Diffuse T1-MRI White Matter Volume Decrease in Patients with Sickle Cell Disease

Soyoung Choi, Adam Bush, Matt Borzage, Anand Joshi, Vidya Rajagopalan, Julie Coloigner, Natasha Lepore, Thomas D. Coates, John Wood

To cite this version:
Soyoung Choi, Adam Bush, Matt Borzage, Anand Joshi, Vidya Rajagopalan, et al.. Diffuse T1-MRI White Matter Volume Decrease in Patients with Sickle Cell Disease. 21sy Annual meeting of the Organization for Human Brain Mapping, Apr 2015, Honolulu, HI, United States. . hal-01792279

HAL Id: hal-01792279
https://hal.science/hal-01792279
Submitted on 15 May 2018

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L’archive ouverte pluridisciplinaire HAL, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d’enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.
Diffuse T1-MRI White Matter Volume Decrease in Patients with Sickle Cell Disease

Presentation · June 2015

DOI: 10.13140/RG.2.2.27062.09280

CITATIONS
0

READS
7

9 authors, including:

Soyoung Choi
University of Southern California

Adam Bush
Stanford University

Anand A Joshi
University of Southern California

Vidya Rajagopalan
Children's Hospital Los Angeles

All content following this page was uploaded by Natasha Lepore on 18 June 2017.

The user has requested enhancement of the downloaded file.
Diffuse T1-MRI White Matter Volume Decrease in Patients with Sickle Cell Disease

Soyoyo Choi1, SA, Adam Bush1, Matt Borzage2, Anand Joshi3, Vidya Rajagopalan4, Julie Cologner4, Natasha Lepore4, Thomas D Coates5, and John C Wood1

1Department of Pediatrics and Radiology, Children’s Hospital Los Angeles USC, Los Angeles, CA
2Biomedical Engineering, University of Southern California, Los Angeles, CA
3Signal and Image Processing Institute, University of Southern California, Los Angeles, CA
4Department of Radiology, Children’s Hospital Los Angeles, Los Angeles, CA
5Hematology/Oncology, Children’s Hospital Los Angeles, Los Angeles, CA

We Treat Kids Better

Introduction
Sickle cell disease (SCD) is a genetic blood disorder associated with anemia, chronic vascular damage, recurrent stroke, silent cerebral infarction and early mortality.1,2 Patients with SCD have increased cerebral blood flow to compensate for their anemia but nevertheless exhibit regional cerebral hypo-perfusion and neuro-cognitive decline.1,4,5 Previous volumetric studies in SCD have shown delayed growth, gray matter (GM) loss, while white matter (WM) loss, and decreased cortical thickness compared with control subjects.6,7 Diffusion-tensor imaging has demonstrated compromised WM integrity in major fiber pathways diffusely throughout the brain.8,9 Further regional investigations of structural outcome could potentially help expand our understanding of the neurobiology of SCD.

Subjects
T1-weighted MRI was obtained on 23 subjects on a 3T Philips Achieva as part of a study on sickle cell disease and neurological outcome. All patients were recruited with informed consent or assent; the study was approved by the Institutional Review Board at Children’s Hospital Los Angeles (IRB 2010-00083). 14 clinically asymptomatic SCD patients (age=21.6±4.8y, F=8, M=6) were compared against 9 ethnically matched control subjects (age=23.0±3.7y, F=6, M=3). Exclusion criteria included pregnancy, previous overt stroke, acute chest or pain crisis hospitalization within one month.

Methods
T1-weighted images were processed using BrainSuite (brainsuite.org) in a semi-automated fashion with limited manual correction of the cortical boundaries—minimizing extraneous inclusion of meninges or exclusion of cortex—to extract, classify tissue types, and render 3D-surfaces of the inner and pial cortices. Surface-Volume Registration (SVReg) was used to automatically segment the brain into two separate segmentation schemes. The first segmentation divided the cerebrum into 4 lobes and the second segmentation divided the brain into 90 regions of interest (ROI). (Figure 1) Average cortical thickness, GM volume, and WM volume were calculated against controls (p<0.05); Blue indicates significantly higher values of SCD patients compared to controls; Red indicates significantly lower values of SCD patients against controls (p<0.05).

Results
6 SCD patients had one or more silent infarcts identified on T2 images. Volumetric comparisons showed no differences between SCD patients with silent infarcts and those without.

Brain lobe comparisons: SCD patients showed WM volume loss in comparison to control subjects bilaterally in the frontal, parietal, and temporal lobes, and cerebellum while sparing the occipital lobe. Total volume loss was seen in the right frontal and occipital lobes, and bilaterally in the parietal lobes. SCD patients had greater cortical thickness in the right temporal lobe in comparison to controls but GM volume was not significantly different. (Table 1)

Subparcellated region comparisons: SCD patients exhibited diffuse gyral WM loss in 28 out of 68 cortical regions, located primarily in the frontal, parietal, and temporal lobes. Left insula was the only cortical region where WM volume was greater in SCD patients against control subjects. Of the 20 non-cortical and extra-cerebral regions, WM volume was significantly lower in the left hippocampus, bilateral caudate, and right putamen in SCD patients. GM volume in these regions showed no differences. (Figure 2)

Conclusions
In SCD, silent cerebral infarcts primarily involve the frontal lobe, followed by the parietal lobe, subcortical nuclei, and temporal lobe, with very few lesions reported in the occipital lobe or cerebellum.1,4,5 We observed a nearly identical pattern for cortical WM volume loss, even though WM volumes were no different in patients with or without silent cerebral infarction. Although we did not identify GM volume loss, our patient population was significantly older than cohorts for whom GM volume loss has been previously described. Further investigation of brain morphological abnormalities in SCD patients will provide insight into disease mechanisms and progression.

References

Figure 1. Displays segmentation schemes on atlas brain (top row) and subject registration results of a representative SCD subject (bottom row). (a) Pial surface segmentation of the right hemisphere into 4 lobes. (b) Single trans-axial slice of the brain segmented into 4 lobes, corpus callosum, and insula with exclusion of the subcortical, ventricles, and extracerebral structures. (c) Pial surface segmentation of the right hemisphere into 90 ROIs. (d) Single trans-axial slice of the brain segmented into 90 ROIs. Gyrall WM boundary is limited by two opposing sulcal edges of the gyrus from the coronal view.

Table 1. % difference of significant results (p<0.05) of volume and cortical thickness in SCD patients compared to controls. Lobe regions include the cerebrum and excludes the brain stem and cerebellum; R: right; L: left. Red indicates significantly lower values of SCD patients compared to controls; Blue indicates significantly higher.

<table>
<thead>
<tr>
<th>Region</th>
<th>Whole Brain</th>
<th>Frontal</th>
<th>Parietal</th>
<th>Temporal</th>
<th>Occipital</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difference</td>
<td>-6.7%</td>
<td>-10.8%</td>
<td>-11.3%</td>
<td>-11.0%</td>
<td>-10.2%</td>
</tr>
<tr>
<td>Total volume</td>
<td>-9.3%</td>
<td>-4.6%</td>
<td>+4.6%</td>
<td>+5.0%</td>
<td>-</td>
</tr>
</tbody>
</table>

Gyrall WM boundary is limited by two opposing sulcal edges of the gyrus from the coronal view.

Figure 2: Significant changes in WM volume, GM volume, and cortical thickness displayed on lateral and midsagittal views of both hemispheres of the atlas template. Red indicates significantly lower values of SCD patients against controls (p<0.05); Blue indicates significantly higher values of SCD patients against controls (p<0.05).