High Resolution MRI in the evaluation of cerebral focal cortical dysplasias

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Purpose

Focal cortical dysplasias (FCD) are localized areas of malformed cerebral cortex and are frequently associated with epilepsy in both children and adults. A broad spectrum of histological abnormalities of cortical laminar structure has been included in the diagnosis of FCD.

Over the years, there has been some degree of heterogeneity in the clinical, surgical, and histo-pathologic literature, and terms like "focal cortical dysplasia", "mild cortical dysplasia", "Taylor-type focal cortical dysplasia", "balloon cell dysplasia", "non-balloon cell dysplasia", and "microdysgenesis" have all been applied to describe architectural and cellular abnormalities of the cortical mantle[1][2][3][4]. For example, some authors use the descriptor Taylor-type FCD only when balloon cells are present, whereas others, including Taylor et al. in their original report, include some Patients whose lesions had dysmorphic neurons but lacked balloon cells[5].

A definitive understanding of the relevance of each cell type or architectural abnormality among the various possible combinations relies on further knowledge achievements about the mechanisms of cortico-genesis and their correlation with clinical and imaging findings.

In this study we used the histological Palmini classification[6] which divided the FCD in two group:

- Type I: without dysmorphic neurons or balloon cells
  1. IA: isolated architectural abnormalities (dyslamination, accompanied or not by other abnormalities of mild MCD)
  2. IB: architectural abnormalities, plus giant or immature, but not dysmorphic neurons

- Type II: Taylor-type FCD (dysmorphic neurons without or with balloon cells)
  1. IIA: architectural abnormalities with dysmorphic neurons but without balloon cells
  2. IIB: architectural abnormalities with dysmorphic neurons and balloon cells

The purpose of this study is to identify, basing on Palmini 2004 classification[6], peculiar high resolution MRI patterns of focal cortical dysplasia subtypes in order to distinguish Type II dysplasia from Type I. In this way it should be possible to make an earlier diagnosis, that would enhance surgical management in a Patients with drug-refractory focal epilepsy.
Methods and Materials

Materials:

We retrospectively reviewed and analyzed neuropathologic and neuroradiological material of 10 Patients (6 boys, 4 girls; age range 2.4 months - 16 years) affected by symptomatic epilepsy with definitive diagnosis of FCD, that were clinically referred to Day Hospital of Department of Infantile Neuropsychiatry and, from January 2002 and June 2011, underwent MR examination at Department of Bio-images and Radiologic Sciences of Policlinic "Agostino Gemelli" in Rome, Italy.

We enrolled all Patients that underwent surgery with final diagnosis of dysplastic disease and excluded those with histopatologic diagnosis of FCD, but with findings of more complicated and wide cortical malformations such as Posterior Quadrantic Dysplasia and other congenital cortical defects (hemimegalencephaly, heterotopias) that are frequently associated to FCD. Otherwise, those cases with morphological and/or signal intensity alteration of hippocampus were also enrolled due to the frequent association between dysplastic lesions and hippocampal disease (dual pathology).

Post-surgical clinical and radiological follow-up was provided for at least one year.

All MR images were read by an expert neuroradiologist blinded to the final histological diagnosis and all pathological specimens were analyzed by a dedicated pathologist. A consensual evaluation was finally obtained.

MRI Protocol

All MR studies were performed using a Signa Advantage 1.5T scanner, applying in all cases the MRI protocol routinely used in epileptic Patients. The FLAIR sequences were obtained only in patients older than 3 years (Table 1).

In Patients with suspicion of temporal lobe epilepsy, axial images were acquired parallel to the major hippocampal axis and coronal images acquired perpendicular to this reference axis. For extratemporal lobe epilepsies sections were acquired parallel and perpendicular to the bi-commissural line.

