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Case Report

Pharmacogenetic application in a patient diagnosed with Schizophrenia and OCD: A case report

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Abstract

Introduction: The efficiency of psychiatric therapy depends on various factors and drug metabolism is one of them. The liver plays a significant role in drug metabolism through the P450 enzyme systems. However, pharmacogenetics aims to assist clinicians in determining the effectiveness of a particular drug and minimizing potential side effects.

Case report: Here, we present a case to demonstrate the potential use of pharmacogenetics in clinical practice. A 33-year-old male patient with a diagnosis of schizophrenia and OCD had symptoms of sexual auditory hallucinations and obsessive images. According to the patient's pharmacogenetic profile (*CYP2D6, CYP2C9, CYP2C19, CYP1A2* and *CYP3A4*), the medication started by giving Risperidone 8 mg/day and Aripiprazole 5 mg/day. Aripiprazole was continued at 10 mg/day and then 20 mg/day in the presence of clinical findings. Olanzapine 10 mg/day and Sertraline 50 mg/day were given. In addition, 20 sessions of TMS and 20 sessions of tDCS, which are neuromodulation treatments, were applied.

Results: The patient in question experienced some improvement and partial remission following a treatment plan based on pharmacogenetic analysis. In summary, pharmacogenetic testing can be a valuable tool in determining an appropriate treatment plan that maximizes clinical improvement while minimizing potential adverse effects associated with medication.

Introduction

Schizophrenia is a persistent and incapacitating psychiatric condition that impacts around 1% of people globally. Schizophrenia is a syndrome that includes a combination of symptoms, which is not yet fully understood. It is mainly characterized by observable signs of psychosis. The most common symptoms of schizophrenia are paranoid delusions and hearing things that are not there, which usually appear during late adolescence or early adulthood [1]. Less than 14% of individuals who experienced a psychotic episode showed consistent improvement in the five years that followed [2]. The results over a longer period of time may show slight improvement. A study that followed patients for 25 years across multiple countries found that there was a further 16% increase in recovery during the later stages [3]. Bleuler, a prominent psychiatrist in the early 1900s, was the first to use the term "schizophrenia." While he wasn't certain about the long-term prognosis of the disease, he highlighted the fundamental features of thought and emotion disorders, such as difficulties with thinking clearly and expressing emotions, indecisiveness and social withdrawal. These features are still

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used today in the diagnosis and treatment of schizophrenia. These early formulations, which arose before the split between neurology and psychiatry, were consistent with the concept of a mental disorder resulting from brain pathology. But with the dominance of psychoanalytic theory, the "brain" was ignored for much of the 20th century [4]. In the second half of the 20th century, with the advent of neuroleptic drugs, the focus was on brain chemistry. Schizophrenia has been recognized as a "dopamine disorder" based on the psychotic effects of dopamine-releasing drugs such as amphetamines and the antipsychotic efficacy of many drugs that block the dopamine D2 receptor [5]. The drugs that emerged thanks to this neurochemical aspect of schizophrenia have shown that patients can be treated outside the hospital and in some cases, the main symptoms of the disease can be alleviated. Early neuroleptic drugs, such as chlorpromazine and haloperidol, have been replaced by "atypical" antipsychotics, which have fewer extrapyramidal side effects but generally appear to be significantly less effective than the original dopamine D2 receptor antagonists [6]. Despite over 300 studies conducted on gene expression in schizophrenia in the last 15 years, no consistent evidence has been found for specific genes that contribute to the risk of developing schizophrenia. Schizophrenia is a severe and complicated mental disorder that is characterized by a combination of positive symptoms, negative symptoms, and cognitive dysfunction. It has a high level of inheritance and a multifactorial etiology, making it a complex polygenic genetic trait [7]. While there are many genetic variations associated with schizophrenia, understanding the biological effects of these variations is crucial for preventing symptoms and developing effective treatments [8].

Obsessive-Compulsive Disorder (OCD) is a prevalent psychiatric condition that affects about 2% of the global population [9]. The onset of OCD is commonly reported in childhood or adolescence, with boys showing a higher incidence of early onset. However, the frequency of the disorder in males and females is similar in adolescents and adults. OCD symptoms are diverse and exhibit significant clinical heterogeneity, leading to discussions of distinct clinical subtypes that may stem from varied neurobiological underpinnings [10]. These subtypes could complicate the interpretation of genetic findings [11]. Despite numerous clinical and demographic factors that are believed to affect treatment response, a satisfactory algorithm has yet to be developed to help clinicians determine which patient will respond optimally to a particular drug. There is a high likelihood that specific gene variants, such as Single Nucleotide Polymorphisms (SNPs), may influence drug outcomes in OCD treatment [12]. Pharmacogenetics (PGx) is a powerful tool that leverages individual patient genomic data to guide clinicians in selecting the most appropriate drug or dosage for a patient [13]. Information obtained from PGx testing and Therapeutic Drug Monitoring (TDM) can help clinicians anticipate the risk of adverse drug reactions, select the right drug, and determine the appropriate dosage for a particular patient, ultimately enhancing treatment outcomes and facilitating personalized treatment planning [14].

