Is there evidence of cerebral arterial inflow hyperemia in idiopathic intracranial hypertension or not?

Gibt es Hinweise auf eine zerebrale arterielle Zuflusshyperämie bei idiopathischer intrakranieller Hypertonie oder nicht?

Dear Editor,

Whether or not there is evidence of an increase in the arterial inflow in patients with idiopathic intracranial hypertension (IIH) has remained controversial. In a recent paper, of the 42 children referred for possible IIH, 45 % had an arterial inflow which was greater than two standard deviations above the mean for the matched controls i. e. they were hyperemic [1]. Modelling of the venous outflow indicated that the increase in blood flow was high enough to induce IIH in these individuals [2]. In their most recent review paper published in the journal Fortschr Röntgenstr, Juhasz et.al. comment that the hypothesis of hyperemia in IIH could not be confirmed [3] and referred to one of their earlier articles as evidence corroborating this [4]. I wish to discuss some discrepancies I have noted in the author's comparison paper.

In the original paper referenced by the author's review, there were 24 IIH patients, who had an arterial inflow averaging 1090 ± 300 ml/min, the controls had a mean inflow of 850 ± 150 ml/min, a significant difference [5]. In the author's comparison paper, there were 20 IIH patients who had an arterial inflow averaging 1189 ± 235 ml/min, with the controls having an arterial inflow averaging 1143 ± 261 ml/min i. e. no significant difference [4]. We can see that the value for the arterial inflow for the IIH patients is almost identical in both papers but the control values are vastly different. Which control value is correct? The expected brain weight for a middle aged female is 1280 g [6], suggesting the controls in the author's paper had a global cerebral blood flow (CBF) averaging 89 ml/100 g/min. In other studies, the CBF in this age group using a similar combination of phase contrast blood flow estimation and brain weight estimation using 3 D T1 imaging was 57.0 ml/min/100 g in males [7] and 58 ml/100 g/min in females, with a comparison arterial spin labelling method also giving a value of 58 ml/100 g/min in females [8]. Therefore, the author's controls had a probable CBF which was 53 % higher than the published normal comparison papers. Clearly, this discrepancy requires an explanation.

The second area of discrepancy is more problematic. The authors found that the proportion of the total cerebral blood flow provided by the anterior circulation (both carotids) was increased by 17 % in IIH compared to controls ($p = 0.05$) but the posterior circulation was reduced by 30 % ($p < 0.01$) [4] but declined to discuss this further. Clearly, according to these findings, the anterior circulation was hyperemic but the posterior was ischemic. The anterior to posterior circulation ratio they found in the controls was 72 %/28 % and was similar to other authors [7, 9] but the IIH patients were very abnormal with 82 % /18 %. There have been several papers measuring the regional cerebral perfusion using various techniques in IIH but none have indicated that there is generalized ischemia affecting the thalamus, occipital lobes, brainstem and cerebellum [10]. Interestingly, one such study noted 2/16 of their patients had a global perfusion which was two standard deviations above the mean compared to their controls (77 and 78 ml/100 g/min) but declined to suggest that these patient's findings were significantly increased [10].

Regarding the high flow in the controls, I would encourage the authors to look back at their original paper, and if possible, measure the brain weights from the 3DT1 data (if acquired) to check for any evidence of megalencephaly. The other possibility for a systematic error would be if the controls had elevated carbon dioxide levels or anemia. The apparent relative reduction in blood flow in the posterior circulation in IIH could be due to an artifact if there was evidence of significant flow aliasing in the basilar artery. This is a known pitfall of the phase contrast technique. This should be checked in the original raw data and if found could be corrected.

Conflict of Interest

The authors declare that they have no conflict of interest.

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