MRI Features
In each exam the following MRI reports of FCD have been carefully detected:

1. Focal cortical thickening classified in slight, moderate or severe (cortical thickening > 7 mm)
2. "Blurring" of junction cortical grey matter-subcortical white matter on 3D IR-prepped High Resolution T1-weighted sequences
3. "Trans mantle" signs
4. Sulcal or gyral abnormal pattern (including the "cortical dimple cleft complex")
5. Lobar hypoplasia
6. Signs of "core atrophy" of white matter
7. Hyperintensity of cortical grey matter on FSE T2 weighted images and FLAIR images
8. Hyperintensity of subcortical white matter on FSE T2 weighted images and FLAIR images (assessed in slight, moderate and severe)
9. Hypointensity of subcortical white matter on 3D IR-prepped High Resolution T1 weighted sequences
10. Hippocampal abnormalities that were evaluated in terms of atrophy and/or T2 weighted images signal intensity alteration of hippocampus even without evidence of volume reduction
11. Presence of other lesions (tumor, vascular malformations, lesions acquired in early life) adjacent to FCD

"Trans mantle" sign is defined as white matter signal alteration tapered from the crown of a gyrus or bottom of a sulcus toward the ventricle and reflects the involvement of radial glial-neuronal units[7][8][9].

All MRI findings were analyzed and compared with the histological diagnosis.

Pathological Findings

All the histological samples were evaluated by the same expert pathologist and the following elements were systemically evaluated in all specimens:

- Immature neurons
- Dymorphic neurons
- Balloon cells
- Giant neurons
- Isolated architectural abnormalities (dyslamination)

In cortical dysplastic lesions these elements may occur in variable combination and lead to specific histopathologic features classified in our study basing on 2004 Palmini classification[6].

Recently it has been proposed a new classification by International League Against Epilepsy (ILAE), which identifies new histological criteria for FCD[10]. (Table 2)

According to the 2011 ILAE classification[10] the difference between type IA and type IB FCD is exclusively in the geometric distribution of dislamination (radial in type IA vs tangential in IB), while the immature and hypertrophic neurons can be present in both entities. Conversely in the 2004 Palmini classification the type IB is characterized by immature and hypertrophic neurons, while type IA is not.

The type IIA and IIB of 2004 Palmini classification (old Taylor type) have the same histological characteristics also in the 2011 ILAE classification.

The type III is characterized by combination of FCD with other principal lesions (hippocampal sclerosis in type IIIA, glial or glioneural tumor in IIB, vascular malformation in IIIC, acquired lesions in IID).

When our study was submitted, the ILAE classification had not been released yet, so the histological diagnoses were based on Palmini classification. In this poster we will refer to the latter, making, when possible, a correlation with ILAE classification.

Images for this section:
<table>
<thead>
<tr>
<th>Protocol</th>
<th>TR</th>
<th>TE</th>
<th>IR</th>
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**Table 1:** MRI Protocol routinely used in our study.
The three-tiered ILAE classification system of focal cortical dysplasia (FCD) distinguishes isolated forms (FCD types I and II) from those associated with another principal lesion (FCD type III)

<table>
<thead>
<tr>
<th>FCD type I (isolated)</th>
<th>Focal cortical dysplasia with abnormal radial cortical lamination (FCD type IA)</th>
<th>Focal cortical dysplasia with abnormal tangential cortical lamination (FCD type IB)</th>
<th>Focal cortical dysplasia with abnormal radial and tangential cortical lamination (FCD type IC)</th>
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<tr>
<td>FCD type II (isolated)</td>
<td>Focal cortical dysplasia with dysmorphic neurons (FCD type IIA)</td>
<td>Focal cortical dysplasia with dysmorphic neurons and balloon cells (FCD type IIB)</td>
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<tr>
<td>FCD type III (associated with principal lesion)</td>
<td>Cortical lamination abnormalities in the temporal lobe associated with hippocampal sclerosis (FCD type IIIA)</td>
<td>Cortical lamination abnormalities adjacent to a glial or glioneuronal tumor (FCD type IIIB)</td>
<td>Cortical lamination abnormalities adjacent to vascular malformation (FCD type IIIC)</td>
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</table>

FCD Type III (not otherwise specified, NOS): if clinically/radiologically suspected principal lesion is not available for microscopic inspection. Please note that the rare association between FCD types IIA and IIB with hippocampal sclerosis, tumors, or vascular malformations should not be classified as FCD type III variant.

**Table 2:** ILAE 2011 Histological classification [10].
Results

Pathological Results

Basing on 2004 Palmini classification [6] the final histological diagnosis were:

- 5 Patients with Type IIB (old Taylor type) FCD with dysmorphic neurons and balloon cells
- 5 Patients with Type I FCD.

