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Surface Lesions of Corpus Callosum*

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Summary. Surface lesions of the corpus callosum, apparently hitherto unrecorded in the literature, are described. They were found in 17 cases among 168 consecutively examined brains. There was no correlation with clinical symptoms, or with general autopsy findings. Their morphology and relationship to neighboring structures suggest that they represent a form of physical trauma to the corpus callosum, but the exact mechanism of their production is not clear.

Zusammenfassung. Oberflächliche Balkenläsionen werden beschrieben, die bisher offensichtlich nicht im Schrifttum aufscheinen. Sie wurden in 17 von 168 laufend untersuchten Gehirnen angetroffen. Beziehungen zu klinischen Symptomen oder den Körperobduktionsbefunden lagen nicht vor. Die Morphologie und Beziehungen dieser Läsionen zu den Nachbarstrukturen lassen sie als eine Folge mechanischer Balkenschädigung interpretieren, doch ist ihr genauer Entstehungsmechanismus bisher ungeklärt.

Key-Words: Corpus callosum—Brain injury—Traumatic lesion—Marchiafava-Bignami-disease—Demyelination—Pressure of arteries.

Introduction

Seventeen cases in which a lesion was found on the external surface of the corpus callosum are described in this report. The pathogenesis of these lesions is obscure. No description of such lesions was found in the literature. This report is presented in the hope that further elucidation of their nature may be forthcoming.

Case Studies

All brains used for this study were fixed in formalin, suspended from the basilar artery. The brains were cut in coronal sections after complete fixation. Tissues were embedded in paraffin and stained with hematoxylin-eosin, Bodian's method, the periodic acid-Schiff reaction and phloxin fast green stains for myelin.

Incidence

During a period of eight months, 420 autopsies were performed at the Institute of Pathology. The brains of 168 of these cases were studied in the Division of Neuropathology. Callosal lesions were found in 17 cases, which represents an incidence of 10%. An additional 3 cases were collected in the same period from other institutions.

Gross Findings

Surface lesions of corpus callosum were usually not noticeable on external examination of the brain but were readily apparent on section. They appeared as slightly depressed, grayish discolored, soft areas immediately underneath the

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Table 1

Case Number	Age Sex	Clinical History	Autopsy Findings		Location of Lesion
			General	CNS	
1	69 F	Carcinoma of uterus, a seizure-like episode, treated with Dilantin	Peritonitis with perforation of left sigmoid colon, papillary adenocarcinoma of uterus, hypertrophy of myocardium	No lesions in CNS	Midline anterior
2	100 F	Macular degeneration with blindness, confusion	Bronchopneumonia, aortic stenosis, hypertrophy of myocardium, amyloidosis of myocardium, atherosclerosis	Organizing infarct, left occipital lobe	Midline anterior
3	83 F	Congestive heart failure, peripheral vascular disease, loss of memory and disorientation, BUN 140	Nephrosclerosis, atherosclerosis, hypertrophy of myocardium, gangrene of feet	Senile changes, atherosclerosis	Lateral anterior
4	75 M	Paraparesis, spinal cord decompression, repeated infections	Acute pulmonary edema, bacteremia, multiple decubiti	Subacute necrotizing, myelopathy	Lateral anterior
5	78 M	Weakness, anemia, abdominal pain, at laparatomy multiple abdominal metases	Adenocarcinoma of stomach, metastatic to pancreas and peritoneum, pulmonary emboli, myocardial infarct	Recent small infarcts of left frontal lobe, probably embolic	Midline posterior
6	93 M	Padget's disease, emphysema, grand mal seizures, treated with Phenobarbital	Bronchopneumonia, pulmonary infarcts, nephrosclerosis, amyloidosis of myocardium	Senile changes, anoxic changes in hippocampus and cerebellum, arteriosclerosis	Midline anterior
7	67 F	Anorexia, weakness, fatigue, lethargy, hypertension, BP 208/98, muscle tenderness, treated with steroids	Polymyositis	Cavitated infarct, right basal ganglia	Midline posterior
8	68 F	Retroperitoneal sarcoma, treated with cytotoxins	Lymphosarcoma involving heart, lungs, pancreas, ovary, skin, bone marrow	Slight atrophy	Lateral anterior

