

neutrophil count (ANC) $< 0.50 \times 10^9$ per L. Benign ethnic neutropenia (BEN) is a condition found in members of African or Middle Eastern descent that is characterized by ANC $< 1.50 \times 10^9$ per L in the absence of other causes. Filgrastim is a granulocyte colony-stimulating factor (G-CSF) that has shown efficacy in reducing the duration of agranulocytosis in some patients who develop clozapine-induced agranulocytosis. It is currently unknown whether filgrastim is beneficial in the treatment of neutropenia due to BEN. We here, for first the time report a case of a patient with BEN who developed agranulocytosis both during the first clozapine trial for schizophrenia and during the rechallenge, despite early stabilization with filgrastim treatment, which highlights the failure of filgrastim in treating BEN.

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Treatment of Odor-Induced Anxiogenesis With Odor-Induced Anxiolysis

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ABSTRACT: Study Objective: To understand the effects of odor on anxiety.

INTRODUCTION: Reduction of odor-induced anxiety through a presentation of an odor has not heretofore been described.

METHOD: Case report: A 69-year-old right-handed male with a five year history of generalized anxiety disorder, presented with a one and a half month history of hypersensitivity to odors of multiple synthetic chemicals manifest by the perception that these odors were more intense and unpleasant inducing nausea, abdominal cramping, coughing, a need to “get away from the smell”, and panic with intense anxiety. These symptoms would occur whenever he was exposed to these smells, 20 to 25 times a day, and would persist for 10 to 15 minutes after the exposure. When odors induced the above symptoms, exposure to the aroma of cinnamon immediately alleviated these symptoms. He now continues using cinnamon odor whenever the odor induced anxiety and associated symptoms arise. This remedy has been effective over the course of treatment, for almost two years.

RESULTS: Abnormalities on examination: Three per second titubation. Archimedean Spiral Test: Saw tooth pattern with macrographia. Anxious, circumstantial, overly inclusive. Unable to determine how to put on shoe covers. Impaired voluntary upward gaze, but intact vertical doll's eyes. Left torticollis. Bilateral finger to nose dysmetria. Low amplitude, high frequency tremor on extension of both upper extremities. Areflexic. Olfactory Testing: hyposmic. MRI of brain with and without infusion: mild generalized volume loss.

CONCLUSIONS: There are myriad mechanisms whereby odor may have reduced the odor-induced anxiety. Since aroma induced anxiogenesis is usually confined to a specific odor, it does not preclude other odors from acting in an anxiolytic manner. The combination of exposure simultaneously of anxiolytic and anxiogenic odors may have acted to increase the threshold of the anxiety producing odor, inhibiting perception of the anxiogenic odor and thus precipitation of anxiety. The two odors could have combined in an additive fashion, changing the olfactory characteristics of the anxiety provoking odor such that it no longer was perceived as the same odor and thus no anxiety. The anxiolytic/anxiogenic odor mixture could have overwhelmed the anxiogenic odor, thus creating the perception of only anxiolytic odor. On a central basis, the anxiolysis and anxiogenesis may have been induced to occur coincidentally with anxiolysis superseding anxiogenesis. Alternatively, the odors may have acted as a distractor, changing the focus of attention from anxiogenic odor to a different odor which does not have the same anxiety provoking effect. Maybe because the patient already has demonstrated a heightened odor emotion linkage, he may be more susceptible to any other odor emotion effects. Trial of odors in those with odor induced anxiety warrants consideration.

