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Disclosure Information:
Gianluigi Ardissino, MD
No, neither I nor my spouse/partner have anything to disclose.
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No, neither I nor my spouse/partner have anything to disclose.
R Benti, MD
No, neither I nor my spouse/partner have anything to disclose.
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Title: Detection of brain involvement in hemolytic uremic syndrome (HUS) by F18-FDG

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Body: HUS is the most common cause of ARF in children between 1 and 4 yrs of age. Central nervous system (CNS) is involved in 20-50% of cases and the case-fatality rate is estimated 3-7%, with a majority of deaths being related to CNS damage. Morphological brain imaging (CT and MRI) detects structural changes in only 20-60% of cases with overt neurological damage. The aim of the study is to assess the usefulness of F-18- Fluoro-deoxi-glucose (F18-FDG) brain PET (Positron Emission Tomography) imaging to investigate brain involvement in HUS. Six pts with HUS (3 males, mean age 6 yrs) were studied at disease onset and after clinical recovery (1-12 mos later). Two pts had neurological symptoms but none deceased for complications. PET/CT brain imaging was obtained 40-60 min after FDG i.v. administration. Visual and VOI-based analysis of PET data were performed comparing early and late PET studies.
Acute PET/CT study showed a mild symmetric and diffuse impairment of cortical metabolism in cerebellum and posterior cortex (parieto-occipital); one pt showed also relevant asymmetric

hypometabolism in left temporal cortex. Subcortical involvement was mild and limited to the caudate nuclei. Recovery PET/CT studies showed normalization of perfusion patterns in parieto-occipital cortex and subcortical gray matter, whereas the cerebellum hypometabolism hold over. Our results seem consistent with both models of brain injury suggested in HUS, featuring both mild/symmetric and reversible involvement of gray matter in most pts (metabolic injury) and further focal/asymmetric cortical involvement in pts with relevant neurological symptoms in acute phase (thrombotic focal vascular damage).
F18-FDG PET/CT seems an interesting tool to investigate CNS involvement in acute HUS. Diffuse impairment of perfusion in posterior cortex and cerebellum is the most common pattern and it is followed by full recovery within few months whereas focal pattern of cortical hypoperfusion-metabolism seems associated with more significant neurological symptoms.
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