In the latter group (Type I FCD) we had:

- 3 cases of type IB dysplasia characterized by abnormal cortical lamination, giant neurons and ectopic neurons localized in the subpial layer and in the subcortical white matter
- 1 case of type IA dysplasia with abnormal cortical lamination with ectopic neurons and without cellular abnormalities.
- 1 case of type IA and IB dysplasia mixed together (it was obtained as histopathologic result of type IA dysplasia at first surgical resection, while type IB was the result of the second resection for persistence of seizures)

Age at epilepsy onset and ictal zones

All Type IIB FCD Patients had an extra-temporal ictal onset zone: left inferior frontal convolution, post-central gyrus, right superior parietal lobule, right superior frontal convolution, and rolandic area.

Instead the Type I FCD group had an extra-temporal location in 2 cases (mesial occipital, superior, medial and inferior frontal convolutions) but a temporal location in 3 cases (2/3 in uncus-mesial temporal lobe).

Moreover the age onset of seizures ranged from 2,4 months to 16 years in Type IIB FCD group (mean age: 110 months), while it ranged from 4 months to 12 years (mean age: 65 months) in Type I FCD group.

Postoperative Outcome

Post-surgical outcome in Type IIB FCD showed absence of seizures in 2 Patients, significant decrease in 2 Patients and persistence with pluri-monthly frequency in 1 case.
Instead in Type I FCD group postoperative outcome was worse, with reduction of seizures' frequency in only 2 cases, while in 2 cases it wasn't registered modification with pluri-daily repetition of crisis, daily even increased in 1 case.

It wasn't observed significative difference between these two groups about frequencies of seizures before surgery

**MRI Findings**

Type IIB FCD forms showed (Figure 1):

- cortical thickness range from 6 mm to 10 mm (mean value: 8 mm) (Figure 2);
- marked blurring between cortical grey matter and subcortical white matter;
- cortical grey matter moderate hyperintensity on FSE T2w images and FLAIR images in 4/5 cases, slight in 1/5 case;
- subcortical white matter moderate hypointensity on 3D IR-prepped High Resolution T1w images in 4/5 cases, prominent in 1/5 case;
- cortical grey matter abnormal hyperintensity on 3D IR-prepped High Resolution T1w images graded as moderate in 4/5 cases, slight in 1/5 case;
- signal abnormality of subcortical white matter reaching the lateral ventricle wall (transcortical sign) with typical funnel-shaped morphology in 4/5 Patients;
- a simplified sulcal pattern with smoothing of cerebral sulci in 3/5 Patients;
- core-atrophy of subcortical and deep white matter in 1/5 case (demonstrated by slight homolateral attraction of lateral ventricle to the lesion);

Type IIB FCD appeared stable for shape, dimension and signal characteristics in all pre-surgery MRI examinations.

Type I FCD group showed (Figure 3):

- cortical thickness range from 5 mm to 9 mm (mean value: 6 mm) (Figure 2);
- blurring between cortical grey matter and subcortical white matter ranged from moderate to marked;
- cortical grey matter hyperintensity on FSE T2w images and FLAIR images was present and anyway slight only in 1/5 case, while absent in 4/5 cases;
- cortical grey matter hyperintensity on 3D IR-prepped High Resolution T1w images was absent in all cases;
- subcortical white matter hypointensity on 3D IR-prepped High Resolution T1w images was always absent;
- transcortical sign was always absent
• simplified sulcal and gyral pattern in 2/5 cases, microgyric pattern in 1/5 case;

Also in Type I FCD group all lesions appeared stable for shape, dimension and signal characteristics in all pre-surgery MRI examinations.

Signal hyperintensity on FSE T2w and FLAIR images of subcortical white matter ranged from moderate to prominent in both type IIB and type I FCD, so this parameter didn't result useful for differentiating the two subgroups.

Overall there were 2 cases where the dysplastic lesion was combined to venous drainage anomaly, more evident on T1 weighted images with Gd-DTPA injection. The histologic diagnosis of these 2 FCD was: 1 case of type IIB and 1 case of type I. These cases should correspond to type IIIC of 2011 ILAE classification.

