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 paresthesia of the legs, and subsequently of the arms. She became incontinent for urine and stools, and 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 paresthesia of the fingers, no nystagmus, incoordination 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/mcL (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, borrellosis and lupus erythematosus. A 10 day treatment with daily administration of 1000 mg intravenous methylprednisolone 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 also received trimethoprim. One day after the intravenous methylprednisolone course had ended, the patient developed generalised urticaria which disappeared after a few days, and which could have been induced by either drug. After informed consent of the patient it was decided to give another course, as the intravenous methylprednisolone course improved her multiple sclerosis. To guarantee minimal risk, we gave 1000 mg intravenous methylprednisolone under close monitoring. After the first infusion there was a reactivation of the skin rash, and difficulty with swallowing and breathing, suspicious of anaphylaxis. We gave methylprednisolone intravenously after, and thought the symptoms immediately resolved. Because of clinical improvement, therapy was continued with a 1000 mg dose of intravenous methylprednisolone divided into two, again under close monitoring. No symptoms developed. The next day we gave the dose close after which the patient developed dyspnoea. We waited two days and reintroduced intravenous methylprednisolone therapy in divided doses. After the second dose the patient again became short of breath, needing 4.0 mg intravenous clemastine. We decided to give the patient the next two doses of 500 mg intravenous methylprednisolone followed by 4.0 mg intravenous clemastine, and no symptoms developed.

A skin reaction and histamine release test were performed to elucidate the pathogenesis of the reaction. Positive results were obtained with a skin reaction of 5.5 mm after subcutaneous injection of methylprednisolone (1.0 ml 5% methylprednisolone in isotonic saline). However, when the same solution was subcutaneously injected in nine healthy volunteers, skin reactions were not observed. Clemastine was given, and which could have been induced by either drug. After informed consent of the patient it was decided to give another course, as the intravenous methylprednisolone course improved her multiple sclerosis. To guarantee minimal risk, we gave 1000 mg intravenous methylprednisolone under close monitoring. After the first infusion there was a reactivation of the skin rash, and difficulty with swallowing and breathing, suspicious of anaphylaxis. We gave methylprednisolone intravenously after, and thought the symptoms immediately resolved. Because of clinical improvement, therapy was continued with a 1000 mg dose of intravenous methylprednisolone divided into two, again under close monitoring. No symptoms developed. The next day we gave the dose close after which the patient developed dyspnoea. We waited two days and reintroduced intravenous methylprednisolone therapy in divided doses. After the second dose the patient again became short of breath, needing 4.0 mg intravenous clemastine. We decided to give the patient the next two doses of 500 mg intravenous methylprednisolone followed by 4.0 mg intravenous clemastine, and no symptoms developed.

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Unilateral auditory hallucinations: ear or brain?

Brási and Perry convincingly describe a boy with unilateral otopathic auditory hallucinations. However, their literature review is very much biased towards their own CNS causes for auditory hallucinations other than from a hyperactive ear. They also do not cite a misleading, postulating dubious CNS causes. However, their literature review is very.

There are several factors that can cause auditory hallucinations from sensory disinhibition. For example, deprivation of hearing or sensory stimulation can lead to auditory hallucinations. Hearing loss can be due to various factors such as noise exposure, aging, or neurological diseases. Sensory deprivation can also occur as a result of hearing aids or cochlear implants. In both cases, the brain can adapt to the loss of sensory input and produce auditory hallucinations as a compensation mechanism.

In addition, auditory hallucinations can also be caused by neurological conditions such as schizophrenia, temporal lobe epilepsy, and traumatic brain injury. In these cases, the brain may not receive proper sensory input, leading to hallucinations. Neuronal discharges in the auditory cortex can also contribute to auditory hallucinations.

Furthermore, auditory hallucinations can be triggered by certain medications, such as antipsychotics and antidepressants. These medications can affect the balance of neurotransmitters in the brain, leading to hallucinations. Additionally, some people may experience auditory hallucinations as a result of alcohol or drug use.

In conclusion, auditory hallucinations can have various causes, including sensory deprivation, neurological conditions, and medication use. It is important to consider these factors when assessing patients with auditory hallucinations to provide appropriate and effective treatment.


11. Brási J and Perry R. Reply: Gordon conjectures that otopathological 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 otopathological, neurological, neuropsychiatric, and combined. Auditory hallucinations may result from the multiple effects of otopathological factors, such as altered signal transduction in hair cells. For example, in response to minimal environmental stimuli, disordered hair cells may produce abnormal frequencies producing white noise perceived as tinnitus in some persons. Auditory hallucinations may also result from neurological illnesses, including after right temporal lobectomy for treatment of intractable epilepsy. We are preparing a manuscript on auditory hallucinations in neurological disorders.
12. Auditory hallucinations due to neuropsychiatric disorders are being studied, particularly in schizophrenia. On functional MRI, two patients with schizophrenia experiencing auditory hallucinations showed reduced responses in 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.