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Classification of acquired lesions of the corpus callosum with MRI

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Abstract MRI has facilitated diagnostic assessment of the corpus callosum. Diagnostic classification of solitary or multiple lesions of the corpus callosum has not attracted much attention, although signal abnormalities are not uncommon. Our aim was to identify characteristic imaging features of lesions frequently encountered in practice. We reviewed the case histories of 59 patients with lesions shown on MRI. The nature of the lesions was based on clinical features and/or long term follow-up (ischaemic 20, Virchow-Robin spaces 3, diffuse axonal injury 7, multiple sclerosis 11, hydrocephalus 5, acute disseminated encephalomyelitis 5, Marchiafava-Bignami disease 4, lymphoma 2, glioblastoma hamartoma each 1). The location in the sagittal plane, the relationship to the borders of the corpus callosum and midline and the size were documented. The 20 ischaemic lesions were asymmetrical but adjacent to the midline; the latter was involved in new or large lesions. Diffuse axonal injury commonly resulted in large lesions, which tended to be asymmetrical; the midline and borders of the corpus callosum were always involved. Lesions in MS were small, at the lower border of the corpus callosum next to the septum pellucidum, and crossed the midline asymmetrically. Acute disseminated encephalomyelitis and the other perivenous inflammatory diseases caused relatively large, asymmetrical lesions. Hydrocephalus resulted in lesions of the upper part of the corpus callosum, and mostly in its posterior two thirds; they were found in the midline. Lesions in Marchiafava-Bignami disease were large, often symmetrically in the midline in the splenium and did not reach the edge of the corpus callosum.

Key words Corpus callosum · Ischaemia · Demyelination · Magnetic resonance imaging

Introduction

The prevalence of acquired lesions of the corpus callosum (CC) was estimated as about 3 % in an MRI study of 450 patients [1]. Because often no obvious neurological deficit is associated with solitary CC abnormalities, they have attracted little attention in the radiological literature.

Differential diagnosis of CC abnormalities may be difficult. Ischaemic, inflammatory, traumatic and neoplastic disorders may involve the CC. A reliable diagnosis can often be derived from the history and other clinical findings. Whereas in small lesions only subtle clinical examination techniques may detect minor changes [2], complete defects of the CC may result in unilateral agraphia, apraxia and tactile anomia. Correlation of symptoms and a CC lesion can pose a problem: it has been speculated that in hydrocephalus, for example, gait disturbance, memory deficits and dementia may be due not to intracranial pressure but to a CC de-

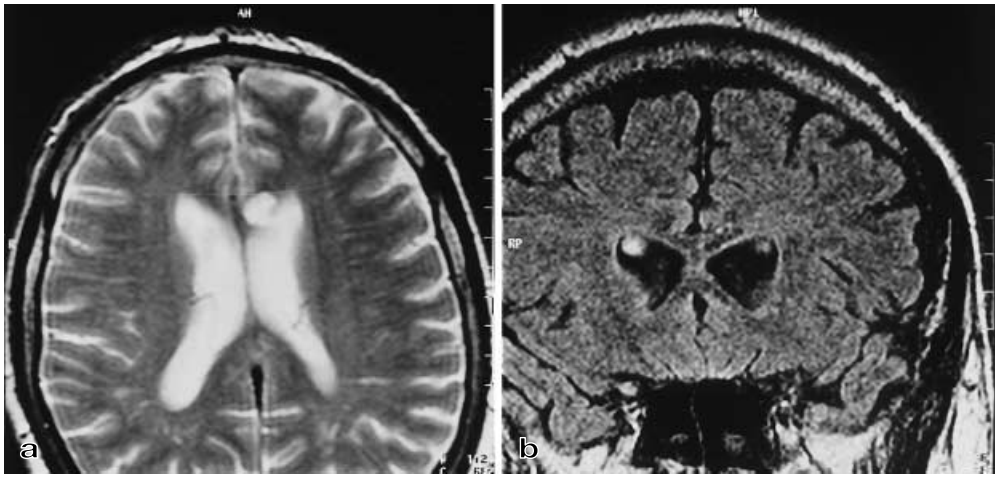

Fig. 1 a, b An ischaemic lesion in the genu in a 74-year-old patient with left carotid occlusion. Arounded ischaemic lesion in the genu on the left is isointense with CSF on T2-weighted spin-echo **a** and FLAIR **b** images. It spares the margins of the corpus callosum, and the midline

fect [3]. Our aim was to define characteristic features of CC lesions of known origin.