Furthermore in 1 case of type I FCD group the lesion, localized in the left inferior temporal convolution, joined with subcortical white matter atrophy of hippocampus/right mesial temporal lobe. This association is reported in the 2011 ILAE classification as corresponding to type IIIA FCD.

Basing on our results we believe that 3D IR-prepped High Resolution T1w sequence is the most useful to differentiate type IIB FCD from type I, because, in addition to better anatomic definition of blurring and cortical thickness, this MR sequence allows to point out two peculiar characteristics of type IIB FCD (absent in type I):

• Prominent signal hypointensity of subcortical white matter, that in some cases reaches periventricular zone (transmantle sign)
• Hyperintensity of dysplastic cortex up to no distinction underlying white matter as "segmentary cortical loss".

These elements may be explained by histopathologic alterations that mark the two different groups. In fact in type IIB FCD (Figure 1): the prominent signal intensity alterations of grey matter (totally absent in type I FCD) and the prominent hypointensity of subcortical white matter histologically corresponded to prominent cortical disorganization, presence of numerous neuronal morphological abnormalities (balloon cells and dysmorphic neurons) associated with remarkable dysmyelination, loss of myelinic fibers, and proliferation of glial cells.

Instead in Type I FCD cases (Figure 4) the histological findings were characterized by a slighter cortical dislamination than type IIB FCD, presence of dysmyelination with loss of myelinic fibers and glial proliferation. There weren't cytological abnormalities, while neurons with normal appearance were located in ectopic subpial layer and in subcortical white matter that resulted in signal intensity alteration mainly of white matter but not of
grey matter. Therefore MRI findings, in terms of signal intensity alterations, reflect in vivo the histological abnormalities at the bottom of dysplastic lesions.

The enhanced images usually didn't add accuracy in differential diagnosis; they resulted useful only at the first MRI examination to exclude a no-dysplastic lesions and also to point out venous drainage anomaly, associated in 2 cases.

In regard to clinical postsurgical outcome, we registered differences between these two groups. In fact type IIB FCD Patients showed disappearance or significant reduction of frequencies of seizures; instead in type I FCD Patients the post surgical outcome was worse, with seizures still frequent and only in one case with an increase of them. These results overlap other studies' results. The best outcome of type IIB FCD seems linked to their more detectability than type I FCD, as demonstrated by MRI examination (the signal intensity abnormality was more evident) and by EEG (a more distinct focality of electrophysiologic alteration). Moreover post-surgical outcome depends on a lot of factors such as epilepsy onset age, age at surgery, but above all the extension and site of surgical resection and concomitant presence of hippocampal sclerosis.

