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Case Report

The Value of Doppler Study of Central Retinal Artery in Diagnosis of Increased ICP

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Abstract

Purpose: To evaluate the diagnostic value of CRA Doppler study in case of increased intracranial pressure.

Methods: We used High Frequency Probe 8MH to study Central Retinal Arteries Doppler by oblique axial view.

Case Report: A case of 32-year-old patient with a moderate Head injury and clear signs of Brain edema in CT [bilateral frontal hemorrhage, sulci effacement, absent CSF in quadrigeminal Cisterna], also, Trans cranial U/S and ONSD revealed signs of increased ICP[LT MCA PI 1.29, RT MCA PI 1.36, LT ONSD 0.62cm, RT ONSD 0.64].

We did complete Doppler study of both CRAs [color and spectral] in this patient during admission, both color and Spectral Doppler revealed absent of diastolic flow which was correlated with Brain edema in CT, and signs of increased ICP in TCD, moreover, repeating the test after 3 days of dehydrating measures and marked improvement of patient condition revealed marked improvement in color flow as well as normalization of CRA RI [0.5]. This improvement was correlated with normalization of PI in both MCAs.

CRA is a superficial, easy accessible without bony obstacle like TCD and the learning curve of its Doppler study can be very steep, so it could has a big role in management of cases with increased ICP.

Conclusion: The change of Doppler flow of CRA can give a good idea about the dynamic changes of ICP during management of Brain edema, this probably will need a big study.

Keywords: *Optic Nerve Sheath Diameter; TCD; Increased ICP; Central Retinal Artery Doppler; CRA Resistivity Index*

Abbreviations

CRA: Central Retinal Artery; MCA: Middle Cerebral Artery; RI: Resistivity Index; PI: Pulsatility Index; ICP: Intracranial Pressure

Introduction

In neurocritical care, the detection of raised intracranial pressure (ICP) remains crucial as it is associated with poor outcome [1]. Invasive ventricular devices are the "gold standard" for continuous and reliable measurement of ICP; however, their placement could be challenging due to blood coagulation disorder or lack of surgical availability [2]. Moreover, malfunction or obstruction of ventricular catheters has been reported to occur as often as 6% [3].

Recently, Julie., *et al.* revealed in his met analysis that ONSD has a good level of diagnostic accuracy for detecting intracranial hypertension, with a pooled sensitivity of 0.9 [4].

The central retinal artery is an end artery branch of the internal carotid artery that joins the optic nerve 1cm behind the globe and enters the retina on the optic nerve head.

Central retinal artery is located inside the optic nerve sheath, and The optic nerve is part of the central nervous system and the intraorbital subarachnoid space surrounding the optic nerve is subject to the same pressure changes as the intracranial compartment [5,6], so we should expect any increase of ICP will compress the central retinal artery exactly the same as basal cerebral arteries.

Central retinal artery circulation is low resistance circulation with good diastolic flow and upper limit of resistivity index is 0.7 [7].

So, any compression of CRA will decrease the diastolic flow and increase RI.

Kamil., *et al.* studied the blood flow velocity changes in orbital arteries by using Doppler sonography in eight patients with brain death and increased ICP. Peak-systolic and end-diastolic velocities and resistive indices of the ophthalmic and central retinal arteries were evaluated. they observed the absence or reversal of end-diastolic blood flow in these arteries.

If the intracranial pressure is higher than the end-diastolic pressure of the cerebral arteries, diastolic flow reversal occurs. If the intracranial pressure exceeds systolic pressure, blood flow is entirely ceased with complete and irreversible loss of brain function [8].

We did a Doppler study of CRAs in case of moderate RTA patient with brain edema and found a close correlation with increased ICP and flow changes of CRAs.

Case Report

A 32-year-old male patient was admitted to our ER as a case of Head injury due to RTA. He was unconscious irritable, with GCS 8/15 E1 M5 V2, hemodynamically stable BP 140/75, HR 80/min sinus, O_2 saturation 98% on 2 litres nasal cannula, Chest Exam: clear, Heart: S1S2, Abdomen: soft, lax.

Because of bad GCS and severe irritability, patient was connected to mechanical ventilation.

CT was done and revealed evidence of brain edema [effacement of sulci, bilateral frontal contusion, absent quadrigeminal Cistern CSF].

Trans cranial U/S at the same time revealed:

1- Bilateral frontal hyper echoic lesions denoting hemorrhage.

2- LT MCA PI 1.29.

