Opinion

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A Patient who Suffers from a Neuropsychological Disorder and May be Triggered by a New Drug to Have Bullous Pemphigoid: A Case Study and a Review of the Literature

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Description

A generalized, itchy, and blistering rash was presented by a 59-year-old woman with schizoaffective disorder. She had begun taking empagliflozin, a type-2 diabetes medication that blocks Sodium Glucose Transporter-2 (SGLT-2) she had been taking paliperidone, an atypical antipsychotic, for one year already. At the time of presentation, anti-pemphigoid antibodies were detected by serology. Eosinophils, perivascular inflammation, and subepidermal blistering were all found on histology. Bullous Pemphigoid (BP) had direct immunofluorescence, with linear IgG and C3 at the basement membrane. Paliperidone and empagliflozin were both discontinued. The blisters, however, persisted. Treatment consisted of: topical dermovate and eumovate ointments for the body and face, respectively, along with 200 mg of doxycycline and 40 mg of prednisolone taken orally for a week (with a 5 mg/week reduction over 8 weeks) However, new blisters continued to form, so dapsone 50 mg was administered, which resulted in significant improvement. Aetiology and treatment are made more difficult by the increasing association between BP and a number of neurological and mental health conditions. We do not fully comprehend the mechanism by which these associations are formed. Because the bullous pemphigoid autoantigens BP180 and BP230 are expressed in the central nervous system, it is possible that neurodegeneration exposes the antigens to the immune system and triggers a cross-reactive immune response. However, BP and neuropsychological conditions appear to be linked in both directions. Additionally, the fact that empagliflozin initiation was associated with BP onset further complicates the aetiology and suggests a novel drug cause of BP.

Neuropsychological Challenges

This case highlights the neuropsychological challenges of managing complex BP cases, a possible new cause of drug-induced BP, and suggests that these challenges will likely become more common in the future. Bullous Pemphigoid (BP) is a skin condition characterized by autoimmune blistering that typically affects older patients with multiple co-morbidities. The relationship between BP and neuropsychological co-morbidities appears to be bidirectional. Additionally, these connections appear to be associated with the neuropsychological condition-treating medication. According to one study, schizophrenia increased the likelihood of developing BP by 2.7 times. However, in order to fully investigate the disease's mechanism, additional research is required. The BP antigens BP180 and BP230 may be exposed by nervous system inflammation, triggering a cross-reactive immune response between neural and cutaneous antigens, according to one theory. Compared to general BP patients, BP patients with neuropsychiatric co-morbidities have worse outcomes, with higher mortality and lower compliance with long-term corticosteroid treatment. Additionally, high-dose corticosteroids are frequently used to treat high blood pressure, despite their reputation for altering mood. The aetiology, presentation, and treatment of BP may be affected by a patient's co-morbidities and medications, so clinicians should carefully consider these factors. Given the rise in polypharmacy, co-morbidities, neuropsychiatric diseases, and elderly populations, which will in the future necessitate more comprehensive care for individuals, this is likely to remain relevant. A 59-year-old woman with a history of schizoaffective disorder presented with a four-week rash that was itchy and blistery. This involved the mucosa and began at the neck and spread to the torso, hands, and soles. One month prior to the onset of her cutaneous symptoms, she had begun taking empagliflozin, a SGLT-2 inhibitor for type 2 diabetes. Atherosclerosis, hypertension, hypothyroidism, and agoraphobia were among her previous medical conditions. Monthly injections of paliperidone, adcalD3, alendronate, chlorpheniramine, citalopram, lansoprazole, lisinopril, levothyroxine, and lercanidipine were among her medications.She presented at the examination with extensive erosions affecting her entire body and mouth, as well as painful blisters on her hands and soles. Blisters on the larynx were seen during a nasoendoscopy. The presentation's serology revealed a positive result for anti-pemphigoid antibodies—interestingly, she had tested negative in 2018. Histology revealed eosinophils and a perivascular inflammatory infiltrate, as well as subepidermal blistering. The basement membrane displayed linear IgG and C3 in direct immunofluorescence. As a possible trigger, she was stopped taking empagliflozin and given