**Images for this section:**
Fig. 1: Type II cortical dysplasia with balloon cells (IIB). Coronal FSE T1 weighted image (A) shows the prominent signal hypointensity of subcortical white matter with "transmantle" sign; a corresponding prominent signal hyperintensity of white matter is evident on Axial FLAIR image (B). The dysplastic cortex is thickened with blurring of the grey-white matter junction and it appears hyperintense on FSE T1 weighted image as for "focal cortical loss". Axial FLAIR image after surgery shows complete resection of left frontal ictal onset zone (C); Histological findings confirmed diagnosis of Type IIB focal cortical dysplasia with balloon cells (D, E): Hematoxylin and eosin staining is used. Micrograph (D) shows a prominent neuronal disorganization and abundant heterotopic white matter neurons. The cortical dysplastic architecture is characterized by failure of layered organization with abnormal clustering of neurons, abnormal polarity and moderately dysmorphic neurons of variable size (small and large), especially in deeper layer. Micrograph at high magnification (E) shows a balloon cell characterized by abundant pale eosinophilic cytoplasm and two eccentric nuclei. MRI and histologic findings are in agreement for diagnosis of Type IIB cortical dysplasia.
**Fig. 2:** Cortical thickness. Sagittal 3D IR-prepped High Resolution T1 weighted images in a Patient with Type IIB FCD (A); and in a Patient with Type I FCD (B). The cortical thickness is greater in A (mean 10 mm) than in B (mean 6 mm). Besides signal hypointensity of subcortical white matter is evident in A, absent in B.
Fig. 3: Signal hyperintensity of grey matter and/or white matter. Coronal FSE T2 weighted image (A) and FLAIR image (A’) in a Patient with Type IIB FCD show the typical "trans-mantle" sign. The prominent hyperintensity of subcortical white matter is evident in the left frontal lobe on both T2w and FLAIR images (note the cortical thickening). Axial FSE T2 weighted images (B) and FLAIR images (B’) in a Patient with Type I FCD demonstrate signal hyperintensity of subcortical white matter in the left temporal pole without signal intensity alteration of grey matter and with substantially normal cortical thickness. Note in both cases the enlargement of local subarachnoid CSF spaces.
Fig. 4: Type I cortical dysplasia. Coronal and Axial FSE T2 weighted and FLAIR images (A, B, G, H) show in the left inferior temporal convolution a lesion characterized by signal hyperintensity of subcortical white matter. It is evident the cortical thickness, the moderate blurring of the grey-white matter junction and signs of parenchymal atrophy, such as documented by slight enlargement of the occipital horn of the left lateral ventricle. Signal intensity of subcortical white matter is normal on Sagittal 3D IR-prepped High Resolution T1 weighted images (C). These findings support diagnosis of Type I cortical dysplasia. Note that 3D IR-prepped High Resolution T1 weighted image affords a better anatomic definition than FSE T1 weighted image (D). The lesion joins with a venous drainage anomaly appearing as linear enhancement on Axial and Coronal FSE T1 weighted image following Gd-DTPA injection (E, F). The Patient underwent surgical resection of temporal pole with hippocampal saving (I) but her post surgical outcome was worse, with seizures still frequent and so a new surgical resection of total ictal onset zone was performed. Histological findings confirmed diagnosis of Type I cortical dysplasia. Hematoxylin and eosin staining is used (L, M). Micrograph (L) shows the laminar disorganization with high cell density and clusters of misplaced neurons (an increased number of pyramidal neurons in layers I or II and abundant ectopic neurons in the subcortical white matter). Micrograph at high magnification (M) demonstrates laminar disorganization occurring together with cyto-architectural abnormalities and, especially in the white matter, there is a population of neurons of different size: some with a large nucleus and a thin rim of...
cytoplasm (immature neurons) and/or giant neurons. MRI and histologic findings are in agreement for diagnosis of Type I cortical dysplasia.
Conclusion

In our experience it was generally possible to distinguish type IIB FCD from type I FCD (basing on Palmini classification) by MRI findings. The principal MRI criteria to differentiate Type IIB FCD from Type I were [Table 3):

- signal hyperintensity of cortical grey matter on 3D-IR-prepped High Resolution T1w images (moderate in 80% of type IIB vs absent in 80% of type I)
- prominent hypointensity of subcortical white matter on 3D IR-prepped High Resolution T1w images (moderate in 80% vs absent in 100%)
- cortical thickness (mean values 8mm vs 6mm)
- "transmantle" signs (present in 80% vs absent in 100%)
- localization of lesions (extra-temporal in 100% vs temporal in 60%)

Conversely in our opinion signal hyperintensity of subcortical white matter on T2w/FLAIR images and blurring of cortical grey matter-subcortical white matter junction can’t be considered useful in differentiating between the two groups, due to their presence in both type IIB and Type I FCD.

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<th>Histological Diagnosis (Palmieri 2004)</th>
<th>Ictal Onset Zone</th>
<th>Age at Epilepsy Onset</th>
<th>Postoperative Seizures Onset</th>
<th>Cortical Thickness</th>
<th>Grey Matter Hyperintensity on T2w/FLAIR</th>
<th>Grey Matter Hyperintensity on 3D-IR-T1w</th>
<th>White Matter Hypointensity on 3D-IR-T1w</th>
<th>Transmantle Sign</th>
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<td>Type II B</td>
<td>Extratemporal 5/5 (100%)</td>
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<td>2/5 Absence (40%)</td>
<td>6-10 mm (mean 8 mm)</td>
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<td>1/5 Prominent (20%)</td>
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<td>4 m-2 years (mean 65 months)</td>
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Table 3: MRI findings related to histotype.
References


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