3- RT MCA PI 1.36.

Both denoting increased ICP and put the patient at high risk category.

4- LT ONSD 0.62.

5- RT ONSD 0.64.

6- We did at the same time both color and spectral Doppler of both central retinal arteries which revealed absent diastolic flow, and this was confirmed by spectral Doppler.

Patient was received a dehydrating measures [mannitol, hypertonic saline, frusemide].

We repeat all studies after 72 hrs which revealed an improvement of the flow in both MCA with normalization of PI in both sides [RT side 0.65, LT side 0.78].

This improvement of flow in MCA was correlated with normalization of flow and RI in both CRAs [RT CRA RI 0.5].

Meanwhile, the patient improved a lot and GCS increased to 13/15 E4 M5 V4, extubated and shifted to the surgical Ward.

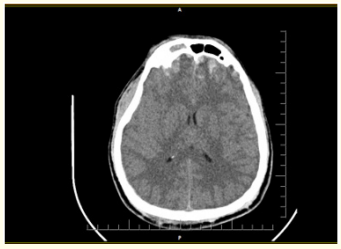

Figure 1: *CT brain, bilateral frontal contusion.*

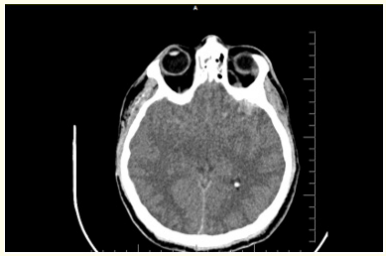

Figure 2: *CT brain, sulci effacement.*

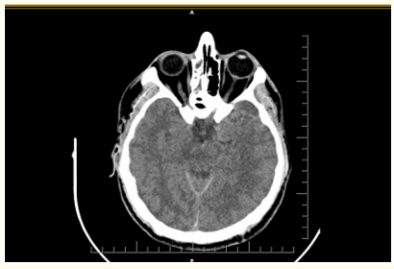

Figure 3: *CT Brain, absent CSF in quadrigeminal Cistern, denoting Brain Edema.*

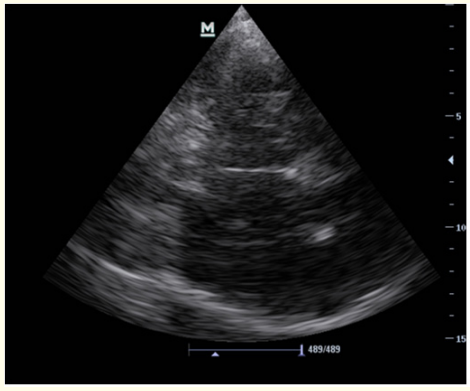