Patients and Methods

MRI of 296 patients with lesions of the CC was reviewed. Imaging was performed at 1.0 or 1.5 Tesla. Images were acquired in at least axial and sagittal or coronal planes. Contrast medium was given for 21 examinations. We analysed lesions visible in two imaging planes and with two differently weighted sequences: T1- and/or T2-weighted (including FLAIR) or diffusion-weighted images. The following were excluded: lesions originating from the hemispheres and invading the CC; lipomas of the CC; developmental abnormalities (hypo- or aplasia; thinning in chronic hydrocephalus; surgical lesions; and lesions of unknown aetiology, where clinical data and follow-up did not enable a definite diagnosis. We selected only representative examples of lesions in multiple sclerosis; most patients with MS were imaged for another study which used only thin axial slices without gaps, with various sequences.

In 59 patients (age 3–77 years; 24 female, 35 male) a diagnosis was established by long-term follow-up and clinical data: 20 had ischaemic lesions, three wide Virchow-Robin spaces (VRS), seven diffuse axonal injury (DAI), 11 MS, five hydrocephalus, five acute disseminated encaphalomyelitis (ADEM) (one postvaccination, four Marchiafava-Bignami disease, two lymphoma, one a glioblastoma and one a hamartoma as part of von Recklinghausen's disease).

Lesions of the corpus callosum were analysed according to the following criteria: 1. their relation to the upper or lower border of the CC or central location in coronal images; 2. their relation to the midline; 3. their position within the CC, divided into five sections: genu, anterior middle and posterior thirds of the body and splenium; 4. their size. As the lateral borders of the CC are not well defined, a vertical line was drawn on both sides from the most lateral point of the pericallosal cistern, to define the CC.

Results

Ischaemic lesions (20, mean age 49 years, 15 male, five female)

In five patients acute ischaemia was seen in strongly diffusion-weighted images (after intracranial surgery in two, from thromboembolic disease in two and vertebral artery occlusion in one). There were 15 patients with older lesions: extracranial artery occlusion (3), thromboembolic in arterosclerotic disease (5), after intracranial surgery (5), from vasculitis (1) and CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarctions and leucencephalopathy) (1). Only one lesion reached the upper or lower border of the CC in the coronal plane except one, and the lesions tended not to cross the midline (Fig. 1). Seven of the lesions reached or minimally crossed the midline (Fig. 2). These were larger (mean diameter 0.9 cm) than the 13 which spared the midline (diameter 0.5 cm; t-test, $p < 0.03$; Table 1). In a subacute lesion (Fig. 2) transient oedema may make the lesion larger. Of the seven lesions involving the midline five were in the middle or posterior thirds and splenium, while eight of the 13 lesions which did not reach the midline were in the anterior third and genu.

Table 1 Mean size of callosal lesions

Aetiology	Patients	Mean size (mm)
Ischaemia; midline not involved	13	5[a]
Ischaemia; midline involved	7	9[a]
Diffuse axonal injury	7	10
Multiple sclerosis	11	3
Acute disseminated encephalomyelitis	5	14

