Musical hallucinations associated with post-thyroidectomy hypoparathyroidism and symmetric basal ganglia calcifications

Bilateral symmetric basal ganglia calcifications most often result from parathyroid disorders and subsequent imbalances in calcium and phosphorus metabolism. Associated neurological and psychopathological symptoms are, however, controversial. "Musical hallucinations" are usually defined as the hearing of tunes and melodies without relevant external stimuli. They are reported to be very rare compared with verbal hallucinations.

A 63 year old, right handed housewife with a 40 year history of post-thyroidectomy hypoparathyroidism presented with a chronic progressive syndrome, with musical hallucinations, mild intellectual impairment, and cerebellar ataxia. She reported having suddenly started hearing music about five months before admission. The tunes, which were characterised by a strophing rhythm ("that terrible modern pop music"), did not vary much ("like a short tune being played all over again"). The hallucinations occurred continuously during the daytime, when awakeing at night, or when talking. Insight into the character of hallucinations was achieved after initial deception.

EAE examination showed a mild paracochlear perceptive hearing loss due to presbyacusis, more prominent on the left side. Neurolinguistic and neuropsychological examinations failed to show signs of aphasia, agnosia, visuospatial disorders, or hemispatial neglect. The patient had an IQ of 86 on the Hamburg Wechsler intelligence test for adults with low scores on verbal scales. Verbal and visual short term memory were impaired slightly, as measured by the Benton visual retention test and Reythauer (IST 70) test indicating acquired cognitive deficits of the organic type.

Electrophysiological examinations (EEG, topographical EEG, and acoustically and visually evoked potentials (AEP; VEP) showed no evidence of epileptic activity or consistent focal activity. P300, AEP, and VEP latencies were in the normal ranges.

Cranial CT showed dense areas of calcification predominantly involving the basal ganglia (for example, pulvinar thalamus, striatum), cerebellar dentate nuclei, and perventricular white matter (figure). At admission ECG showed massive prolongation of the Q-T interval (0.48-0.58; frequency adapted upper limit: 0.36 ms); ECG was normal on the day of discharge. Complete blood count and all routine laboratory tests were in the normal ranges, except serum concentrations of calcium on admission and discharge of 1.45 and 2.48 (normal range 2.0-2.75) mEq/L and inorganic phosphorus concentrations of 0.03 and 1.45 (normal range: 0.8-1.5) mg/l. Twenty four hour urinary calcium excretion rates on the day of admission and discharge were 0.77 and 0.17 (normal range: 3.25-8.25) mEq/L and inorganic phosphorus excretion rates were 7.3 and 21.7 (normal range: 10-32) mEq/L. Intact parathormone was below the detection limit (<1 pg/ml; normal range: 10-60 pg/ml).

The patient initially received 1500 mg CaCl2 and 4 mg dihydrotechochol orally per day. Three additional intravenous applications of CaCl2 were given to avoid manifest tetany. The hallucinations started to fade on day 12 of treatment. Phosphate was adjusted to 2 mg per day. Three weeks after admission the patient was free of hallucinations. Cerebellar symptoms, and signs of neuromuscular instability (Crepuscule's and Trousseau's signs) improved significantly. Tinnitus and mild deafness remained unchanged three months later, the patient had no complaints, and her neurological state was normal on the medication described.

The disappearance of the musical hallucinations coincided with the normalisation of serum electrolyte balance. Although electrolyte disturbances after hypoparathyroidism have long been known to cause neuropsychiatric symptoms, musical hallucinations have not been reported.

Two basic theories regarding the pathogenesis of complex auditory hallucinations have been proposed.

The "perceptual release" theory is based on the pathogenetic role of the sensory impairment. Our patient had had a mild progressive hearing loss associated with tinnitus. Despite the disappearance of auditory hallucinations, however, there was no improvement in hearing and tinnitus. This is not compatible with either the persistent nature of otogenic auditory hallucinations due to progressive hearing loss, or with transient hallucinations of acute otopathic origin, as no acute lesion could be found. Complex auditory phenomena may also occur with lesions of the tegumentum of the pons and lower midbrain; however, CT and AEP failed to disclose a brainstem lesion in our patient.

According to the other pathogenetic concept, local metabolic dysfunction might lead to a dissociation of circuits in the association cortex. In our patient, cranial CT showed that the principal areas of extensive bilateral calcifications included the striatum, the pulvinar thalamus, and the cerebellar dentate nuclei (figure). Calcifications of the dentate nuclei are usually thought to result in cerebellar ataxia. Medial (internal) pallidal efferents are supposed to inhibit thalamocortical neurons, and an increased thalamocortical drive to lateral orbitofrontal and anterior cingulate cortex due to lesions near the pulvinar thalami might lead to auditory hallucinations. There is still considerable controversy regarding a link

Cranial CT (unenhanced) with bilateral symmetric and confluent calcifications of dentate nuclei of the cerebellum, (A, B) pulvinar of thalamus and striatum (caudate nucleus and putamen), (C) and hemispheric white matter (C, D).
between symmetric basal ganglia calcifications and psychiatric or neurological symptoms, as no studies who associations regarding aetiology, localisation, volume, or symptoms have been ascertained. Patients with basal ganglia calcifications are, however, reported to be exceptionally vulnerable to metabolic and traumatic conditions. Thus an increased vulnerability due to increased thalamocortical drive associated with basal ganglia calcifications and an abnormal excitability of neurons due to the electrolyte imbalance might have brought forth the acoustic phenomena in our patient. There has been one previous case report describing a patient with bilateral basal ganglia calcifications who developed a chronic progressive neurological syndrome of extrapyramidal and cerebellar symptoms, pyramidal signs, and epilepsy 32 years after thyroideotomy, which improved partially after adequate treatment of hypoparathyroidism. In comparing this report with that of the present patient, differences in the clinical syndrome might well be explained by differing regions of involvement, in agreement with the distribution of calcifications.