OCD and schizophrenia can be affected by both genetic and environmental factors. Many studies have been conducted on the use of genetic testing of cytochrome enzymes before initiating psychotropic drug therapy. Some studies advocate performing this test before clinical treatment [15,16].

In this case report, we describe the pharmacogenetic testing approach in a patient with schizophrenia and OCD who was hospitalized with a complex history.

Case report

A 33-year-old male patient was hospitalized for 1 month in Psychiatry Clinic, NP Istanbul Brain Hospital (Istanbul, Turkey), with symptoms of sexual auditory hallucinations and obsessive images. His family had no kind of genetic or psychological history at that time. His anxiety and worries started in high school after a normal childhood. His school performance had declined and by time obsessions about being successful and cleaning and washing hands started. Psychiatric treatment was started for the first time at the age of 17 years - 18 years. During this period, he had always the idea that someone was following him. He has benefited from the first treatment, but although his skepticism continued, he could continue his daily activities. Auditory delusions and sexual images started 1.5 years before hospitalization, serious illnesses of his parents may have affected him as a stressor. During this period, drug treatments were increased and ECT (Electro-Convulsive Therapy) was applied. After the treatments, sexual images increased and auditory delusions decreased but did not disappear.

After the initial examinations in NP Istanbul Brain Hospital, he was diagnosed with Schizophrenia and OCD. To optimize the medication, and also depending on the previous medication reports, FGx genotyping test was applied to him after obtaining signed informed consent. The peripheral blood sample was used for DNA isolation (Genomic DNA İsolation Kit, Thermo Fisher Scientific, Waltham, USA). SNP detection in relevant polymorphic regions was analyzed using specific probes with Real-Time PCR (Thermo Fisher Scientific, QuantStudio 3 Real-Time PCR, USA). The obtained alleles were evaluated by comparing them with the genome data of healthy individuals. Results were summarized in Table 1.

Polymorphisms examined in genetic analysis;

CYP2D6: *2, *3,*4,*6,*7,*8,*9,*10*,*12,*14,*17,*41, xN (Copy Number),

CYP2C9: *2,*3,*4,*5,*6,*11,

Table 1: PGx panel results of the patient.

GENE	Genotype	PREDICTIVE PHENOTYPE
CYP2D6	*4/*41 (xN)	Normal Metabolizer (EM)
CYP2C9	*1/*1	Normal Metabolizer (EM)
CYP2C19	*1/*1	Normal Metabolizer (EM)
CYP3A4	*1/*22	Intermediate Metabolizer (IM)
CYP1A2	*1A/*1F	Increased Induction

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CYP2C19: *2,*3,*4,*5,*6,*7,*8,*9,*10,*17,

CYP1A2: 1A, 1F, 1C

CYP3A4: *1,*1B,*2,*3,*12,*17*22

Pharmacogenetic analysis results

Due to the results of PGx, he was given Risperidone 8 mg/day, Aripiprazole 20 mg/day, Olanzapine 5 mg/day, and Sertraline 50 mg/day. Aripiprazole treatment was started at 5 mg/day, then the dose was updated to 10, 15 and 20 mg/day. 20 mg/day was continued as a treatment dose. In addition, 20 sessions of TMS and 20 sessions of tDCS, which are neuromodulation treatments, were applied. The patient partially experienced benefits from the treatment and partial clinical remission.

Discussion

In this case report, we describe a patient with the *1/*22 genotype of the *CYP3A4* gene, which is predicted to have reduced enzyme activity due to the PGx analysis. The goal of PGx testing is to have information on a patient's genetic profile regarding a drug's efficacy, guide drug dosing and reduced adverse reactions. Especially in psychiatry, PGx testing is widely used to predict the patient's response to certain medications. Although there are more functional polymorphisms in the genes metabolizing the widely used drugs; those analyzed SNPs give very important and informative information [17].