9	83 F	Hypertension, BP 220/120, aphasia, dysphagia, left hemiparesis	Bronchopneumonia, myocardial infarct, myocardial hypertrophy	Multiple old and recent infarcts, right and left cerebellum, senile changes	Midline posterior
10	76 M	Congestive heart failure, chronic hypertension, chronic lung disease	Bronchopneumonia, pulmonary emboli, myocardial hypertrophy	Old infarcts right cerebellum, atherosclesosis	Midline anterior
11	76 M	Gastrointestinal bleeding, post-op fever, old cerebrovascular accident	Acute peritonitis, bronchopneumonia, myocardial hypertrophy	Bilateral neuronal loss in globus pallidus and hippocampus, consistent with posticteric encephalopathy	Midline posterior
12	80 M	Hypertension old cerebrovascular accident, hemiplegia, senility	Multiple myocardial infarcts, bronchopneumonia	Old infarcts in caudate n. and pons, senile changes	Midline and lateral anterior
13	74 F	Multiple myeloma	Multiple myeloma, pulmonary infarct	Epidural myeloma, recent infarct left frontal, old infarcts left basal ganglia	Midline entire corpus callosum
14	72 M	Pemphigus, vulgaris, congestive heart failure, treated with steroids	Nocardiosis of pericardium, lungs, myocardial hypertrophy, pyelonephritis	No lesions	Midline anterior
15	75 M	Pulmonary emphysema, heart failure, malnutrition	Myocardial infarcts, myocardial hypertrophy, pulmonary emboli	Diffuse cortical atrophy, arteriosclerosis, small infarcts right basal ganglia and left frontal lobe	Midline posterior
16	80 M	Hip fracture, "chronic brain syndrome"	Asphyxiation by food particles, myocardial hypertrophy, nephrosclerosis	Atrophy, old infarcts in basal ganglia, occipital lobe, cerebellum	Midline anterior
17	69 M	Head trauma 2 months prior to death, evacuation of subdural hematoma	Myocardial hypertrophy, nephrosclerosis, pulmonary emboli, aspiration pneumonia	Contusions of right temporal lobe and cerebellum; post. fossa subdural hematoma	Midline entire corpus callosum

external surface of the corpus callosum. The average depth of the lesion was 2 mm; some lesions were rounded, with a diameter of 2—3 mm; the majority were elongated, extending sagitally along the corpus callosum. Some cases had multiple lesions.

Lesions were distributed at random along the corpus callosum, from genu to splenium. Fourteen of the lesions were located exactly in the midline; four, somewhat laterally, the center being up to 3 mm off the midline. In one case, two lesions appeared side by side. All of the lateral lesions were found in the anterior half of the corpus callosum. Of the midline lesions, seven were in the anterior half and five were in the posterior half. In two of the cases the lesions extended along the entire length of the corpus callosum.

Microscopic Findings

In H. E. preparations the lesion appeared as a sharply delineated, pale zone under the pial surface. There was considerable rarefaction of the tissue, but no disruption of tissue continuity, either at the pial surface or deeper; cellular elements were greatly diminished (Figs. 1, 3). Tissue reaction was minimal; reactive astrocytes were found only in few instances; and, widely scattered macrophages with foamy cytoplasm were seen between the remaining fibers. Blood vessels running through the lesion appeared intact. Myelin stain demonstrated complete loss of myelin from the lesions. Bodian stains showed either complete loss of axons or few scattered axons crossing the lesions. A few axonal swellings were found at the edge of the lesions (Fig. 2).

One case showed a thin layer of persisting myelinated fibers at the surface of the corpus callosum covering the lesion. In four cases the cross-section of the lesion was roughly V shaped, with intact myelin sheaths in a small central, subpial island of fibers (Fig. 3).

Five of the lesions showed a clear correlation with the stem or branches of the anterior cerebral artery which was either overlying the lesion or was partly impressed into the lesion (Fig. 1). All of the lesions with overlying arteries were observed lateral, in the anterior half of the corpus callosum. No correlation with blood vessels was observed in the posterior portion of the corpus callosum.

