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.

Catatonia and Systemic Lupus Erythematosus – A Case Report

Dr Natalie Da Silva, Dr David Hall and Dr Tobias Rowland

Coventry & Warwickshire Partnership NHS Trust, Coventry, United Kingdom

doi: 10.1192/bjo.2025.10717

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.

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

Dr Aditi Dawar and Dr Shaimaa Aboelenien

EPUNFT, Rochford, United Kingdom

doi: 10.1192/bjo.2025.10718

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.

Conclusion: Understanding such cases is vital for accurate diagnosis and effective intervention, especially given the reinforcing nature of shared delusions and the persistent nature of DP.

A Case Series Highlighting Complexities in the Management of Bipolar Disorder With Parkinson's Disease

Dr Lawrence Dianga, Dr Mathew Madu, Dr Alana Durrant, Dr Leia Penfold and Ms Joanna Pepper

Norfolk and Suffolk Foundation Trust, Bury St Edmunds, United Kingdom

doi: 10.1192/bjo.2025.10719

Aims: Evidence is emerging regarding the association between Bipolar Affective Disorder (BPAD) and Parkinson's disease (PD). Studies have shown patients with BPAD have a higher risk of developing PD. This case series explores the complexities encountered in the management of patients with PD and BPAD.
Methods: 'A' is a woman in her 70s with BPAD. Parkinsonian symptoms were noted for several years, suspected to be medication-induced. She was diagnosed with Parkinson's disease, after a positive DaT scan, treated with co-beneldopa, which induced a manic episode, requiring hospitalization. Co-beneldopa was stopped, she improved with lamotrigine and clonazepam.

'B' is a woman in her 70s with BPAD with a family history of Parkinson's disease. She was hospitalized after relapse of BPAD. She was noted to have a unilateral tremor, stooped posture and a shuffling gait. She is now being assessed for Parkinson's disease.

'C' is a woman in her 60s with BPAD, on sodium valproate and aripiprazole. She was reviewed by neurology due to bilateral tremor, rigidity and unsteady gait, and subsequently diagnosed with drug-induced parkinsonism. Due to miscommunication, GP started her on co-beneldopa, for Parkinson's disease. Subsequently, she developed mania warranting hospitalization. The ward team was unaware of the misdiagnosis. Co-beneldopa was subsequently stopped. She continued having poor oral intake, intractable mania, treated with ECT.
Results: Literature review shows BPAD is associated with Parkinson's disease (PD). Evidence indicates, a diagnosis of BPAD, increases the risk of developing PD. With no established intervention for patients with co-morbid BPAD and PD, treatment becomes complex. Proposed pathology suggests BPAD is exacerbated by heightened dopamine levels, while PD from reduced dopamine. This makes it challenging to treat one without impacting the other. Mood stabilizers and antipsychotics can contribute to drug-induced parkinsonism (DIP), which may clinically be indistinguishable from PD. Antiparkinsonian medications like dopamine agonist and pramipexole can cause manic symptoms.
Conclusion: Historically, heterogeneity in psychiatric disorders, both in presentation and response, remains the norm. In this case series, we try to highlight the complex relationship between BPAD and PD. To establish a direct causal relationship is challenging due to the various confounders. This being a niche topic with limited research, emphasizes the need for large sample studies, which could shed more light on the longitudinal course and relationship between the two disorders and help establish future treatment guidelines.

Beyond Psychiatric Illness: Paediatric Autoimmune Neuropsychiatric Syndrome Presenting as Psychosis

Dr Akshaya Dixit[1] and Dr Saikat Roy[2]

[1]Central and Northwest London NHS Foundation Trust, London, United Kingdom and [2]West London NHS Trust, London, United Kingdom

doi: 10.1192/bjo.2025.10720

Aims: Paediatric Autoimmune Neuropsychiatry Syndrome is a rare condition with exact prevalence not known due to dearth of large-scale population studies. Its diagnostic as well as treatment guidelines are not yet validated. There are a few international guidelines but none available in the UK yet.
Methods: The referral of a 15-year-girl presenting with symptoms suggestive of PANS to our team prompted us to write this case report. The patient was brought in to the A&E by her parent after an episode of bizarre and chaotic behaviour at school, which included her throwing belongings at the staff while being emotionally labile. On assessment she reported symptoms suggestive of paranoid delusions, delusional perception, auditory and gustatory hallucinations, thought broadcasting and delusional memory. She was also taking more frequent showers, with clothes on. Her sleep was disrupted.

Extensive blood and radiological investigations initially didn't yield any positive findings. Antipsychotic (olanzapine) was initiated and the patient's mental state quickly improved. Two days later blood investigations showed raised antistreptolysin O titres (200–400 U/Ml). Hence, antibiotic (phenoxymethypenicillin) was initiated. The patient was discharged and followed by our team in the community.

The patient continued to present with residual psychotic symptoms which fluctuated in the community setting. But the overall functioning significantly improved.
Results: The acute onset of psychosis, characterized by behavioural disturbances, anxiety, sleep disruption, irritability, and emotional lability, along with elevated antistreptolysin O titres, suggested a diagnostic formulation aligning with PANS.
Conclusion: The case underscores the importance of considering PANS as a differential diagnosis in school-aged children presenting with symptoms of OCD, first episode of psychoses or unexplained emotional lability.