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Prospective Evaluation of the Economic Utility of Combinatorial Pharmacogenomics in Generalized Anxiety Disorder and Major Depressive Disorder

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ABSTRACT: Mental illness is one of the leading causes of disability, with direct and indirect costs posing a significant financial burden. Previously, a large prospective economic utility study (n > 13,000) showed that the GeneSight® test, a psychiatric pharmacogenomic

decision support tool powered by CPGx® technology, reduced medication costs, increased adherence, and reduced polypharmacy for patients who had failed monotherapy for psychiatric disorders. The current study, which is a sub-analysis of this larger study, assessed cost savings associated with combinatorial pharmacogenomic testing in patients with generalized anxiety disorder (GAD) and major depressive disorder (MDD). Medication costs were extracted using pharmacy claims data provided by Medco, a large pharmacy benefits manager, for patients with GAD (n = 318) and MDD (n = 459). Medication cost savings per member per year (PMPY) for 1 year following the test were compared between patients whose medication regimens were congruent with the test recommendations and those whose medication regimens were incongruent with these recommendations. When healthcare providers' decisions were congruent with combinatorial pharmacogenomic testing, PMPY savings was \$6,747 (p < 0.004) for GAD patients and \$3,738 (p < 0.004) for MDD patients versus incongruent decisions within these disease states. Among the congruent group, GAD patients experienced greater savings in central nervous system (CNS) medications (2-fold) compared to MDD patients. Additionally, analysis of a subset of patients prescribed at least one benzodiazepine six months prior to testing (n = 660) demonstrated a significant decrease in benzodiazepine drug counts (p < 0.001) and refills (p < 0.001) after testing. Using the GeneSight test as a treatment decision support tool for patients with GAD or MDD resulted in significant medication cost savings when HCPs made congruent decisions with the combinatorial pharmacogenomic results. Furthermore, use of the GeneSight test decreased the use of benzodiazepines.

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Pseudogout Induced by Vortioxetine

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ABSTRACT: Study Objective: Vortioxetine induced monoarticular pain has not heretofore been described. Such a case is presented.

METHOD: Case study: A 49 year old right handed female with a past history of multiple hospitalizations for chronic severe migraine, presented with complaints

of depression and stress. She has had depression for 20 years, which has been constant and worsened in the past 5 years. As her migraines became more frequent, her depression also worsened. She has never been suicidal, but does endorse sadness, crying spells, fatigue, demotivation, lack of interest, poor concentration, irritability, anger, guilt, hopelessness, helplessness, anorexia, PM insomnia with frequent awakenings, absent libido and racing thoughts.

RESULTS: Abnormalities in her mental status examination: Orientated times 2. Disheveled. Defensive. Motor retardation. Mood: depressed, anxious and irritable with blunted affect. Remote memory: President: Obama,?. Beck Depression Inventory II: 23 (moderate depression). Beck Anxiety Inventory: 25 (moderate anxiety).

The patient was begun on 5 mg of vortioxetine every night. Within two days, she developed pain and swelling of the distal interphalangeal joint of the left great toe. The pain was so severe that she demonstrated an antalgic gait. After five days the medication was discontinued and two days later, there was full resolution of the swelling and pain, and ambulation returned to normal.

CONCLUSIONS: The mechanism whereby vortioxetine induced this monoarticular pain is unclear. Underlying depression alone can precipitate arthritic exacerbation (Trivedi, 2004). This was unlikely given the long duration of her depression as well as the timing of the precipitant (vortioxetine use) and resolution shortly after the medication was discontinued. Alternatively, in the depressed state, there may be a greater perception of somatic pain, which allowed her to appreciate any arthritic pain which may have pre-existed the use of vortioxetine (Howard, 1991). As such, this may have represented a correspondence bias (Gilbert, 1995). Furthermore, mild new pain is perceived as more intense in those who are depressed (Howard, 1991). Thus, any minimal arthritic injury may be viewed as more intense. Vortioxetine may have paradoxically exacerbated anxiety and anxiety can precipitate pain (Narasimhan & Campbell). Alternatively, vortioxetine could have caused a generalized allergic reaction, which may have initially manifest in the great toe. If the patient continued the medication, she may have developed a generalized systemic reaction including involvement of multiple joints. Another possibility is that it caused an allergic histamine mediated hive like reaction, generalized, as well as on the toe. Continued use of the joint may have caused this to be intensified, with associated swelling, while the general reaction subsided. Inquiry about monoarticular involvement in those taking vortioxetine is warranted.

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