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Ice Cream Headache: Cerebral Blood Flow Evaluation

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Ice cream headache is a well-known type of headache occurring in children and adults. The recent International Headache Classification (ICHD-3,4.5.2) describes the ice cream headache as a "short-lasting frontal or temporal pain in susceptible people by the passage of cold material over the palate and/or posterior pharyngeal wall."[1] The pathophysiology of ice cream headache is not well known. It has been postulated that ice cream headache may represent a model of migraine. A vascular phenomenon or a neurovascular disturbance may have a role in the pathogenesis of ice cream headache. Unfortunately, studies evaluating the role of cerebral vascular velocity in pathogenesis are limited.[2,3]

A 9-year-old boy was brought to the pediatric out-patient clinic complaining of headache triggered by eating ice cream, for at least one year. The patient described his headache as typically sharp, stabbing pain with bilateral frontotemporal location, disappearing within approximately one minute when he stops ingestion of ice cream. He avoids eating ice cream and drinking water with ice. Migraine or migraine-variants were not documented from personal and family histories. We evaluated cerebral blood flow of both the patient and an age- and gender-matched control before and during eating ice cream. The informed consent forms were taken prior to the evaluation. Middle cerebral arteries (MCA) were examined through the temporal window, using a 2-MHz pulsed-wave Doppler ultrasound (Toshiba Aplio Unit, Tokyo, Japan). Maximum velocities (Vmax), end diastolic velocities (Ved), resistive index (RI), and pulsatility index (PI) values and flow were measured 3 times by the same radiologist, and average values were obtained (Table 1). Velocities measured during eating ice cream were very slightly lower than before, both in the patient who developed headache during measurement and the control who did not. Flow volume was also decreased slightly. All measurements, including velocities and RI, were within normal limits both in the patient and the control.

In the short case report, we measured the middle-cerebral-arterial flow velocities before and while eating ice cream. We found a slight decrease in flow velocities, in both the child with ice cream headache and the healthy child. In another study including 3 subjects, the middle cerebral blood flow velocity was measured during eating ice cream. The author of that study detected a decrease in mean flow velocities of 2 subjects who developed a headache. However, no change was observed in the flow velocity of the subject without a headache while eating ice cream. Therefore, he postulated that decrease of cerebral blood flow secondary to vasoconstriction might be important in the development of ice cream headache.[2] Our finding was partially compatible with the opinion mentioned above because of a similar decrease in cerebral blood flow velocity of our child without headache. Although the exact mechanism of ice cream headache is not clear, cerebral vascular changes are accepted as one of 2 theories. According to the theory, the exposure of the palate or the posterior pharyngeal wall to a very cold substance may trigger rapid constriction and dilation of vessels resulting in activation of the nociceptors in the vessel wall.[4] In another large-scale study to determine the change in mean flow velocity and cerebrovascular resistance of MCA, 77 healthy adult volunteers were analyzed by Hensel et al.[3] Interestingly, in contrast to our finding and previous studies, volunteers with a headache attack provoked by ice water ingestion had higher mean flow velocity rates than volunteers without headache.

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Table 1. Results of Transcranial Doppler Study of Right and Left Middle Cerebral Arteries (MCA) When Not Eating and When Eating Ice Cream. Patient (a) and Control (b)

	MCA				
a.	**Vmax (cm/sn)**	**Ved (cm/sn)**	**RI**	**PI**	**Flow (L/min)**
Right					
Before	119.7	55.5	0.54	0.79	1.88
During eating	97.4	41.7	0.57	0.88	1.52
Left					
Before	121	57.5	0.52	0.75	2.04
During eating	84.4	38	0.54	0.78	1
	MCA				
b.	**Vmax (cm/sn)**	**Ved (cm/sn)**	**RI**	**PI**	**Flow (L/min)**
Right					
Before	129.9	62.2	0.52	0.79	2.99
During eating	110	48	0.55	0.85	2.14
Left					
Before	109.3	56.9	0.48	0.7	3.75
During eating	107.6	60.9	0.43	0.6	3.03

*Vmax, maximum velocities; Ved, end diastolic velocities; RI, resistive index; PI, pulsality index (PI) values and flow.

Additionally, volunteers with a positive ice cream headache history but negative headache provocation had a moderate mean flow velocity increase. They explained the mechanism of increased cerebral blood flow velocity by a reduction in cerebrovascular resistance secondary to trigeminal-parasympathetic activation[3]. However, it remains unclear to say mean flow increase instead of decrease is the most important pathogenic mechanism in light of the literature, including our result. The contrary findings of Hensel et al. may be related to recruitment of only adult volunteers and continuous recording of the velocity of both MCA during a study protocol involving the ingestion of lukewarm and ice water. Moreover, they expressed all data as mean at the end of ice water ingestion.

In conclusion, further studies are required, especially in children, to clarify the mechanism of ice cream headache.

Informed Consent: Written informed consent was obtained from all participants who participated in this study.

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