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V Leuzzi (vincenzo.leuzzi@uniroma1.it)[1], M Mastrangelo[1], A Celato[1], C Carducci[2], C Carducci[2]
1.Department of Pediatrics, Child Neurology and Psychiatry, Sapienza, University of Rome
2.Department of Experimental Medicine, Sapienza, Università di Roma
Correspondence: V Leuzzi, MD, Child Neurology Division, Department of Paediatrics, Child Neurology and Psychiatry, Via dei Sabelli 108, 00185 Roma, Italy.
Tel: ++39 0644712282 |
Fax +39644712229 |
Email: vincenzo.leuzzi@uniroma1.it

## Transient ischaemic attack in a case of a morbidly obese girl associated with metabolic syndrome

Sir,

It is well known that transient ischaemic attack (TIA) associated with hypertension occurs in the elderly, but there is scant information available on the incidence of such a condition in children (1). We recently encountered a morbidly obese girl who developed sudden onset of hemiparesis, transient symptoms with no neurological sequelae, and was diagnosed with TIA. Transiently worsened hypertension with a background of metabolic syndrome associated with increased insulin resistance was considered to contribute to the underlying pathogenesis that caused the TIA in the present patient.

A 12-year-old girl, who had been suffering from simple obesity since entering elementary school, fell down outside at her home in the morning hours of June, when she was intending to go to school. On that occasion, she felt sudden weakness in her left upper and lower leg, followed by a temporal blackout (several seconds). Her blood pressure was immediately measured by automated sphygmomanometer by her mother, who worked as occupational health nurse, and was unexpectedly elevated to 160/102 mmHg. The patient was unable to communicate smoothly, but she could complain of mild sensory disturbance on the left side of her body. However, all of these symptoms subsided rapidly (the duration of the symptoms was within 10 min), when she was brought to the emergency visit from the hospital.

Upon her arrival at the hospital, her consciousness was clear, and the systolic blood pressure was 150/95mm Hg. Her height was 162 cm (+1.8 SD above the average Japanese girls height for age), and body weight, 75 kg (+3.8 SD). Her BMI (body mass index) was 28.3 (>97th percentile). Her growth chart is shown in the Figure 1. By further detailed questioning, it was confirmed that she had not lost any memory of the entire course of events except for the loss of several seconds. No abnormal neurological signs including the optic fundus were observed except for decreased left grip strength of 12 kg (right grip was not measured). In the family history taken by her parents, antihypertensive medications were indicated for essential hypertension. Ambulatory blood pressure monitoring of the patient during her daily life revealed that the average systolic and diastolic blood pressures were 145 and 85 mmHg, respectively, indicating the persistently elevated blood pressure.

As the cause of TIA, brain computed tomography, magnetic resonance (MR) imaging and MR angiography revealed normal parenchymal and vascular imaging: no findings of arteriopathies, including moyamoya disease, vasculitis or dissection, were evident. Laboratory examinations of plasma renin activity, plasma concentrations of electrolytes, ACTH, cortisol, thyroid hormones and catecholamines were all within the normal range. Thus, endocrine diseases responsible for secondary hypertension were ruled out.

In the evaluation of the patient's obesity, increased waist circumference (82.6 cm), associated with elevated serum triglyceride concentration (140 mg/dL) and elevated blood pressure, met the criteria of metabolic syndrome in Japanese children (2). Serum total cholesterol and high-density lipoprotein–cholesterol (156 and 50 mg/dL, respectively) levels were normal. The abdominal CT scan showed remarkable fatty changes of the liver and increased visceral fat accumulation [the proportion of visceral fat to subcutaneous fat was notably increased to 23.14%

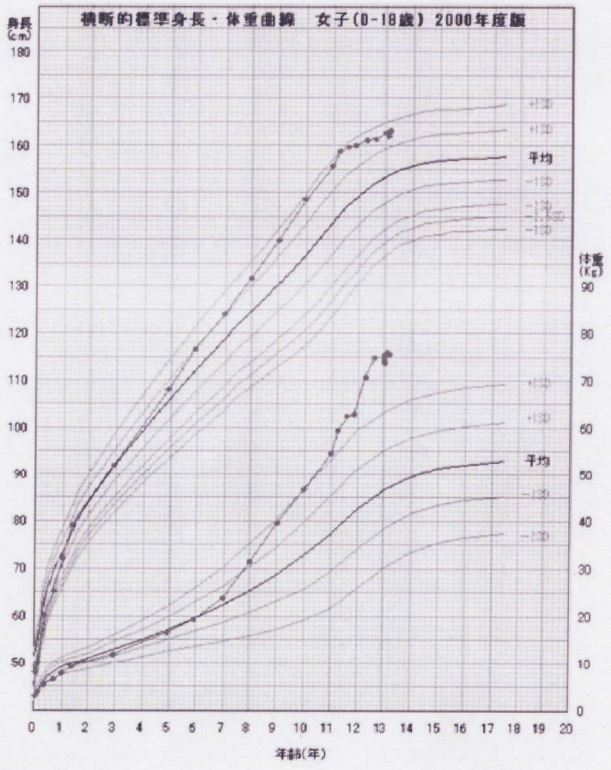


**Figure 1** Standard growth curve for healthy Japanese female children in 2000. Red line indicates the curve for our patient (upper: height, lower: weight).