Clinical Data

There were 10 males and 7 females ranging from 67 to 100 years; the majority were in the seventies. Five of the patients had a definite history of hypertension; a sixth was questionable. A clinical diagnosis of arteriosclerosis was made in three of the cases; three had malignant neoplasms. A definite history of trauma was obtained in only one of the cases. Confusion and loss of memory were the most common symptoms referable to the central nervous system, occurring in seven of the patients. Four of the patients had localizing signs consistent with vascular disease of the brain. Seizures occurred in two. Only two of the patients had a lumbar puncture shortly before death. Three patients were on steroid therapy; two received analeptics and two, cytotoxic drugs.

Autopsy Findings

Myocardial hypertrophy was the most common autopsy finding; it occurred in ten cases. Five of the patients died with myocardial infarcts; six, with pulmonary embolization and infarction. Nephrosclerosis was found in four instances. Myocardial amyloidosis in two of the patients was thought to be an incidental finding.

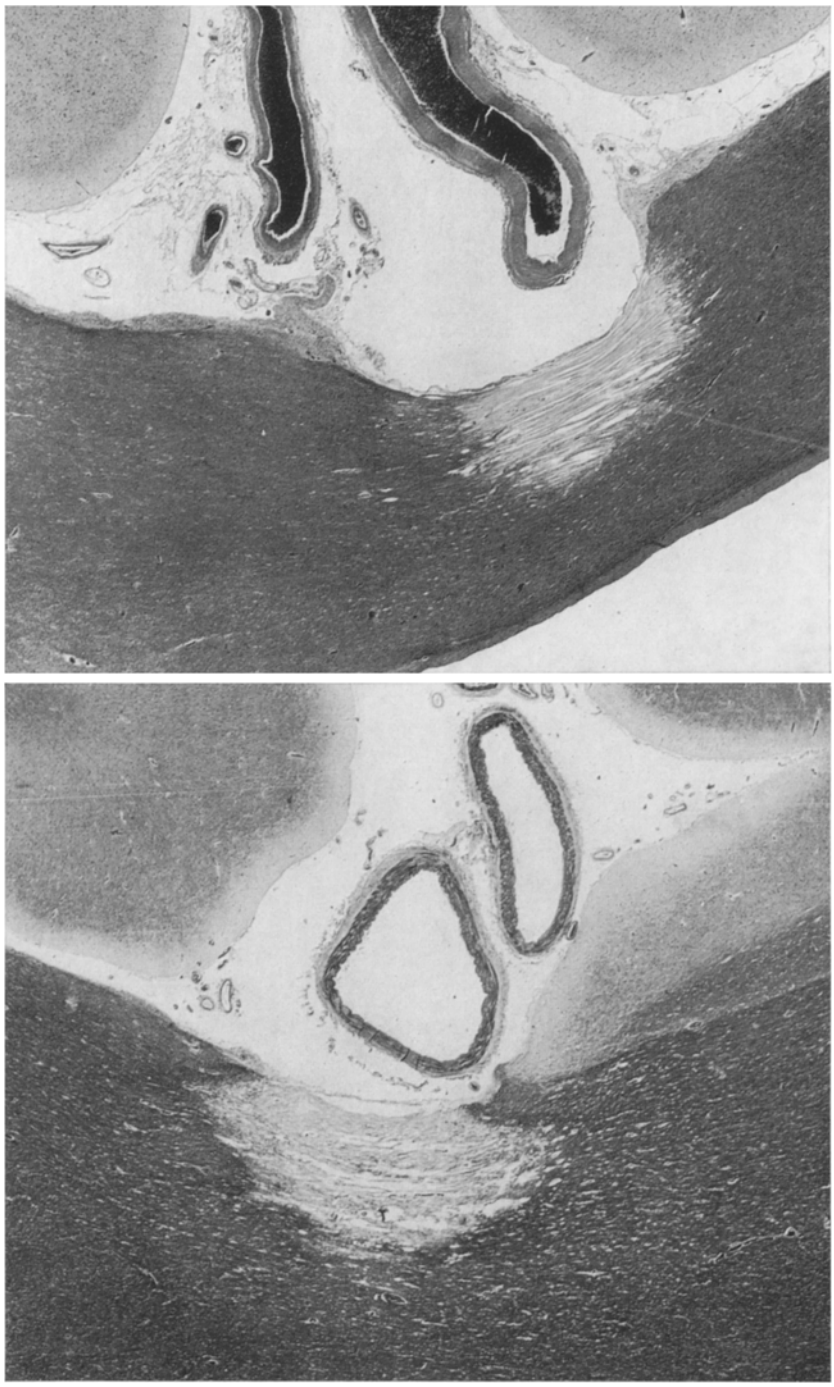

Fig. 1. Two surface lesions of corpus callosum showing relation to overlying arteries. There is considerable rarefication of tissue, but continuity is preserved. Bodian's stain; 11 ×