[a] $p < 0.03$

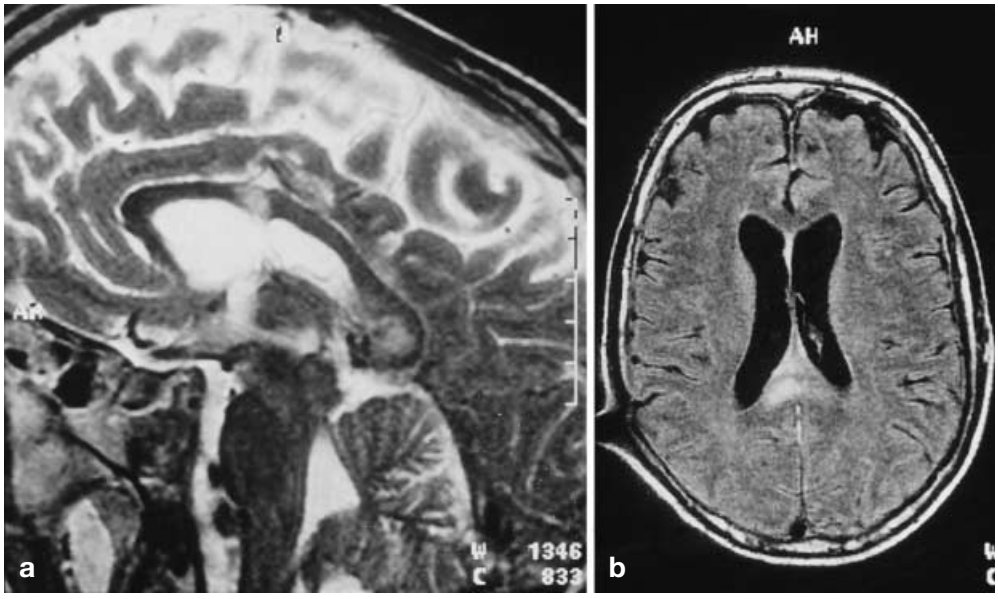

Fig. 2 a, b Subacute ischaemic lesion of the splenium in a 40-year-old patient with subarachnoid hemorrhage. **a** The lesion is near the mid-line and **b** is mainly on the right. A second lesion at the junction of the anterior and middle thirds was caused by temporary external CSF drainage

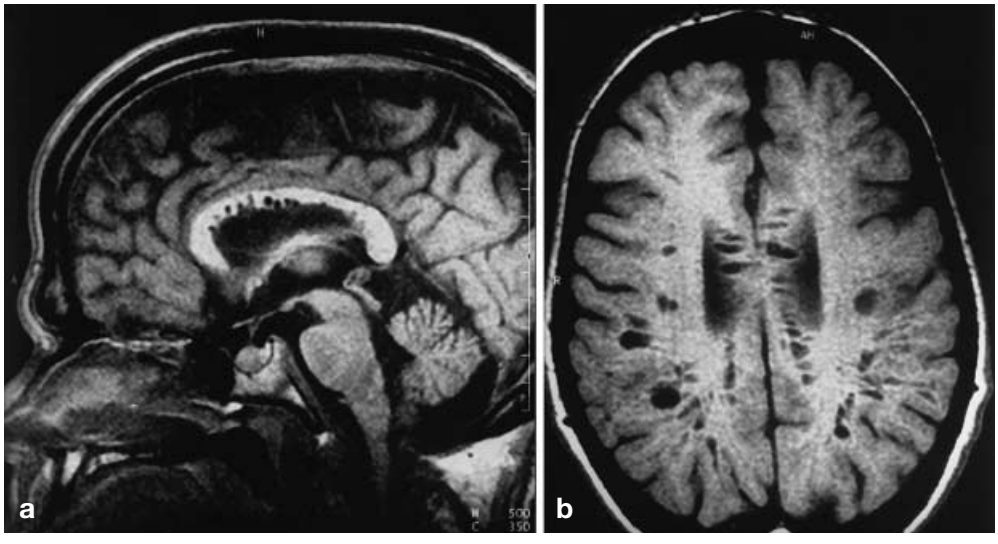

Fig. 3 a, b Wide Virchow-Robin spaces in a 25-year-old patient with a mucopolysaccharidosis. Wide VRS can be seen **a** in the corpus callosum in the sagittal plane and **b** in the white matter

Large perivascular spaces (VRS)

These could be delineated centrally in the CC at its lower or upper border in three men 25, 60 and 77 years old. They were rather small (mean diameter 0.23 cm) and oval. They occasionally reached or passed the midline and lay in all parts of the CC. The younger patient had mucopolysacharidosis (Fig. 3); the two elderly patients had generally atrophic brains with multiple VRS in the basal ganglia and cerebral white matter as well the CC.

