



worsening of liver functions due to starvation hepatitis, while on admission for refeeding and her gradual recovery.

**Methods:** 'A' is a 34 year-old lady, who is known to specialist eating disorders team with long-standing history of anorexia nervosa, restrictive sub-type, in the background of coeliac disease. Body mass index (BMI) on admission was 11.4, and reported food intake till that point was less than 300 calories/day. Ward dietician started her on stage 2 refeeding menu – 750 calories, 25 g protein, 1350 ml fluid with appropriate thiamine, multivitamin and mineral cover. Liver function was mildly deranged (Alanine transaminase ALT 256 U/L, Gamma-glutamyl transferase activity GGT 38). 'A' struggled to eat on the ward, and over the next week deteriorated with LFT as follows – ALT 2362 U/L, AST 2288 U/L, GGT 88 U/L. Upon shifting to medical bed and failure of less restrictive options, 'A' was treated under the Mental Health Act with full nasogastric feeding with 1:1 supervision. Liver appeared normal on Ultrasound abdomen and serum electrolytes were mostly normal, ruling out refeeding hepatitis. Over the course of several weeks, as slowly BMI increased with improvement in nutrition, liver parameters improved with ALT dropping down to 346 U/L on day of transfer out of medical bed for psychological treatment.

**Results:** Starvation hepatitis, as in this case, appears when weight is at lowest with markedly elevated transaminases, normal liver appearance on radiological investigations. In this patient, BMI went to as low as 10 kg/m<sup>2</sup> and expectedly, LFT derangements worsened, and improved on gaining weight.

**Conclusion:** Though anorexia nervosa has a plethora of medical complications, it is important to anticipate hepatitis as an important complication, and be aware of potential differential diagnoses including starvation hepatitis and refeeding hepatitis, which needs to be analysed carefully to delineate, and treat appropriately.

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## “I Cannot See”; Inverse Anton’s Syndrome: A Case Study

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**Aims:** Abnormalities of vision have long been documented in psychosis. One syndrome of interest is Inverse Anton’s syndrome. This is a rare manifestation of visual abnormality where a person describes being blind despite objective evidence against this. In this case report, we discuss a patient who presents with a complaint of blindness despite evidence to the contrary.

**Methods:** Case Report.

A 46-year-old male part-time worker, with a childhood history of photosensitive myoclonic seizures which were treated with anti-epileptic medications. At 21 years, he required mental health services as he complained of episodic blindness. He was diagnosed with a delusional disorder. His symptoms resolved drastically after he was treated on olanzapine which he discontinued and remained well for over 2 decades.

He returned to services following a recurrence. His reported blindness is associated with emotional distress and self-harming. He is able to independently mobilise, complete forms and questionnaires but would insist that he is blind despite doing these. He reported mood changes which were treated with sertraline but with

no benefits. He was unsuccessfully treated on olanzapine and then switched to quetiapine.

Cognitive Behavioural Therapy based Initial Interventions were unsuccessfully attempted. He was reviewed by neuropsychiatry and complex psychosis service. He is engaged to Occupational Therapy interventions aimed at maximising practical skills. He was assessed by the ophthalmologist and opticians with no significant findings. He was referred to the neurologist. He had a brain Magnetic Resonance Imaging scan which found no abnormality.

**Results:** Discussion.

Inverse Anton’s syndrome is scantily described. There are few case reports with similar presentations. In a 2019 case report, the presentation was similar to this. The exact cause of this syndrome is unclear. It is thought to result from a structural disconnection of the parietal lobe attentional systems from visual perception. In the absence of radiological evidence, this leads to a suggestion of a functional illness or a neuropsychological syndrome in which visual perception and cognitive awareness are dissociated. In the absence of known secondary gain, it continues to present a diagnostic dilemma. This puzzle requires multidisciplinary efforts to solve. Management is focussed on secondary prevention and rehabilitation to improve quality of life.

**Conclusion:** Inverse Anton’s syndrome continues to present with unclear aetiology and diagnostic dilemma. It is hoped that multidisciplinary efforts together with advances in neuroimaging would help understand this syndrome better. We hope that our case report contributes to the body of knowledge and adds more perspective to this.

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## Unmasking Cognitive Decline: A Case Report on the Diagnostic Challenges in Language Variant Frontotemporal Dementia

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**Aims:** Language variant frontotemporal dementia (lvFTD) is a neurodegenerative disorder primarily affecting language, often presenting with speech and comprehension difficulties, commonly in people aged 45–65 years. lvFTD presents a perplexing diagnostic challenge, often masquerading as primary progressive aphasia (PPA) while progressively dismantling communication and cognition. Despite growing recognition of lvFTD, a critical gap remains in distinguishing its early presentation with overlapping neurodegenerative syndromes, delaying accurate diagnosis and intervention.

**Methods:** This case report highlights the challenges of diagnosing lvFTD in a patient with atypical early symptoms and the implications of late diagnosis on patient care and outcomes.

Patient in her late 50s, with background of anaemia and hypothyroidism presented with memory issues, word-finding difficulties, and trouble understanding conversations which began in early 50s, initially attributed to menopause. Symptoms worsened over time, revealing frontal lobe atrophy on brain imaging and a psychotic episode, including auditory hallucinations and paranoid delusions.