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($60.30\ cm^2/200.32\ cm^2$)]. The calculated homoeostasis model of assessment–insulin resistance based on the concentrations of blood sugar (80 mg/dL) and insulin (29 $\mu$U/mL) by fasting blood sampling revealed 5.7 (normal $< 1.6$), indicating the remarkably increased insulin resistance. Therefore, we considered that the cause of TIA in the present obese girl was because of temporally worsened hypertension, which was a manifestation of the component of metabolic syndrome. In metabolic syndrome, hypertension may occur, in part, via the effect of insulin on renal sodium reabsorption or sympathetic nervous system activity (3). Furthermore, reduced cerebral vasodilatation in the state of insulin resistance may also enhance cerebrovascular events in metabolic syndrome (4).

Metabolic syndrome may increase the risk of cerebrovascular disease, even in children (5). To our knowledge, our case is the earliest manifestation of TIA because of hypertension associated with metabolic syndrome. As similar cases in childhood will increase in the future along with the emerging epidemic of obesity in developing countries, paediatricians should consider the appropriate management of hypertension (1,6) as a modifiable risk factor for cerebrovascular disease.

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**George Imataka (geo@dokkyomed.ac.jp), Go Ichikawa, Naoto Shimura, Osamu Arisaka**

Department of Pediatrics, Dokkyo Medical University, Shimotsuga-gun, Tochigi, Japan

Correspondence: George Imataka M.D., Ph.D., Department of Pediatrics, Dokkyo Medical University, 880 Mibu, Shimotsuga-gun, Tochigi 321-0293 Japan.

Tel: +81-282-86-1111 |

Fax: +81-282-86-2947 |

Email: geo@dokkyomed.ac.jp

## Methaemoglobinaemia caused by the ingestion of poisoned meat in a Romanian community in Italy

Sir,

Methaemoglobinaemia is a potentially life-threatening condition caused by the exposure of haemoglobin to oxidizing agents (1).

Less than one percent of haemoglobin's iron is normally oxidized. Haemoglobin's oxidized form (methaemoglobin) cannot bind oxygen and produces cyanosis, hypoxia, dizziness, nausea, malaise and, at higher levels, stupor, coma, metabolic acidosis and cardiovascular failure. The accumulation of methaemoglobin in the erythrocytes occurs for three main reasons: first, a dominantly inherited abnormality in haemoglobin's structure preventing the reduction of methaemoglobin to haemoglobin; second, a recessively inherited deficiency in the methaemoglobin-reductase enzyme; last, the exposure to haemoglobin-oxidizing chemicals or drugs such as nitrites/-nitrates in contaminated well-water and meat, which are the most common causes of methaemoglobinaemia (2). Children from lower social class or ethnic groups with specific dietary habits are at higher risk (3–5). G6PD deficiency should be excluded almost on familiar and personal history before methylene blue, the antidote for nitrite/nitrates-induced methaemoglobinaemia, is administered. We report two separate clusters of Romanian children living in the same urban setting in north-western Italy presenting within few days with dizziness, cephalalgia and, in two cases, cyanosis after consuming meat with high nitrate content.

In the first cluster, a 7-year-old boy was conducted to our Emergency Department because of vomiting, abdominal pain, cephalalgia, dizziness, malaise and cyanosis few hours after eating a hamburger. No history of favism was reported. On admission, he was in mediocre condition. Body temperature was 36.3°C. Physical examination revealed pale-grey skin with acrocyanosis. Cardiological, respiratory and neurological examinations were all normal; oxygen saturation rate was 87%. The patient remained cyanotic despite oxygen administration. Blood tests revealed severe methaemoglobinaemia (40.7%); the boy's condition rapidly improved after the administration of intravenous methylene blue, and methaemoglobin level returned to normal (0.6%) 4 h later. He was discharged 24 h later in good conditions. Further investigation included complete haemoglobin profile, proving negative. His parents, who ate the same hamburgers and referred dizziness and cephalalgia, had a methaemoglobin level of 5.9% and 4.3%, respectively; they were treated supportively with oxygen, monitored closely and discharged the day after. Also, the boy's uncles participated in the meal and referred dizziness and cephalalgia, but did not present to the Emergency Department. His 11-year-old sister did not eat hamburger, was asymptomatic and had normal methaemoglobin level.

Five days later, five siblings were conducted to our department because of abdominal pain, nausea, cephalalgia, dizziness and malaise after eating meat; one of them, ageing 5 and the most unwell, showed also cyanosis. His methaemoglobin level was 34.8%, so intravenous methylene blue was administered after history of favism was excluded. One hour later, the boy's condition returned to baseline (methaemoglobin: 1.9%). Out of the other siblings, three girls of 11, 13 and 15 years presented elevated methaemoglobin levels (12.5%, 14.4% and 6.6%, respectively) and were treated with intravenous methylene blue. Five hours later, their levels were 0.6%, 0.7% and 0.5%, respectively, and complete regression of the symptoms was observed. A fifth girl of 14 years with nausea and cephalalgia had a methaemoglobin level of 2.6%, was administered oxygen and monitored; 5 h later, her level was 0.6% and she was discharged.

Further investigations on these two separate clusters revealed they had all eaten meat bought at the same butcher shop. In the same period, two other different clusters of adults with methaemoglobinaemia after eating meat bought at the same butcher shop were reported in other hospitals of our area. A police investigation was conducted, and the butcher was found to adulterate the meat with high-dose nitrate in order to preserve it over the legally permitted time.

Ours are two typical clusters of methaemoglobinaemia after the ingestion of a high