Diffuse axonal injury (DAI)

Trauma resulted in the largest lesions (mean diameter 0.96 cm). Seven patients (mean age 27 years, three male, four female) had sustained severe head trauma. MRI was performed 1–2 weeks after the trauma in three patients, and four were studied 1–10 years after the accident. Traumatic lesions were asymmetrical, crossed the midline and often involved the complete coronal extent of the CC, including the upper and lower aspects, depending on their size (Fig. 4). In one case the whole length of the CC was affected; three lesions were in the genu and three in the splenium.

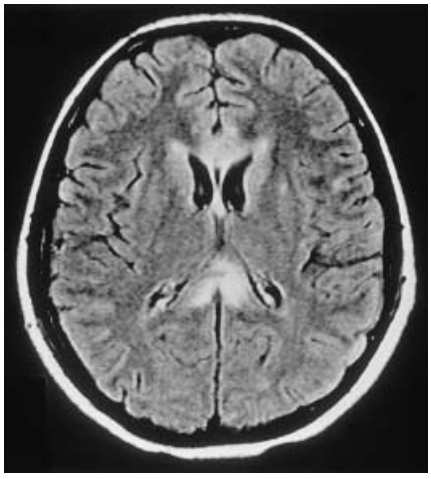

Fig. 4 Traumatic lesion along the corpus callosum in a 48-year-old patient had an accident 2 years before the MRI. Diffuse axonal injury caused a large, asymmetrical areals gliosis involving the entire corpus callosum

Multiple sclerosis

Most patients with MS (9/11) were undergoing long-term follow-up after a primary diagnosis 2–30 years previously (mean age 40 years, three male, eight female). Two, 30 and 34 years old, seeing the neurologist for the first time, both already had multiple white-matter lesions. Of the 11 patients nine demonstrated a typical pattern of asymmetrical CC lesions in the midline above the septum pellucidum (Fig. 5). They were in the middle and posterior thirds of the CC. In two patients larger, round foci of demyelination were found laterally in the midportion. The mean diameter of all CC lesions was 0.3 cm (Table 1).

Acute disseminated encephalomyelitis (ADEM)

Five patients (mean age 29 years, two male, three female) had CC involvement in inflammatory disease. The first examination was 2 days to 2 weeks after the onset of symptoms. Long term follow-up followed 1–5 years later. One patient also had extensive cervical spinal cord involvement. All patients had other white-matter lesions as well. The CC lesions lay asymmetrically near the midline (Fig. 6). They reached the upper and lower margins, depending on their size, and four of the five were in the splenium.

Marchiafava-Bignami disease (MBD)

In the four patients (mean age 38.5 years, one male, three female) given their diagnosis, a history of chronic alcohol and drug abuse was known. Three had acute an illness (one was examined for Wernicke's encephalopathy) and one was studied for staging of bronchial carcinoma. They had lesions in the splenium, symmetrically in the midline. The nonacute lesion was cystic.

Hydrocephalus (five patients, mean age 43.8 years, four male, one female)

This resulted in lesions at the upper margin of the CC in the midline under the falx. Of the five, four were in the middle and posterior thirds, with a notch-like impression and signal change suggesting adjacent gliosis (Fig. 7). One patient had irregular high signal in the anterior and middle thirds on T2-weighted images shortly after ventriculoperitoneal shunting. The cause of the hydrocephalus was occlusion of the foramen of