Figure 4: *TCD, Bilateral frontal contusion.*

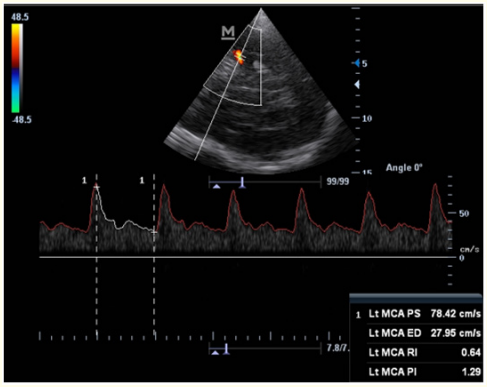

Figure 5: *TCD LT MCA PI 1.29 [increased, denoting increase in ICP].*

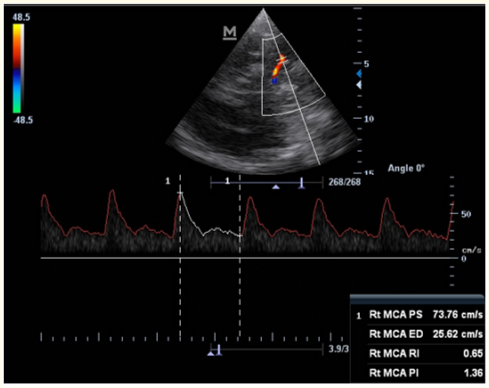

Figure 6: *TCD RT MCA PI 1.36 [increased, denoting increase ICP]*

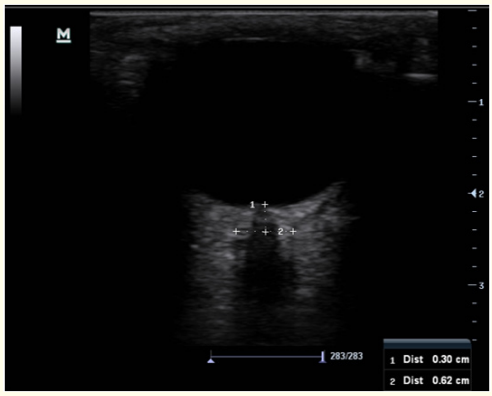

Figure 7: *TCD LT ONSD 0.62[increased, denoting high ICP].*

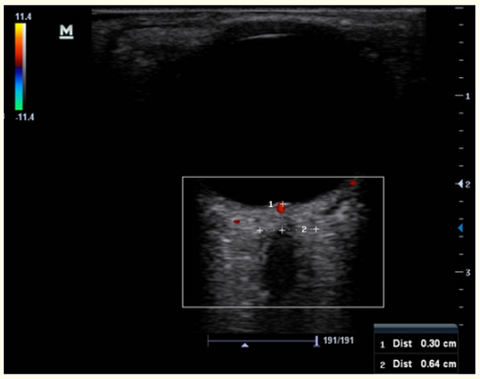

Figure 8: *TCD RT ONSD 0.64 cm [increased, denoting high ICP].*

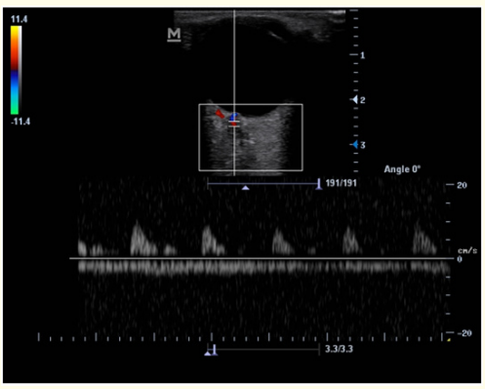

Figure 9: *Spectral Doppler of RT CRA revealing absent diastolic flow due to high intracranial pressure.*

Figure 10: *Video of the color Doppler of RT CRA revealing absent of diastolic flow due to increased ICP.*

https://www.dropbox.com/s/kfd2xj3s3qy569i/CRA%20bad%20FLOW.mp4?dl=0

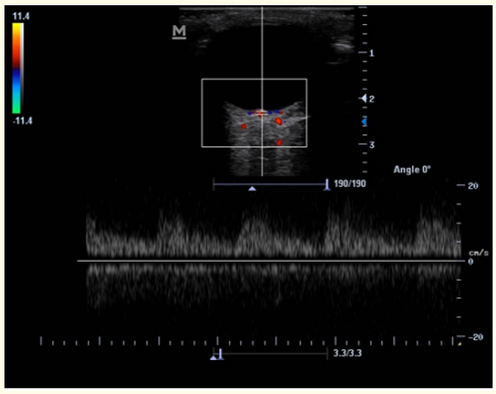

Figure 11: *F/U study Spectral Doppler CRA after dehydrating measures revealing very good diastolic flow with normalization of RI 0.5.*

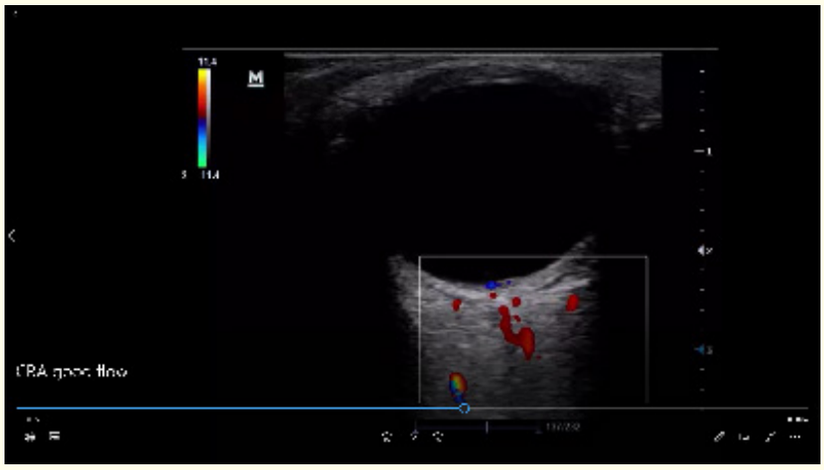

Figure 12: *Video of color Doppler of RT CRA revealing good diastolic flow after decreasing ICP*

