. In these cases massive leucocytocic microglial infiltration reached diffusely also the grey matter.

The question arises, what pathomechanical factors play a role in the development of purpura cerebri and the acute haemorrhagic inflammation of the central nervous system. It is a generally accepted view that purpura is caused by toxic injury to the vascular walls. First, vasodilation sets in with stasis, to be followed by vasoparalysis. Then the vessel wall becomes permeable to plasma and blood cells. Lastly, the surrounding tissues necrotise owing to hypoxaemia.

A grave damage leads to thromboses and histologically demonstrable lesions on the vessel wall (Scheinker). The aetiology of acute haemorrhagic leuco-encephalitis cannot as yet be regarded as solved. Margulis, Soloviev, and Schubladze reported recovery of a neurotropic virus from the blood in one case of encephalomyelitis, and from the brain tissue in another.

Hurst suggested that the haemorrhagic and demyelination lesions represent gradually different tissue responses to the same noxious agent, and that certain interrelations are possible between haemorrhagic leuco-encephalitis and other demyelinating diseases on the one hand, and the so-called purpura, on the other.

Since in our cases the clinical history offers no support concerning the aetiology, we have to resign ourselves to conclusions from microscopic examination. Conclusions may be drawn, in our opinion, from the coincidence of petechiae, effusion of plasma, and demyelination. According to Gerlach's experimental results, hyperergic inflammation is frequently of haemorrhagic character. Serum extravasation is held by many authors (Rössle, Eppinger, etc.) to be a sign of inflammation and allergic in origin. As to demyelination, researches of the last years indicate that it is the main histological sign of neuroallergic conditions. On these grounds, the assumption appears to be justified that haemorrhagic leuco-encephalitis is really a hyperergic inflammation of the nervous system, in spite of the lack of direct proof of an antigen-antibody reaction.

Some observations show that in parainfectious encephalomyelitis the haemorrhagic character may come into prominence. Encephalomyelitis of haemorrhagic character and undisputably allergic in nature was observed by Russel following salvarsan hyperaesthesia, by Csermely after tetanus vaccination, and by Huber on repeated intravenous injection of novocaine. Giedion reported a case of haemorrhagic encephalomyelitis following vaccination. Petechiae in the white matter may also be found in parainfectious and post-vaccinal encephalitides, particularly when autopsy is carried out shortly after the manifestation of neurological symptoms. Demyelination, however, stands in the foreground of the histological picture, and haemorrhage is relegated to the background. In haemorrhagic leuco-encephalitis, the situation is just the reverse, bleeding being the dominating feature, and demyelination of secondary importance. McArdle, van Bogaert, and Lhermitte raised the point whether the difference between perivenous encephalitides and haemorrhagic leuco-encephalitis may not be dependent upon the grade of reaction of the organism, and whether haemorrhagic leuco-encephalitis is not just the peracute form of the identical process. In our opinion, the time factor can be taken to explain some of the differences between the two syndromes. In some peracute cases petechiae are chiefly formed. If, however, the disease is taking a slower course, in the wake of the haemorrhages the extravasation of plasma and inflammatory

elements will press into prominence, together with demyelination. In other words, by taking the time factor into account, it is quite possible to establish an interrelation between haemorrhagic leuco-encephalitis and allergie demyelinating encephalitis. It is therefore ventured to suggest the denomination »haemorrhagic hyperergic leuco-encephalitis«.

Környey emphasizes the wide range of histopathological changes in haemorrhagic leuco-encephalitis, leading from simple purpura to perivascular demyelination and fatty decomposition, respectively, accompanied by inflammatory cell infiltration and perivenous microglial proliferation. With all these histopathological signs present, the diagnosis seems indisputable. As, however, in most cases only a few of these signs are detectable, it is difficult to decide on a given case and may only be possible on careful and simultaneous consideration of the clinical, macroscopic, and histopathological data.

On the basis of the foregoing, the following classification of brain purpura accompanied by formation of petechiae in the white matter and of haemorrhagic leuco-encephalitis is suggested. (1) Brain purpura is always secondary, and a sequel of infectious or toxic conditions. Its essential histopathological feature is pericapillary haemorrhage, in some instances with microglial proliferation. The hemorrhage is the result of some toxic injury to the vessel walls. (2) In the pathological picture of haemorrhagic leuco-encephalitis, there appear inflammatory and demyelinating processes along with the ball and ring haemorrhages. An allergic mechanism would seem to explain the pathogenesis.

Summary

Brain purpura and leuco-encephalitis need to be differentiated histopathologically from each other. The essential histopathological feature of purpura is a ring haemorrhage without cellular infiltration or demyelination. The pericapillary haemorrhage results from a toxic injury to the vascular walls. In the histopathological picture of haemorrhagic leuco-encephalitis inflammatory and demyelinating processes appear together with ball and ring haemorrhages. In the author's opinion, a hyperergic mechanism is responsible for the condition.

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ПУРПУРА МОЗГА И ГЕМОРРАГИЧЕСКИЙ ЛЕЙКОЭНЦЕФАЛИТ

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Резюме

Опираясь на гистопатологический анализ 4-ех случаев, авторы обсуждают вопрос морфологической тождественности и разницы пурпуры мозга и геморрагического лейкоэнцефалита. На основе литературных данных и собственных исследований они считают, что разделение заболеваний белого вещества головного мозга связанных с возникновением точечных кровеизлияний, на пурпуру мозга и на геморрагический лейко-энцефалит является обоснованным.

1. Пурпура головного мозга всегда является вторичным изменением, сопровождающим какое-нибудь токсо-инфекционное заболевание. Патогистологическая сущность этого изменения заключается в перикапиллярных кровеизлияниях, в некоторых случаях в пролиферации микроглии, без воспалительной клеточной инфильтрации и без демиелинизации.

2. Геморрагический лейко-энцефалит является первичным заболеванием нервной системы, в патогистологической картине которого наблюдается кроме очаговых и кольцообразных кровеизлияний и воспалительный процесс и демиелинизация.

Возникновение этого заболевания объясняется аллергическим механизмом.