J Anesth (1999) 13:110–111

Journal of
Anesthesia

Clinical reports

Recurrent facial paralysis with plasma hypertriglyceridemia and hyperlipoproteinemia: case report

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Key words: Recurrent facial paralysis, Child, Hyperlipoproteinemia

Introduction

Idiopathic facial paralysis in children under 15 years of age is rare [1] and is considered to have a good prognosis [2], because the ratio of the cross-sectional area of the facial nerve to that of the facial canal in children is smaller than in adults. This anatomical difference may be the reason for decreased facial nerve compression in children [3]. The etiology of facial paralysis still remains unclear. However, it is thought to be due to viral infections such as herpes simplex and varicella-zoster [4,5]. Recurrent facial paralysis may be due to either recurrent viral infection or exacerbation of indolent viral antigens within the nerve following recurrent viral exposure [6].

Abraham-Inpijn et al. [7] suggested that in patients with facial paralysis complicated by diabetes mellitus (DM), the incidence of recurrent facial paralysis is 2.5 times higher than in those without DM. Futher, the elevated triglyceride and total lipid levels seen in DM and vascular factors might be related to the etiology or pathogenesis of facial paralysis. It is generally agreed that the pathogenesis of facial paralysis is associated with ischemia of the facial nerve [8]. In children, hyperglyceridemia and hyperlipoproteinemia are known to be cardiovascular risk factors [9]. This case report describes a 14-year-old male patient who developed recurrent peripheral facial paralysis complicated by hyperglyceridemia and hyperlipoproteinemia.

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Received for publication on November 25, 1997; accepted on October 22, 1998

Case report

A diagnosis of recurrence of left-sided facial paralysis was made on a 14-year-old boy who was then introduced to our clinic. Two years earlier, he had experienced left-side facial paralysis and was treated with stellate ganglion blocks and physical therapy, leading to a complete recovery. His serum triglycerides (TG) were elevated to 360 mg·dl^{-1}; however, antiviral serum antibodies were not measured. His family history states that his mother has been hyperglycemic for the past five years and is presently on diet therapy.

He was hypertensive, with an initial evaluation showing 16 points (of 40 points) according to Yanagihara's criteria [10] and Group III according to the House-Brackmann grading system [11]. Electroneurography revealed 52% degeneration of the facial nerve. Antiviral serum antibodies were measured by ELISA. On the first visit, tests for herpes simplex virus, varicella-zoster, cytomegalovirus, Epstein-Barr, mumps, and rubella antibodies (IgG and IgM) were all negative, and the same examinations repeated 14 days and 6 weeks after the initial testing also gave negative results. Acoustic acuity according to the audiogram was within normal limits. No abnormal findings were observed on cephalic CT scan. Serum TG was elevated to 545 mg·dl^{-1} (normal, 30–150) on the first visit. The laboratory results were total cholesterol, 230 mg·dl^{-1} (normal, 130–250); chylomicrons, 34.6 mg·dl^{-1} (normal, 8.7–18.5); pre-β-lipoprotein, 314 mg·dl^{-1} (normal, 74–147); β-lipoprotein, 609 mg·dl^{-1} (normal, 140–440); LDL-C, 102 mg·dl^{-1}; phospholipid, 308 mg·dl^{-1} (normal, 140–230); HDL-C, 65.6 mEq·l^{-1} (normal, 42–63); ApoAI, 132 mg·dl^{-1} (normal, 112–162); ApoAII, 56.4 mg·dl^{-1} (normal, 25.9–37.7); ApoB, 86 mg·dl^{-1} (normal, 59–99); ApoE, 8.9 mg·dl^{-1} (normal, 2.9–5.3); ApoCII, 5.6 mg·dl^{-1} (normal, 2.2–4.6); and ApoCIII, 11.0 mg·dl^{-1} (normal, 4.5–10.5). Type I hyperlipoproteinemia was suspected from those data. Diet therapy was started for hyperlipoproteinemia and

hypertriglyceridemia. Four months after the onset of diet therapy, his TG decreased to 130 mg·dl^{-1}; however chylomicrons, ApoAII, and ApoE were still high (23.2, 41.9, and 6.4 mg·dl^{-1}, respectively). Thyroid function, blood sugar, urinary protein, and liver function for secondary hyperlipoproteinemia and hypertriglyceridemia did not show any abnormalities. For the facial paralysis, stellate ganglion block and physical therapy were applied. The patient's facial paralysis showed complete remission in 5 months.

Discussion

Although the etiology of facial paralysis remains unclear, Kettel's hypothesis [8] is generally accepted. It is commonly speculated that local ischemia can cause edematous swelling of the facial nerve to induce palsy. Abraham-Inpijin et al. [7] suggested that vascular factors might be related to the etiology or pathogenesis of facial paralysis. Blunt [12] suggested that vascular disorders might cause edema of the facial nerve to compress nerve fibers within the bony structure of the Fallopian canal. In animal experiments, facial nerve degeneration is easily provoked by elevating blood pressure [13]. Children with hyperlipidemia are reported to have had atherosclerosis and disease of the microvasculature in childhood [14,15]. Facial paralysis associated with hypertension has been reported in children [16,17]. Our patient's hypertension, probably due to hyperlipoproteinemia and hypertriglyceridemia, may have been the cause of his facial paralysis.

The incidence of facial paralysis in children is lower than in adults, and recovery from the palsy is better in children than in adults [1,2]. Because the facial canal cross-sectional area ratio in children is smaller than in adults, compression of the facial nerve is less probable in children than in adults [3]. Nevertheless, recurrence of facial paralysis is more frequent in young patients than in adults [18]. These patients may have anatomical or metabolic problems that lead to the compression of the facial nerve. Recurrent facial paralysis may be caused by either a recurrent viral attack or exacerbation of indolent viral antigens within the nerve with recurrent viral exposure [19]. In our patient there was little probability of viral infection, as several antiviral serum antibodies were negative on three spaced examinations. Thus, hyperlipoproteinemia and hypertriglyceridemia with subsequent vascular disorders such as hypertension may be associated with the onset of paralysis in this case.

In summary, a 14-year-old boy who suffered from recurrent facial paralysis complicated by hyperlipoproteinemia and hypertriglyceridemia was observed. Hypertension probably caused by hyperlipoproteinemia and hypertriglyceridemia may be intimately associated with the pathogenesis of his facial paralysis.

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