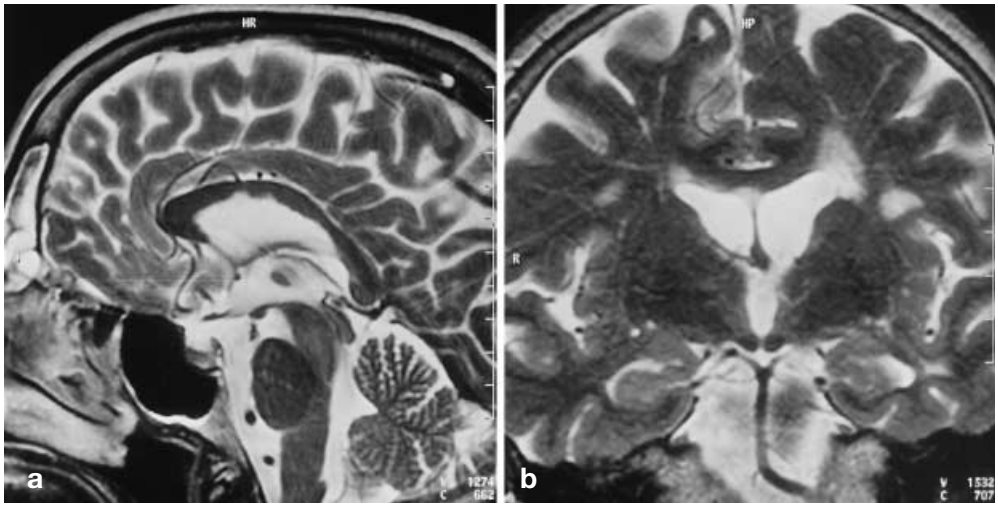

Fig. 5 a, b Lesions at the callosal-septal interface in a 59-year-old woman with MS for many years. Small, asymmetrical lesions at the callosal-septal interface are seen **a** in the T2-weighted midsagittal and **b** coronal sections, crossing the midline. Other white matter and periventricular lesions are seen

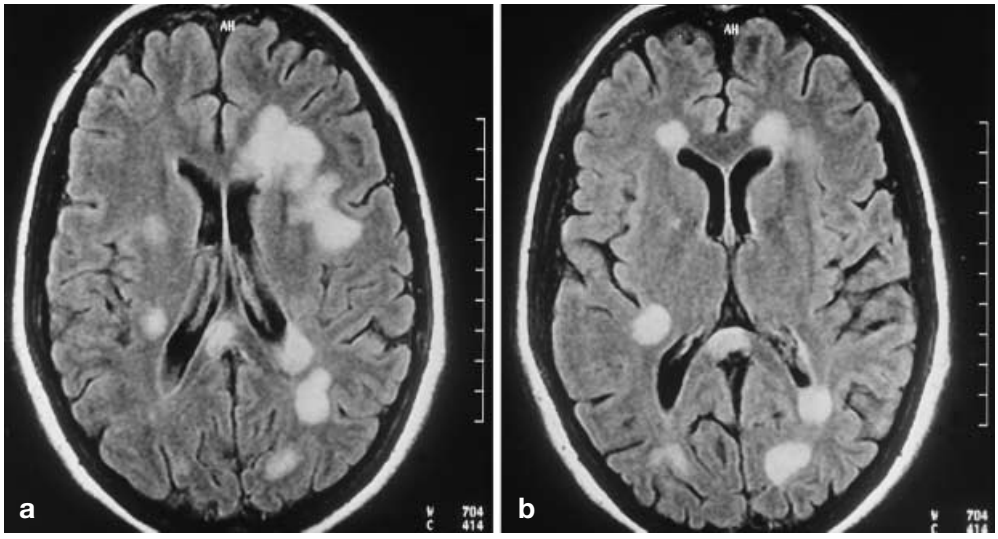

Fig. 6a, b Multiple corpus callosum and white matter lesions in a 30-year-old patient with acute ataxia and hemiparesis as a manifestation of acute disseminated encephalomyelitis. The lesion in the splenium is large, extending across the corpus callosum and crosses the midline asymmetrically

