Anaphylactoid reaction to intravenous methylprednisolone in a patient with multiple sclerosis

A 44 year old woman was admitted to our clinic because of progressive multiple sclerosis. One year before admission she had developed paresthesias of the legs, and subsequently of the arms. She became incontinent for urine and feces. She complained of numb feelings and muscle cramps in her legs. The medical history mentioned hypertension for which she used propranolol and hydrochlorothiazide. The family history was negative for multiple sclerosis. On examination there was vertical nystagmus, slight paresis of the arms she had normal coordination of the arms, and loss of sensation from a mid-thoracic level. The tendon reflexes of the legs were very brisk, and both plantar responses were extensor. Examination of CSF showed eight white cells/mm³ (all lymphocytes), and an immunological production of IgG and IgM. Brain MRI and the cervical part of the spinal cord showed multiple white matter lesions. Additional investigations excluded other diseases—for example, borreliosis and lupus erythematosus. A 10 day treatment with daily administration of 1000 mg intravenous methylprednisolone was started. Methylprednisolone was given in its injectable form, methylprednisolone sodium succinate, which hydrolyses to methylprednisolone in the body. The infusion period was one hour. Because of cystitis she received intravenous clemastine, and no symptoms developed.

After the first infusion there was a reaction of the skin rash, and difficulty with swallowing and breathing, suspicious of an allergic reaction. Our patient developed a skin rash reaction. Our patient developed a skin rash reaction after intravenous methylprednisolone treatment. Additional investigations were performed to elucidate the mechanism of this reaction to intravenous methylprednisolone.

The “allergic” reactions are probably not based on an IgE mediated allergy, but could have been caused by fast administration of methylprednisolone leading to high plasma concentrations. Therefore, various concentrations were not found. The histamine release reaction for methylprednisolone sodium succinate was not indicative of an IgE mediated reaction, but could have been caused by a (dose related) toxic effect of methylprednisolone on the basophil granulocytes. In conclusion, the clinical symptoms which developed during high dose intravenous methylprednisolone are rare, but can be dangerous. Therefore, patients with multiple sclerosis who receive an intravenous methylprednisolone treatment for the first time should be carefully monitored. According to this case the mechanism of the reaction seems to be IgE independent, and may have been induced by toxic concentrations of methylprednisolone on the basophil granulocytes. Skin testing with methylprednisolone is unreliable, and should be interpreted with care.
Unilateral auditory hallucinations: ear or brain?

Brasic and Perry convincingly describe a boy with unilateral otopathic auditory hallucinations. However, their literature review is very much limited. They list four CNS causes for auditory hallucinations other than from a hyperactive ear. They also do not cite a relevant prior case.

Their paper starts: "Unilateral auditory hallucinations...are associated with contralateral CNS lesions". Their only supporting reference is Toulouse (1892), who reviewed four his ears or hearing; his 1892 case, also an unilateral auditory hallucination due to a clear neurological lesion in someone with normal ears and hearing, the only proved cause of auditory hallucinations is otological.

CORRESPONDENCE

Brasic and Perry reply: Gordon conjectures that auditory pathology is the necessary and sufficient condition for auditory hallucinations. We disagree. We hypothesise that auditory hallucinations have many aetiologies which can be classified as otological, neurological, neuropsychiatric, and combined. Auditory hallucinations may result from the multiple effects of otopathological, such as altered signal transduction in hair cells. For example, in response to minimal environmental stimuli, diseased cochlear otology, such as altered signal transduction in hair cells. We have found frequencies producing white noise perceived as tinnitus in some persons. Auditory hallucinations may also result from neurological illnesses, including after right temporal lobectomy for intracerebral hemorrhage without seizures. We are preparing a manuscript about auditory hallucinations in neurological disorders. Auditory hallucinations due to neuropsychiatric disorders are being studied, particularly in schizophrenia. On functional MRI, two patients with schizophrenia experiencing auditory hallucinations showed external auditory activation alongside reduced responses of the temporal cortex to external auditory stimulation. Therefore, auditory hallucinations in some patients with schizophrenia may correspond with maximal activation of the auditory association cortex. The physiology of thinking in words was assessed utilizing PET in six persons with schizophrenia who experienced auditory hallucinations, six persons with schizophrenia.