The following case report discusses a patient with a CYP3A4 gene *1/*22 genotype, which can cause a decrease in enzyme activity as per the PGx analysis. The relevant cytochrome P450 enzymes for antipsychotics include CYP2D6, CYP1A2, and CYP3A4/5. Genetic variations in these genes can impact the drug's plasma levels, with CYP2D6 responsible for 40% of antipsychotic metabolism, CYP3A4 for 23% and CYP1A2 for 18% [18]. Certain antipsychotics like haloperidol, risperidone, olanzapine, and aripiprazole rely on CYP2D6 for metabolism, while clozapine and olanzapine rely on CYP1A2. Additionally, haloperidol, clozapine, quetiapine, ziprasidone and aripiprazole rely on CYP3A4 for metabolism [19,20]. The patients' PGx results revealed that the patient had *CYP2D6* (*4/*41 XN), *CYP2C9* (*1/*1) and *CYP2C19* (*1/*1) genotypes; which is predicted as a normal activity, and *CYP3A4* (*1/*22) as a reduced enzymatic activity.

Olanzapine can block the activity of certain enzymes in the liver, including CYP2D6, CYP2C9, CYP2C19 and CYP3A4. These enzymes are responsible for breaking down olanzapine and other drugs in the body. When they are inhibited, olanzapine can build up in the body and cause more side effects. This means that patients taking olanzapine should be monitored for any potential adverse effects, especially if they are also taking other drugs that are metabolized by these enzymes [21]. The *CYP1A2*1F* polymorphism has been reported to affect the inducibility of *CYP1A2*. The *CYP1A2*1F/*1F* genotype alone resulted in a 22% reduction in olanzapine serum concentrations normalized for dose-/body weight compared to *CYP1A2*1A* carriers (both without induction) [22]. The fact that the patient was a normal metabolizer (EM) for CYP2D6, CYP2C9 and CYP2C9 but had decreased activity due to being a CYP3A4 Intermediate Metabolizer (IM) necessitated the preference for low doses in the use of "Olanzapine". However, as our patient was associated with increased induction (*1A/*1F) in terms of *CYP1A2*, olanzapine was discontinued after treatment.

Sertraline is transformed into N-desmethyl sertraline during metabolism, which doesn't have any clinical effects. The process of N-demethylation of sertraline involves at least five different cytochrome P450 (CYP) isoforms: CYP2B6, CYP2C9, CYP2C19, CYP2D6, and CYP3A4 [23]. *In vivo* studies suggest that CYP2C19 is the primary enzyme involved in this process [24]. Previous research indicates that the CYP3A422 allele reduces CYP3A4 protein expression levels and activity [25]. Since CYP3A4 is responsible for metabolizing 9% of sertraline [26], this genetic polymorphism can affect the metabolism of the drug and its efficacy in treating the disease. Additionally, the CYP3A422 allele can impact the pharmacokinetics of other drugs like tacrolimus, cyclosporine and statins. Given that our patient had the CYP3A4*22 allele, we opted to prescribe lower doses of sertraline [26].

Aripiprazole, a commonly used antipsychotic medication, is broken down and eliminated from the body primarily by two enzymes called CYP2D6 and CYP3A4. The recommended dose of aripiprazole is reduced by 50% for individuals with a genetic variation that affects CYP2D6 function. In addition, for people with this genetic variation who are also taking medications that strongly inhibit CYP3A4, the dose of aripiprazole should be reduced even further, to one-quarter of the usual döşe [27]. CYP3A4 is involved in the breakdown of more than half of all prescription drugs. Recent research has found that a specific genetic variation called CYP3A4*22 is linked to lower levels of the CYP3A4 enzyme, which may impact the breakdown and effectiveness of certain medications, including aripiprazole, haloperidol, pimozide and risperidone [28]. Based on the patient's genetic testing results, they were initially given the standard dose of aripiprazole. However, their dose was later adjusted based on the pharmacogenetic information.

Risperidone is an atypical antipsychotic drug commonly used to treat psychotic disorders like schizophrenia. In the liver, it undergoes extensive metabolism to 9-hydroxyrisperidone, which is an active metabolite that contributes to its therapeutic effects [29-31]. This metabolic process is mainly carried out by two cytochrome P450 enzymes, CYP2D6 and CYP3A4/3A5. The effectiveness of risperidone is thought to be linked to the levels of the active moiety (a combination of risperidone and 9-hydroxyrisperidone) present in plasma, with 9-hydroxyrisperidone being the primary component. Various studies, both *in vitro* and *in vivo*, have suggested that CYP2D6 is the primary enzyme involved in metabolizing risperidone [32]. Since our patient was a normal metabolizer with the CYP2D6 predictive phenotype, we followed a standard treatment protocol for risperidone.

To sum up, having knowledge about how second-generation antipsychotics are metabolized by the cytochrome P450 system can guide clinicians in avoiding and managing drug interactions related to these enzymes. Furthermore,

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incorporating pharmacogenetic analyses can assist healthcare providers in providing personalized treatment plans based on an individual's genetic makeup and how it impacts the drug's effects and metabolism.

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