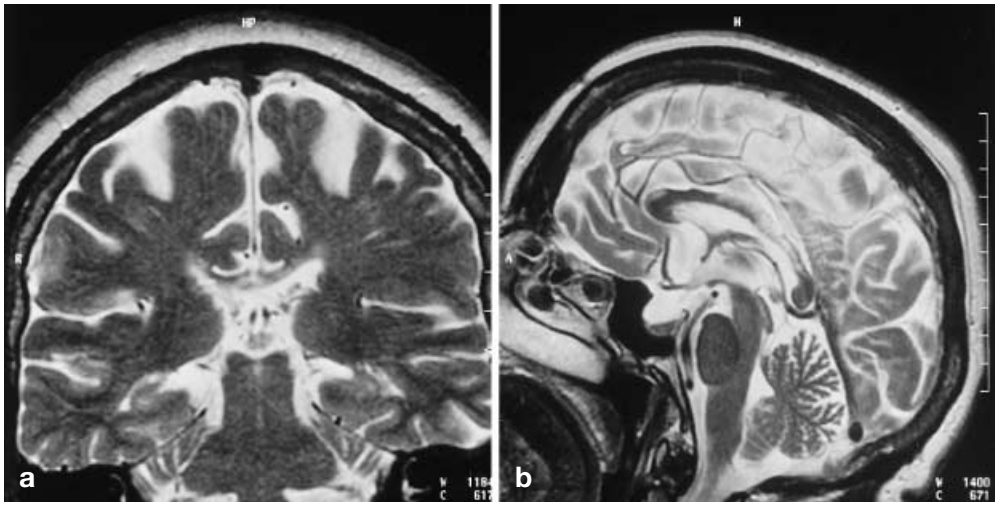

Fig. 7a, b A lesion due to hydrocephalus in the middle and posterior thirds of the corpus callosum in a 58-year-old woman with a supranuclear palsy after revision of ventriculoperitoneal shunt. There is extensive thinning and signal change, most extensive in the upper part of the corpus callosum, under the falx cerebri

Monro (in one patient), aqueduct stenosis (in two), occlusion of the aqueduct by a tectal astrocytoma (in one) and suba (1).

Tumours

Lymphoma and glioblastoma showed enhancement. The glioblastoma (Fig. 8) enlarged the CC, with slight enhancement; a second lesion was seen in the left temporal region, and biopsy revealed glioblastoma. One patient recurrent primary cerebral non-Hodgkin's lymphoma, a small, contrast-enhancing, asymmetrical lesion at the lower margin of the CC. The other had multifocal cerebral lymphoma with an asymmetrical lesion centrally on the coronal images. The hamartoma was a round lesion in the splenium in a patient being followed for optic glioma. It lay away from the midline and the borders of the CC.

Discussion

MRI has facilitated assessment of the CC [4, 5]; imaging in the sagittal [6] and coronal [7] planes in very useful.

The CC is said to be relatively resistant to ischaemia [8]. Of 352 patients with cerebral infarcts, only 28 (8%) showed CC lesions [9]. This can be explained by the architecture of the vessels: the main arteries supplying the CC run in pairs around the genu and anterior two thirds (pericallosal artery) and the splenium and a variable part of the dorsal third (posterior pericallosal). They lie 2–5 mm from the midline and give multiple angiographically invisible branches, maximum length 8 mm

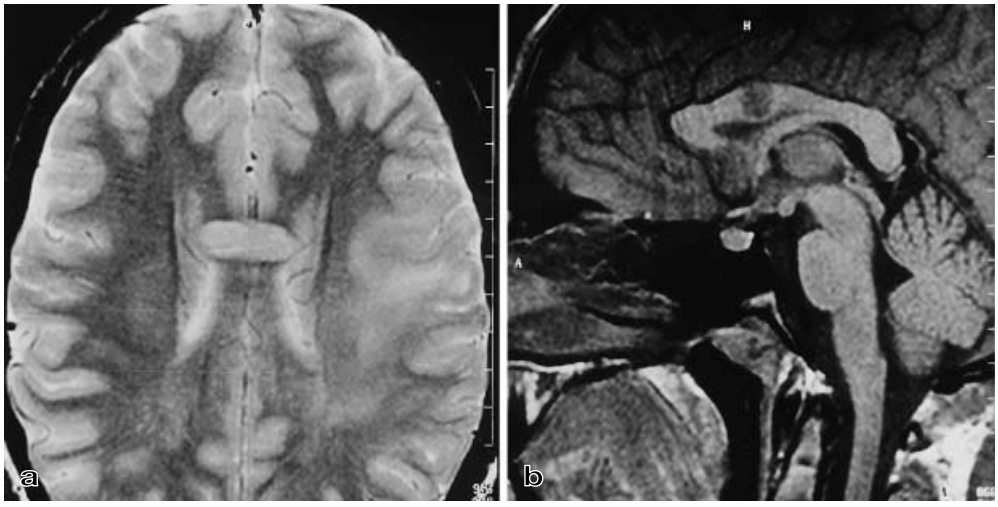

Fig. 8 a, b A 28-year-old patient with rounded lesion in the anterior third which expands the corpus callosum; a glioblastoma was found at surgery