https://www.dropbox.com/s/agq0qwd33uajb9n/CRA%20good%20flow.mp4?dl=0

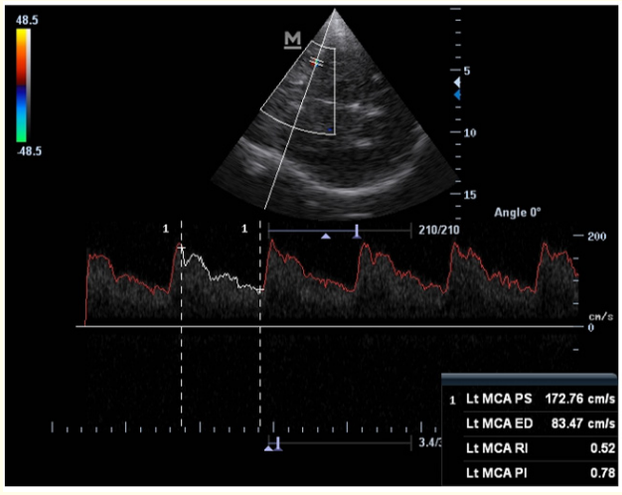

Figure 13: *F/U TCD LT MCA PI 0.78 [normal].*

Discussion

We present a case of 32-year-old male patient who was a victim of RTA with moderate Head trauma and GCS 8/15. There was a clear evidence of Brain edema in CT, bilateral frontal contusion, effacement of sulci, and absent CSF in quadrigeminal Cistern.

We did Trans cranial U/S at the same time, it revealed clear bilateral frontal contusion which was correlated with CT picture, it also showed a clear evidence of increased intracranial pressure by, first, bilateral increase of MCA PI, LT 1.29, RT 1.36, both more than 1.25 which put the patient at high risk category [9], Second, bilateral increase of ONSD, LT ONSD 0.62 cm, RT ONSD 0.64 cm.

Non-invasive ocular ultrasonography has recently been proposed to detect elevated ICP. The retrobulbar segment of the optic nerve is surrounded by a distensible subarachnoid space which can inflate during increase in CSF pressure [6,10] and this was found in our case report.

In our case of moderate Brain injury and increased ICP, we examined the Blood flow of both central retinal arteries by spectral and color flow Doppler.

The central retinal artery is an end artery branch of the internal carotid artery that joins the optic nerve 1cm behind the globe and enters the retina on the optic nerve head.

Central retinal artery is inside the optic nerve sheath, and The optic nerve is part of the central nervous system and the intraorbital subarachnoid space surrounding the optic nerve is subject to the same pressure changes as the intracranial compartment [5,6], so we should expect any increase of ICP will compress the central retinal artery exactly the same as basal cerebral arteries.

Central retinal artery circulation is low resistance circulation with good diastolic flow and upper limit of resistivity index is 0.7 [7].

Any compression of CRA will decrease the diastolic flow and increase RI.

In our patient, we clearly saw the absent of diastolic flow in both color and spectral Doppler on admission during Brain edema, and this was correlated with decrease of diastolic flow velocity and increase of PI in both MCAs, moreover it was matching the increase of ONSD in both optic nerves.

Amazingly, this absent diastolic flow in both CRAs improved a lot after 3days treatment by aggressive dehydrating measures, this was clearly seen in color Doppler, and in Spectral Doppler there was a normalization of RT CRA RI [0.5].

This was matching the improvement and normalization of both MCAs PI [LT MCA PI 0.78, RT MCA PI 0.65].

To our Knowledge, no one studied the CRA Doppler flow velocity in case of Brain edema, and the matching of Doppler flow between CRAs and MCRs during brain edema.

Karaali., *et al*. studied that the blood flow velocity changes in orbital arteries by using Doppler sonography in eight patients with brain death and increased ICP. Peak-systolic and end-diastolic velocities and resistive indices of the ophthalmic and central retinal arteries were evaluated. They observed the absence or reversal of end-diastolic blood flow in these arteries [8].

If the intracranial pressure is higher than the end-diastolic pressure of the cerebral arteries, diastolic flow reversal occurs. If the intracranial pressure exceeds systolic pressure, blood flow is entirely ceased with complete and irreversible loss of brain function.

Conclusion

The change of Doppler flow of CRA can give a good idea about the dynamic changes of ICP during management of Brain edema, this probably will need a big study.

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