Utility of Flat Panel Detector CT (FPD-CT) in Perfusion Assessment of Brain Arteriovenous Malformations

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Introduction. Various imaging techniques have been applied to the assessment of perfusion changes within and around arteriovenous malformations (AVMs), with mixed results. Flat Panel Detector CT (FPD-CT) is a new technique that can easily be performed in less than one minute immediately before, during, or after an endovascular procedure. The applicability of FPD-CT for perfusion assessment has been shown to be of good reliability in patients with ischaemia [1] and there is evidence that this modality provides information about the parenchymal blood volume (PBV) [2]. So far, the value of FPD-CT in AVMs has not been assessed. The aim of this study was to determine the role of FPD-CT in the evaluation of regional perfusion alterations in AVMs. FPD-CT perfusion data were compared with perfusion data obtained with two different MR-techniques: Dynamic Susceptibility Contrast MRI (DSC-MRI) and quantitative Arterial Spin Labelling (ASL).

Methods. Four patients with glomerular AVMs (patients 1-4) and one with a proliferative AVM (patient 5), without previous treatment or haemorrhage, were investigated with FPD-CT on a biplane FPD-angiography system (Axiom Artis dBA, Siemens), and also with DSC-MRI and Vessel-Encoded Pseudocontinuous ASL (VEPCASL) [3-5] on a 3T TIM Verio scanner (Siemens, Erlangen, Germany). For FPD-CT, visualisation of contrast agent within the superior sagittal sinus (SSS), supposed to reflect a steady state of contrast agent between the vascular and parenchymal compartment, was selected as the trigger time point for data acquisition. For ASL, absolute cerebral blood flow (CBF) and blood arrival time were obtained, and for DSC, relative CBF (rCBF) and cerebral blood volume (CBV) were calculated. Eight regions of interest (ROI) were drawn on a T1-weighted image with contrast (T1wCE) using the ITK-SNAP software as follows: Two peri-nidal (pn), two vicinity (vic), two remote (rem), and the thalamus and putamen for reference (ref), as well as corresponding ROIs in the contralateral hemisphere. All masks were superimposed onto the various data maps.

Results. The peri-nidal ROIs superimposed on the FPD-CT map showed a good overlap with normal appearing tissue (Fig. 1). In the four glomerular AVMs perfusion ratios were highest in the peri-nidal areas with decreasing values with increasing distance from the AVM. This observation did not apply to the proliferative AVM (Fig. 2). For FPD-CT, variability in the values of the peri-nidal masks across patients 1-4 was lower than for DSC, but higher than for ASL (Fig. 2). FPD-CT perfusion data correlated best with ASL-CBF (r= 0.60), followed by DSC-rCBF, whereas the correlation with DSC-CBV was weak. FPD-CT showed a moderately negative correlation with ASL-arrival time (Fig. 3).

Discussion. The good overlap of the peri-nidal ROIs with normal appearing tissue argues for the reliability of the FPD-CT data. In contrast to studies on ischaemia, a high correlation between FPD-CT and CBV could not be observed [2]. However, the relatively high correlation between FPD-CT perfusion and ASL-CBF, followed by rDSC-CBF indicates that in AVMs perfusion data obtained with FPD-CT are closer to CBV than to CBF. This can be explained by the early appearance of contrast in the SSS in AVM patients, i.e. the vascular and parenchymal compartments have reached a steady state, due to a high-flow shunting effect. The different perfusion patterns between glomerular and proliferative AVMs are most likely due to differences in composition of abnormal vessels between AVM subtypes, i.e. a local area of enlarged vessels in the glomerular and a multifocal distribution of fragile vessels in the proliferative subtype.

Conclusion. With FPD-CT images are weighted by both CBF and CBV, depending on the trigger time point chosen for data collection and the type of vascular lesion present. This challenges the utility of FPD-CT for a reliable perfusion analysis in AVMs. Differences in perfusion patterns between AVMs are most probably related to differences in abnormal vessel composition between AVM subtypes.