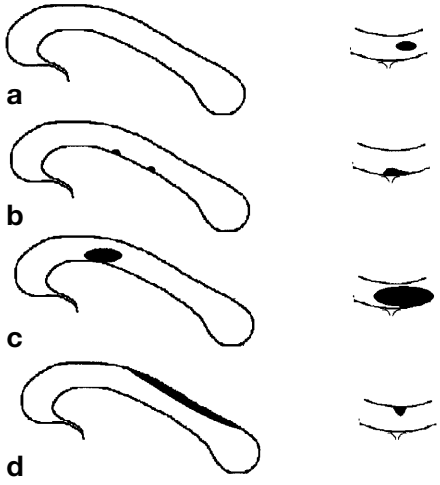

Fig. 9 a–d Typical distribution of **a** ischaemic, **b** inflammatory, **c** traumatic and **d** hydrocephalus-related lesions

and width 100 μ, perforating the CC vertically. These vertical branches give off terminal and collateral branches laterally and medially which run between the nerve fibres. They anastomose with neighbouring contralateral vessels [8] and, thus, supply the midline from either side.

Ischaemic lesions are usually central in the CC. The upper and lower margins are not involved. Because of the bilateral arterial supply, CC infarcts are usually confined to one side (Fig. 1) but large acute ones may cross the midline (Fig. 2). The midline tissue is supplied by the unaffected side. Our findings match published examples [9] as two thirds respected the midline.

A third small artery origing from the anterior communicating artery, seen in > 75 % of postmortem angiograms [10, 11], is not paired. Occlusion of this artery may result in midline ischaemic lesions. The longest branches of the pericallosal vessels travel in the depths of the callosal sulcus and supply the cingulate gyrus and the lateral quarter of the CC. Infarcts in this region were not included in this study.

The preservation of the upper and lower borders of the corpus callosum may also be explained by the angioarchitecture. The upper margin is supplied many lot of short and intermediate length arterioles. Branches of the longer perforating arteries turn laterally and backwards to the surface of the CC, at an angle of more than 90° [8]. The longer perforating vessels end in the centre of the CC. The lower aspect of the CC is subependymal and therefore also supplied by ependymal vessels.

Widen VRS [12] are not common in the CC. A review of 816 MRI studies revealed 314 cases with wide VRS, but only in the basal ganglia and upper cerebral hemispheres [13]; no CC VRS were reported. In our cases VRS differed from vascular lesions by their involvement of any part of the CC, including the upper and lower borders (Fig. 3). Delineation of perivascular fluid around the smallest branches of the CC vessels to the level of capillaries is possible [13]. Because of their anastomoses, wide VRS crossing the midline can be seen. In the CC the vascular network follows the commisural fibres [8]; this explains the configuration of the wide spaces in our patients, which ran along the fibres of the CC. For the differentiation between gliotic changes and perivascular spaces FLAIR-sequences are helpful.

Shearing forces occurring in rotational movement cause DAI, which are usually difficult to show on CT. Pathophysiologically there is supposedly an indirect influence of the falx cerebri: positive or negative acceleration and/or rotational forces distort the cerebral hemispheres from the relatively fixed stiff CC. The falx cerebri is relatively immobile [7].

About 10% of patients with head trauma have CC lesions [7, 14], whose number depends on the severity of the trauma [15]. Initial and follow-up CT will not show CC lesions unless they are haemorrhagic. Detection of CC lesions plays a part in prognostication about neuropsychological deficits and may be used in medicolegal expert opinions [16]. Most traumatic CC lesions are reported to be in the splenium [7, 14] but, depending on the direction of the forces applied, the middle and anterior thirds or genu [17] may be involved as well. This pattern can be seen if the forces are lateral or oblique frontal (Fig. 4). In severe trauma the entire CC may be damaged.