subcutaneous insulin by diabetic specialists. Dermovate ointment was applied topically to her trunk and limbs once a day under paste bandaging, and Eumovate ointment was applied topically to her face once a day. She began taking prednisolone 40 mg daily orally and doxycycline 200 mg daily orally for a week, decreasing by 5 mg per week for eight weeks. However, new blisters continued to appear, so dapsone 50 mg taken orally was added, which significantly improved the condition. She did not show up for virtual or face-to-face appointments after she was discharged, which may have been due in part to her history of mental health issues. She was admitted again to general medicine one year later due to concerns about methaemoglobinaemia and shortness of breath. Her admissions doctors held her dapsone, causing her arms to blister again. Because her methaemoglobin level was stable (1.3 percent), dermatology advised that methaemoglobinaemia was unlikely to be the cause of her shortness of breath. However, infection was more likely. She did well on antibiotics, and her saturations got better. Dapsone 50 mg daily was started again, and her blisters got better. Despite this, she was released; She was admitted again a week later with covid pneumonitis, of which she tragically passed away. The prevalence of BP is estimated to range from 2.5–42.8 cases per million people per year in Europe6 to 4.3 cases per 100,000 people per person year in the UK7, with a one-year mortality rate of 26.7%. Rates appear to be rising over time, particularly in those over 60 years old; With an ageing population, this is a trend that is likely to continue. In addition, there are known connections between BP and neuropsychological conditions, like ours, so a thorough history is essential. A population study conducted in Finland found that neurological co-morbidities such as Multiple Sclerosis (MS), epilepsy, cerebrovascular disorders, dementia, and Parkinson's disease all significantly raise the risk of developing high Blood Pressure (BP).1A co-morbidity of MS confers the highest risk (OR 5.9), with these patients acquiring BP on average 12 years earlier than BP patients who did not have MS. Patients with blistering conditions also showed a higher risk of developing psychiatric disorders (HR 1.30), after correcting for BP medications and diuretics. Additionally, several psychiatric diseases have also been associated with BP, but with generally lower risk ratios compared to neurological co-morbidities. This association appears to be bidirectional, with a meta-analysis study showing that the risk of developing MS in BP patients was 12.4 times higher than that in controls.8 Schizophrenia had an OR of 2.7, personality disorders had an OR of 2.2, and schizotypal and delusional disorders had an OR of 2.1. The aforementioned study also suggested that psychiatric diagnoses could occur 7–11 years before BP. A Danish study from 1994 to 2016 demonstrated that BP patients with pre-existing psychiatric disorders had a 79% increased risk of developing the condition. This study supported the finding that the highest risk was associated with intellectual disorders (4.18-fold), schizophrenia, and personality disorders (2.01-fold), independent of psychiatric medications.6 A more recent large population study demonstrated that BP patients with depression had an all-cause mortality rate that was 19% higher than that of those without depression. Long-term corticosteroid treatment compliance was also lower in BP patients who had a history of anxiety and depression.

Neurodegeneration

Uncertainty surrounds the mechanism by which BP and neuropsychiatric illness are linked. Subtypes of the autoantigen BP230 have been identified in both the central and peripheral nervous systems, and the consensus in the literature is that neuroinflammation and neurodegeneration could precipitate a cross-reactive immune response between neural and cutaneous antigens. Since skin and nervous tissue are derived from the same ectoderm, Although more research is required, there is evidence that neuropsychiatric illness may influence BP's immunological phenotype. In a retrospective study, a higher seropositivity rate of anti-BP230 was found in BP patients with co-morbid neuropsychiatric disease than in those without (67.7% *vs.* 36.5%). In addition, the circulating levels of eosinophils, which are thought to have neurotoxic effects, were higher in these patients than in general BP patients. Because the BP180 enzyme-linked immunosorbent assay demonstrated inverse associations with cognitive function, as measured by the scores on the mini mental state examination, another study found that BP180 autoantibodies are associated with more severe forms of dementia.