In terms of epidemiology, no association between basal ganglia calcifications and particular neuropsychiatric symptoms could be established. Extensive basal ganglia calcifications in these two patients, however, may be able to interact with the effects of non-specific noxious conditions, such as electrolyte imbalances and determine their psychiatric symptoms. Although it is uncertain whether basal ganglia calcification progression can be stopped by adequate treatment of hypoparathyroidism, our patient shows that both pathopsychological and neurological symptoms may be improved. Therefore, prescribing "antipsychotic" drugs should be avoided due to the increased vulnerability to extrapyramidal side effects in patients with basal ganglia calcifications and the likelihood of neuroleptic non-response in this type of auditory hallucinosis.

Dr Fellner, Bad Windsheim, kindly permitted publication of CT scans.

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**MATTERS ARISING**

**Chronic fatigue syndrome**

Chronic fatigue syndrome: a follow up study by Bonner et al. reported that 47 patients initially diagnosed with "chronic fatigue" were contacted for follow up four years later. The authors indicated that "These patients were initially assessed before the current criteria for chronic fatigue syndrome became available, but most would have satisfied the criteria retrospectively" (p 617). At the outset, all patients were offered cognitive behavioural treatment and some were offered antidepressant medications. Each patient then made a decision to either undergo or decline cognitive behavioural treatment. Four years later, those patients who reported functional improvement were more likely to have elected to receive the cognitive behavioural treatment. Additionally, patients in the group that did not report any functional improvement were more likely to score higher on measures of depression.

The US Centers for Disease Control and Prevention (CDC) case definition, the proposed revisions to the CDC case definition, and the guidelines for research set forth by Sharpe et al. were cited, but the researchers did not make comparisons. We would like to note the authors did not specify just how many of the initial 47 patients met any of the cited criteria for chronic fatigue syndrome, as opposed to chronic fatigue. In short, they did not differentiate the exact number of chronic to either undergo or decline cognitive behavioural treatment.

Only 29 of the original 47 patients (62%) agreed to be interviewed for the follow up. Thus 18 (38%) of the original patients were not included in this study, where it was reported that subjects reported little or no improvement and 19 subjects reported improvement or recovery. The authors acknowledged that the small patient sample size constituted a methodological problem, but nevertheless concluded "that there is a strong association between successful completion of [cognitive] treatment and the absence of functional disability at the four year follow up" (p 620). They further suggest that costs associated with long term disability could be reduced by the utilisation of cognitive therapy in the treatment of chronic fatigue syndrome. We would like to emphasise that the small patient sample size, together with the lack of availability of almost 40% of the initial patients for interview at follow up, make such conclusions highly inappropriate.

There are other possible explanations for the results found in this study. Because of the ambiguity regarding the diagnostic criteria that was employed, it is unclear whether the two groups of patients came from the same population. Perhaps those who elected to have cognitive behavioural treatment were at a different stage of their illness and recovery than those who refused this treatment. It is also possible that subjects who opted for cognitive treatment had evidence that they were not coping effectively with life. Additionally, it is possible that those subjects who refused cognitive treatments might have had more evidence of a physiological illness so severe that they were not physically well enough to engage in cognitive therapies.

Although the researchers reported that no neurological or physical illnesses developed over four years, their diagnostic methods were not specified, other than references to telephone contact with physicians of non-responders. We would like to know the question of whether any illnesses that might have developed were missed. Based on the information in the article, it does not appear that comprehensive testing for any physical illness was part of the research protocol.

We find the report of no new neurological or physical illnesses after four years surprising, as two of the three patients in their own chronic fatigue syndrome related research, found that in the first 75 patients evaluated for fatigue, 50 had chronic fatigue syndrome and 28% had diagnoses of curable illnesses. Moreover, in this research, four patients with chronic fatigue syndrome developed concurrent illnesses such as hypothyroidism, hyperthyroidism, and diabetes.

Bonner et al. construe their finding of greater psychiatric morbidity at the follow up in the group that did not report any functional improvement as evidence that the "progression of severe chronic fatigue syndrome appears to be associated with psychiatric morbidity and in particular depression" (p 620). We, however, view these findings as evidence of reactive depression in this group of patients. In this research, the patients had a severe illness that did not improve, as there were no significant differences between the two groups on the Beck depression inventory or hospital anxiety and depression questionnaire four years earlier.

The presence of depression in any clinical situation needs to be considered and treated, whether the patient has chronic fatigue syndrome, coronary disease, cancer, or any other illness. The suggestion, however, that symptoms reported by patients with an illness for which there is not yet available any reliable diagnostic laboratory measure will only reflect the attention of their physicians (p 621) is not only highly unfair and unfounded, but it can also potentially hinder the medical support that these patients require.

Cognitive behavioural treatment might, in the end, be deemed to hold a place in the treatment of chronic fatigue syndrome. It is important, however, to note that cognitive behavioural treatment over an extended period can be extremely expensive, and the benefit of such treatment must be carefully assessed before any generalisations regarding its use are made. We very much hope that future studies on this issue will apply uniform criteria to the diagnosis of chronic fatigue syndrome, include adequately large numbers of patients, and randomly assign patients to various blinded treatment groups.
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