The assumed common mechanism of MS and ADEM is perivenous inflammation. In MS up to 93% of patients have small lesions at the lower margin of the CC, the socalled callosal-septal interface [18]. These are thought to be caused by perivenous inflammatory changes [19] around the subependymal veins. We found lesions in the midportion in nine of 11 cases (Fig. 5). The typical periventricular [19] elliptical lesions [20] are more lateral and were not included in this study. The periventricular and elliptical lesions are not pathognomonic [21]. A regression analysis which attempted tried to eludicate the MRI factors which predict conversion to definite MS [22] did not take CC/septal into account and we cannot therefore decide how specific these lesions are. Two patients with ADEM had similar changes, suggesting similar pathophysiology [18]. One lesion a patient with MS was lateral to the midline in the mid portion of the CC. Lesions at the callosal-septal interface are not an early sign of MS. Most patients with these lesions had been diagnosed years previously and had various other lesions. Nevertheless these lesion may assist in the differentiation from vascular encephalopathy [18].

ADEM is a monophasic inflammatory reaction to the deposition of antibodies in small vessels, usually an immunological reaction to a virus infection some weeks earlier. Patients often present with a severe multifocal neurological syndrome. MRI is abnormal in nearly all cases, showing large periventricular and white matter lesions in the brain stem, cerebellum, cerebral hemispheres, spinal cord and optic nerve. In some cases the changes are rather symmetrical [27]. Our patients had multiple hemisphere and CC white-matter lesions (Fig. 6). In four cases there were rather large, asymmetrical foci, associated with severe clinical deficits.

MBD is an acute inflammatory demyelination of the CC frequently reported in patients with alcohol abuse, especially red-wine drinkers. It has been linked to a disorder of vitamin B2 metabolism. In the acute stage there is focal, partially haemorrhagic [28] demyelination of the CC, often ending up as a cystic defect, although remissions have been reported [29]. Typically, a single lesion centrally and symmetrically involves the splenium. Lesions can also be multiple, along the CC [2].

In chronic hydrocephalus different types of lesion are described in the upper part of the CC, generated by the hydrocephalus itself or its treatment. Dilated ventricles elevate the CC and lead to indentation of the middle and posterior thirds by the falx cerebri. This "mechanical ischaemia" with reduction of the capillaries in the long term [23] leads to thinning of the posterior part of the CC in the midline [3]. Animal experiments support this hypothesis [24]. In our patient (Fig. 7) a notch-like indentation of the upper part was shown. There are also lesions following over-drainage of hydrocephalus [25] causing reversible oedema in the anterior and middle thirds, probably due to stretching of small perforating branches of the pericallosal artery [25], resulting in a so-called scalloped deformity [26].

Tumour dissemination along white matter tracts, especially across the CC, is typical of high-grade glial neoplasms. Involvement of the CC is an unfavourable prognostic factor [30]. In our patient with multifocal glioblastoma we saw a circumscribed CC lesion (Fig. 8). Periventricular infiltration is often seen (83%) in multifocal lymphoma; the CC is involved in 60% of multifocal centroblastic tumours [31]. Our patient with a lesion at the lower margin probably had subependymal invasion by lymphoma cells [32]; in the other patient, central CC infiltration was seen.

Thus, the patterns of CC damage are not specific for a certain disease, but may be regarded as characteristic of groups of lesions (Fig. 9). A lesion of the lower border may, for example, represent MS, ADEM or lymphoma, which all cause perivenous change. Characterisation of CC lesions may focus differential diagnosis: ischaemic lesions tend to be central and spare the midline; traumatic lesions to involve the complete extent of the CC in coronal sections, as they are large; MS lesions cross the midline asymmetrically at the callosal-septal interface; hydrocephalic lesions are at the upper border, in the midline; and in MBD lesions are symmetrical, in the splenium.

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