Following two self-harm attempts, detention under the Mental Health Act led to first mental health admission. Neurological investigations, including PET CT Brain, suggested a diagnosis of

potential Logopenic Primary Progressive Aphasia (lvPPA) with mixed dementia and Lewy Body dementia (LBD). Neurologist review confirmed diagnosis of lvFTD. Antipsychotic trial undertaken with aripiprazole, only to stop as led to worsening behavioural symptoms. Subsequently started on mirtazapine, quetiapine, lorazepam and rivastigmine. Improvement noticed in symptomatology. Currently awaiting DAT scan for further evaluation and on waitlist of Young Onset Dementia Psychology.

**Results:** This case underscores the complex diagnostic challenges in patients with overlapping neurodegenerative and psychiatric symptoms. The patient, in her late 50s, presented with progressive language impairment, memory issues, and psychotic features including auditory hallucinations and paranoid delusions. Neuroimaging revealed frontal lobe atrophy and significant asymmetrical hypometabolism in the left frontal, temporal and parietal lobes, findings suggestive of lvPPA. However, reduced tracer activity in the occipital cortices raised the possibility of mixed dementia, potentially co-existing with LBD. These overlapping features highlight the need for a comprehensive, multidisciplinary approach to refine diagnosis and optimize management strategies.

**Conclusion:** Breaking through the diagnostic fog, this case exposes the intricate challenge of untangling overlapping neurodegenerative and psychiatric disorders. The patient's progression from language deficits to memory loss and psychotic symptoms along with neuroimaging showing left hemispheric hypometabolism and frontal lobe atrophy, suggested lvPPA, potentially complicated by mixed dementia and probable LBD. She was diagnosed as lvFTD. This complexity calls for early multidisciplinary evaluation for prompt diagnosis and tailored intervention for improved patient outcomes.

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## Catatonia and Systemic Lupus Erythematosus – A Case Report

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**Aims:** Catatonia is a complex neuropsychiatric syndrome of disturbed psychomotor function, abnormal behaviours and withdrawal. It remains under-recognised and under-diagnosed, especially within the acute hospital setting.

While often associated with primary mental illness, it can also occur secondary to systemic medical conditions such as systemic lupus erythematosus (SLE), an autoimmune disease in which neuropsychiatric manifestations are commonly described.

We present a case which highlights the diagnostic challenge and importance of recognising catatonia in the context of lupus.

**Methods:** A 34-year-old female with a three-year history of SLE presented with decline in functioning accompanied by malar rash, joint pains, paucity of speech and altered mental state. She had previously experienced command hallucinations in the context of lupus flares and though the psychotic component resolved between episodes, she was prescribed a daily maintenance dose of olanzapine 2.5 mg.

Assessment revealed an agitated, distracted patient with suspected auditory and visual hallucinations, profound paucity of speech and incoherent mumbling. She required assistance with personal care, displayed rigid posturing, and had stopped eating and drinking. Laboratory results were consistent with an acute SLE flare, and it was

proposed that her presentation was SLE-related psychosis, initially addressed by increasing olanzapine dose with minimal effect.

Further clinical deterioration prompted a lumbar puncture after which the patient began to talk and regain some normal functioning. Thorough examination of notes revealed midazolam had been administered so it was proposed that this was catatonia and therefore partially resolved with a benzodiazepine. Further examination revealed waxy flexibility, catalepsy, stupor and mutism. Regular lorazepam was added to the schedule of cyclophosphamide and high-dose prednisolone and led to prompt substantial clinical improvement.

**Results:** Data suggests neuropsychiatric symptoms are common in SLE and though there are some reports in literature of lupus-associated catatonia, its precise prevalence is uncertain.

It is proposed that the diversity of symptoms can arise due to various pathophysiological mechanisms in lupus, which include autoimmune inflammation of the central nervous system, metabolic disturbances or adverse effects of medication. While treatment of the underlying cause is key, timely recognition of catatonia and pharmacological therapy can result in rapid clinical improvement.

**Conclusion:** Catatonia is associated with significant morbidity and mortality if untreated. It should be considered as a differential in patients with lupus, particularly those with concurrent neuropsychiatric symptoms, thus resulting in improved patient outcomes.

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## Ekbom Syndrome With Folie à Deux – an Examination of Nature vs Nurture Through a Case Study

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**Aims:** Delusional parasitosis (DP), or Ekbom syndrome, is a rare psychiatric disorder in which individuals falsely believe they are infested with parasites. When shared by another person, it is classified as folie à deux (shared psychotic disorder). This case study explores a unique DP case within a close relationship, examining clinical presentation, potential causes, and treatment outcomes.

**Methods:** A 64-year-old woman sought mental health services, convinced she had fatally infested her 26-year-old neurodivergent son with scabies and transferred her heart disease to him. She had believed for years that she had a scabies infestation, a delusion shared by her mother, who had recently passed away at 89. Multiple dermatology consultations ruled out any infestation, yet she continued self-treating with bleach, essential oils, borax, and horse skin infection chemicals. She also took excessive baths, scrubbing herself vigorously.

Additionally, she was convinced she had heart disease and past cancers, though no medical evidence supported these claims. She exhibited significant anxiety and distress but denied perceptual disturbances and lacked insight into her condition. Treatment was initiated with a combination of an antipsychotic and an antidepressant, leading to a gradual reduction in delusional intensity and increased engagement with mental health services. Psychological support was also provided.

**Results:** DP is challenging to treat, particularly when reinforced by family members, as seen in this case. The patient's condition worsened following her mother's death. However, a multidisciplinary approach is essential to enhance engagement and compliance, which are crucial for a positive prognosis.