Infarction was the most common finding in the brain, occurring in nine cases. Some of these infarcts were recent; others, old, without consistent regional distribution, and involved cerebral hemispheres or cerebellum.

As expected from the clinical histories, senile changes and atrophy were also common, occurring in seven cases. History of trauma in one case was confirmed by the finding of subdural hematoma and contusions. Two of the brains showed no gross or microscopic changes whatsoever.

Discussion

Artefacts were ruled out as a cause of surface lesion of the corpus callosum. The presence of scattered macrophages and axonal swellings and the complete loss of myelin and oligodendroglia cells were inconsistent with a post-mortem artefact. Attempts to produce an equivalent lesion in other brains—by either

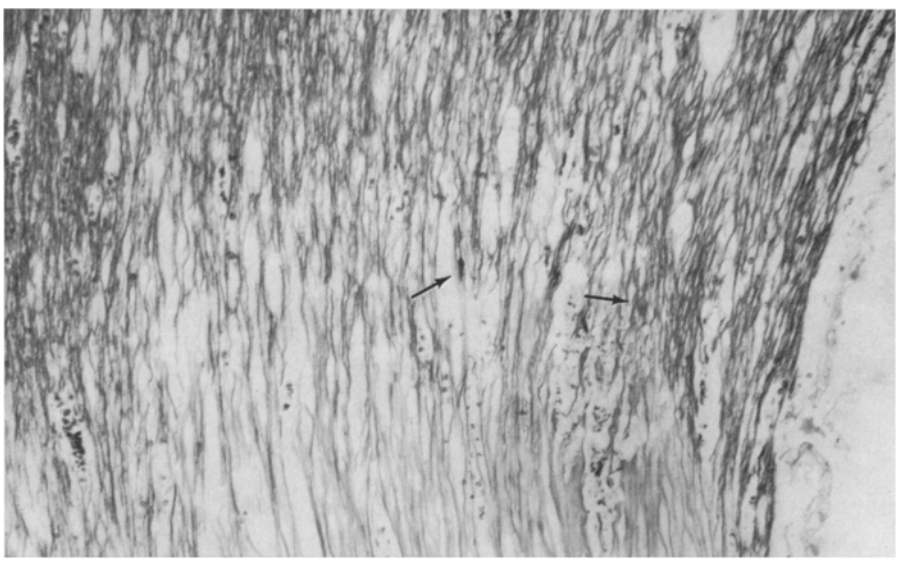

Fig. 2. Edge of lesion showing partial preservation of axons and occasional axon swellings (arrows). Bodian's stain; 157×

pressing the brain against the falx or pulling the cerebral hemispheres apart—were not successful. Artifical tears of the tissue were easily distinguished from the surface lesions, showing disruption of the tissue without loss of myelin and reactive changes.

We found it impossible to date surface lesion of corpus callosum because of the sparse reactive changes in macrophages and astrocytes which could indicate either relatively recent or extremely old lesion, or a type of lesion with unusually slight tissue response.

There were no symptoms in the clinical histories of our cases that could be related to the presence of surface lesions of the corpus callosum. Advanced age appeared to be the only consistent finding. None of the autopsy findings were consistently correlated with the presence of the lesions. There is no indication for a vascular, toxic, or metabolic etiology of the lesions. The loss of myelin sheaths and oligodendroglia along with preservation of tissue continuity and the

sharp delineation of the lesions are reminiscent of demyelination; however, the regional distribution of the lesions and the loss of axons in them argues strongly against demyelinating disease. The lesions in our cases likewise differed from those of Marchiafava-Bignami disease; in the latter the dorsal fibers are usually spared. Marchiafava-Bignami disease also shows involvement of other commissural systems and has a history of alcoholism which was absent in our cases.

