India. As per available information, KC was asymptomatic until 1 month ago. Over the next week, the family members observed KC to be withdrawn, quieter than usual and skipped work. He would reluctantly accept food and eat less than usual. Family members consulted general practitioners who advised multivitamins and supportive measures, with no improvements in KC's symptoms.

In the week preceding hospital visit, family members got concerned as KC would spend an entire day without speaking. He would occasionally nod yes/no or utter 1–2 words at his own will. He clenched his jaws when family attempted to feed him. Family googled KC's symptoms and guided by the suggestions over the internet, they decided to visit the ENT clinic for further evaluation.

Upon presentation to the clinic, KC appeared dishevelled and had clearly lost weight, with his clothes hanging loosely. He refused cooperation for the oral and laryngoscopic examination. Neurological examination revealed increased rigidity in both limbs following which a psychiatry referral was done and a provisional diagnosis of catatonia was established.

After a lorazepam challenge, the rigidity reduced, and KC became more compliant, accepting small amounts of food. The laryngoscopy was performed when KC was cooperative, revealing no structural abnormalities or any pathological finding. His speech returned, and he expressed fear, saying, "I can hear people plotting against me". With a diagnosis of psychosis, oral risperidone 4 mg per day (divided dose) was started. Routine blood tests revealed nutritional deficiencies likely due to prolonged food refusal. Two weeks into the treatment, KC resumed normal interaction, and his speech was fully restored.

Results: The case highlights the collaborative approach of ENT and mental health professionals in prompt identification and treatment of aphonia. Unlike many patients who present to ENT clinics with aphonia due to stress-related causes, this case is unique because psychotic symptoms overlapped with the aphonic presentation.

Conclusion: This case emphasizes the importance of a multidisciplinary approach in the diagnosis and management of aphonia. It serves as a reminder to consider a broader differential diagnosis when encountering aphonia, particularly in patients with unusual behavioural changes or neurological signs.

Abstracts were reviewed by the RCPsych Academic Faculty rather than by the standard *BJPsych Open* peer review process and should not be quoted as peer-reviewed by *BJPsych Open* in any subsequent publication.

Kaleidoscope in the Mind: Charles Bonnet Syndrome

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Aims: Charles Bonnet syndrome (CBS) is a phenomenon characterized by complex visual hallucinations in visually challenged patients. CBS may go unrecognized and be misdiagnosed as early dementia or psychosis.

Methods: We present the case of an 83-year-old Caucasian gentleman 'X' who was referred to the older adult home treatment team.

X reported that his problems began when some men turned up at his door a few weeks earlier, regarding an application he had made to the council for solar panels. X suffered from a complex delusional framework, believing that these men have stayed around in his home and are living in his loft. He believed that there are microphones in his bedroom and that his conversations are being transmitted to malicious people who work for the council.

X reported distressing visual hallucinations of men in his kitchen threatening him with guns. Other hallucinations included his ex-partner engaging in sexual activities with the aforementioned men, which led him to believe that she has been forced into the sex trade. He reported seeing distorted faces in his bathroom mirror, bodies floating up against the ceiling, and children with metallic prostheses for limbs. X recently received a diagnosis of a mild cognitive impairment following a referral made by the GP to the memory clinic. Prior to this, he did not have a history of psychiatric or neurological illness.

Interestingly, X had previously undergone excision of his right eyeball for the treatment of cancer, and has a skin graft at the site. He has only partial vision in his remaining eye due to macular degeneration.

Results: Three criteria should be fulfilled to diagnose CBS; namely, visual loss, clearly formed recurrent visual hallucinations, and insight into the unreal nature of the hallucinations.

In our case study, X fulfilled the first 2 criteria, but was unable to appreciate that the images that he sees are not real. We surmise that X may initially have understood that his hallucinations are not real, but these hallucinations gradually became amalgamated with his delusional belief systems, leading to a loss of insight.

Conclusion: CBS should be considered as a diagnostic possibility in persons with visual impairment presenting with visual hallucinations, and differentiated from psychotic and neurological disorders such as dementia, delirium and late-onset psychosis.

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SLC6A1 Neurodevelopmental Disorder: A Case Report

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Aims: Solute Carrier Family 6 member 1 (SLC6A1) genetic mutations are rare and mostly reported in the paediatric population. Few reports on adult cases have been mentioned in the literature. At present only pathological and likely pathological variants can confirm a diagnosis of SLC6A1-related neurodevelopmental disorder.

Methods: We report the clinical presentation of an adult male with a heterozygous variant of uncertain significance of the SLC6A1 gene. He presented with developmental delay initially and only in later years presented with other features in keeping with SLC6A1-related neurodevelopmental disorder. This includes moderate intellectual disability, epilepsy, autism, attention deficit disorder, behavioural difficulties including aggression. A further EEG at age 14 also showed focal and general abnormalities, however an MRI was normal. He was trialled on methylphenidate, lis-dexamphetamine and atomoxetine which were all unsuccessful. In 2024, genetic testing revealed a heterozygous variant of uncertain significance of the SLC6A1 gene.

Results: Our case adds further credence to the growing literature on SLC6A1 gene-related disorder and SLC6A1-related neurodevelopmental disorder. This individual illustrates the diverse clinical phenotype. Given the lack of published evidence, the result from