

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