Mechanical factors appeared to be the most probable cause of surface lesions. Local compression of the corpus callosum or general cranio-cerebral trauma

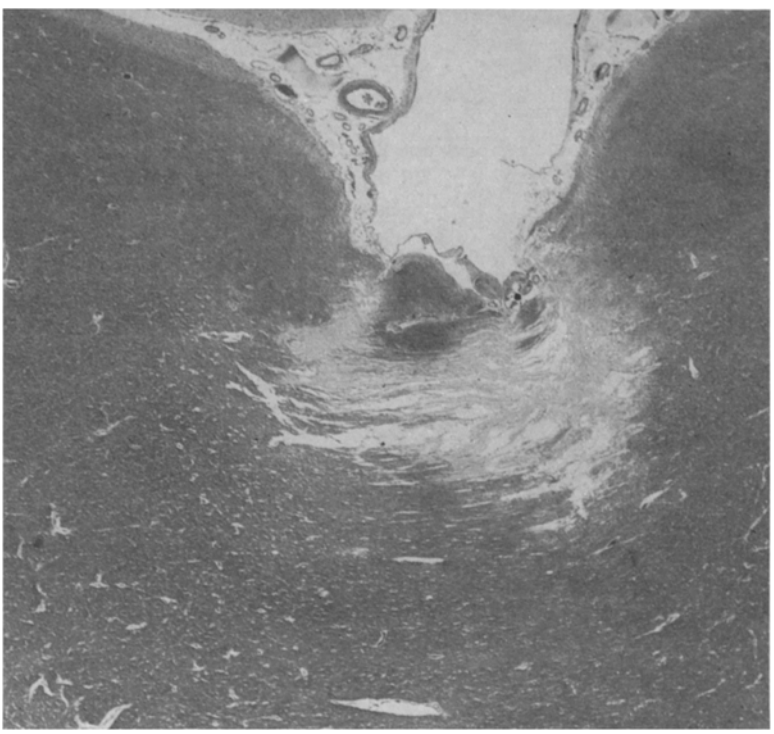

Fig. 3. Midline lesions in the posterior portion of the corpus callosum; there is no relation to arteries. An island of intact fibers is seen in the middle portion of the lesion. Hematoxylin-eosin; 11×

might be considered. The features of traumatic lesions of corpus callosum reviewed in 51 cases by LINDENBERG et al. (1955) usually presented themselves as ischemic necrosis or as a massive hemorrhagic disruption. Of particular interest is the discussion given by these authors of the various theories proposed for the pathogenesis of traumatic lesions of corpus callosum. They discount the theory of ROWBOTHAM (1949) that such lesions result from striking against the falx, because the falx except for its most posterior portion is too far removed from the corpus callosum. JUNET (1938) postulated that a sudden increase in intraventricular pressure pushes the corpus callosum against the falx. This theory was also discounted because a pressure pulse, rather than displacing the corpus callosum toward the vertex, results in diffuse extension of the ventricles. LINDENBERG *et al.*

agreed with HÄMÄLÄINEN (1929) who suggested that tearing of the corpus callosum might result from a sudden bilateral displacement of the cerebral hemispheres.

We cannot rule out the possibility that surface lesions of corpus callosum result from tears produced by slight, unrecorded craniocerebral trauma. However, this interpretation is not entirely satisfactory because some of our lesions were clearly related to overlying arteries. In two of the cases the lesions extended to the inferior surface of the rostrum, where traumatic lesions are never found (LINDENBERG *et al.*, 1955). No relation was found between arteries and lesions in the posterior portion of corpus callosum. In this portion the lesions were always strictly in the midline, suggesting a relationship to the falx; the arterial branches were usually located laterally under the cingular gyrus.

It is therefore suggested that the surface lesions of the corpus callosum are produced by either pressure of the corpus callosum against the cerebral arteries or the falx, or by pressure of the arteries or falx against the corpus callosum. Our data offer no clues as to the direction of the pressure and as to its causes. A compression effect seems consistent with the occasional observation of an island of preserved fibers in the center of the lesion. Shearing forces and resulting tissue distortion are minimal in the center of a uniformly compressed tissue and are maximal at its periphery. Such shearing forces were shown to be responsible for the damage produced by tissue compression (HOLBOURN, 1944)

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