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Pharmacogenomic Profiling for Personalized Treatment of Major Depressive Disorder

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ABSTRACT

Depression is the leading mental health issue, major depressive disorder (MDD) is diagnosed in more than 264 million people worlds over and there is a significant heterogeneity in the response of individuals to antidepressant treatment. The use of traditional approaches tends to be based on a trial-and-error approach, resulting in a long-term suffering process, high costs of healthcare avenues, and the risk of suicide. The study examines how a pharmacogenomic profile could be used in individualizing antidepressants among patients with MDD. Based on a cohort sample of 150 patients with MDD, genotypic differences in CYP2D6, CYP2C19, SLC6A4 and HTR2A were measured in order to establish a relationship between their drug metabolism and treatment reaction. The result of our analysis shows a considerable statistically significant association between genotype pattern and clinical outcomes, especially the SSRIs and tricyclic antidepressants. The paper has come to the conclusion that pharmacogenomic-guided therapy offers massive improvements in the effectiveness of treatment, the reduction of bad effects, and can transform the psychiatric treatment approach. These findings justify the introduction of pharmacogenomic screening practice into routine psychiatric care to maximize antidepressant treatment and enhance the quality of life of MDD patients.

Key Words:

Major Depressive Disorder (MDD), Pharmacogenomics, Antidepressant Therapy, Personalized Medicine, CYP2D6, CYP2C19

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1. INTRODUCTION

The 21st century is a period marked by depression which, according to the research, remains amongst most serious public health issues in all age groups, cultures, and socioeconomic backgrounds[1]. Major Depressive Disorder (MDD), the most frequent and bulky depression, is not only emotionally and physically disabling but also introduces a devastating impact on the society in terms of economics[2]. While pharmacotherapy has developed significantly, up to now a significant percentage of patients do not respond to such therapies and endure long lasting symptoms with compromised quality of living[3]. In this regard, the employment of personalized medicine in the provision of psychiatric treatment, in the form of pharmacogenomic profiling, provides a revolutionary solution to enhancing the success of interventions[4]. Pharmacogenomics has the potential to eliminate the trial-and-error method of prescribing that

was previously standard practice and replace it with precision-based decisions through pharmacological intervention that considers a specific genetic constitution of an individual[5].

1.1 Background Information

The Major Depressive Disorder (MDD) ranks as the worldwide main problem that causes disability and is characterized by such symptoms as an ongoing sad mood, the impossibility of finding pleasure (anhedonia), and disturbed thinking[6]. Even though antidepressants are extensively used (SSRI, SNRI, and TCA), the response to treatment is heterozygous, and only a third of the patients eventually enter into remission[7]. This variability lies within environmental, physiological and more so genetic factors. Pharmacogenomics--the study of the impact of genes on drug response provides an encouraging means through which antidepressant drug treatment could be personalized[8]. Variants of the enzymes CYP2D6 and CYP2C19, and of serotonin-related genes, including SLC6A4 and HTR2A, can be used to predict the success of treatment and its side effects. The adoption of phamacogenomic testing of clinical practice can improve treatment of MDD[9].

1.2 Statement of the Problem

MDD is a problem and still has challenges in achieving optimal treatment outcomes notwithstanding decades of research activities and the presence of various classes of medications in the form of antidepressants. One of the greatest weaknesses of modern psychiatry is that it depends on the non-individualized, generalized method of treatment that does not consider individual genetic peculiarities. This makes patients experience several consecutive weak rounds of treatment, with the result of time wasting, risk of various negative reactions, and non-compliant medication. The low success of treatment is part of the reason that leads to chronic disease courses, suicide risk, and expenses on society.

1.3 Objectives of the Study

- To identify the association between specific genetic variants—particularly *CYP2D6*, *CYP2C19*, *SLC6A4*, and *HTR2A*—and response to antidepressant medications.
- To evaluate whether pharmacogenomic-guided therapy results in improved clinical outcomes, including symptom reduction and reduced adverse effects, compared to conventional treatment approaches.
- To propose a practical framework for the integration of pharmacogenomic testing into psychiatric treatment protocols for MDD.

1.4 Hypotheses

To guide the investigation, the study proposes the following hypotheses:

- H1: Genetic polymorphisms in drug-metabolizing enzymes such as *CYP2D6* and *CYP2C19* significantly influence the efficacy and safety of antidepressant therapy in MDD patients.

- H2: Patients receiving pharmacogenomically guided antidepressant therapy will exhibit significantly improved clinical outcomes compared to those receiving standard, non-guided treatment.

2. METHODOLOGY

The research question of this study was to examine the usefulness of the application of pharmacogenomic derived antidepressant therapy to enhance clinical outcomes of patients with diagnosis of Major Depressive Disorder (MDD). Using a quasi-experimental design, the study examined the effect on symptom reduction and adverse effect profiles associated with including genetic profiling in treatment decisions as opposed to normal treatment. The subsequent paragraphs elaborate the design framework and the participants of the study, the methodology of the data collection, and analytical approaches used to evaluate the effect of personalized pharmacogenomic interventions.

2.1 Research Design

This paper employed a quasi-experimental, comparative cohort study to examine how pharmacogenomic-guided antidepressant treatment influences the outcome of clinical trials in terms of patient diagnosed with Major Depressive Disorder (MDD). Researchers used the randomized assignment of the participants to create two groups of equal size; one group is called an intervention group in which researchers prescribed the clinical treatment depending on this genetic profile, and the other group is called a control group; they will receive a standard antidepressant therapy according to, the available current clinical guidelines.

2.2 Sample Details

The study included 150 adults selected to participate in the study (aged between 18 and 65 years), who all met the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) definition of a Major Depressive Disorder (MDD). The recruitment was done in two tertiary centres of psychiatric care in New Delhi and Bengaluru, India. Inclusion criteria included having a baseline score of 18 or more on the Hamilton Depression Rating Scale (HAM-D), a measure of moderate to severe depressive symptoms, in order to create a clinical study group with a relatively benign condition and control confounding factors. People who presented with co-morbid psychotic disorders, substance use disorders, or had significant neurological impairment were not included in the study. The members were placed uniformly and not randomly into two of the 75 groups: an intervention group where the antidepressant treatment was done with the help of pharmacogenomic profile and a control group where the participants were offered standard treatment of antidepressants which was conducted based on traditional clinical evaluation and reasoning.

2.3 Procedure and Data Collection Methods

When the participants were enrolled, they were introduced to the first clinical testing, with HAM-D test taken and a psychiatric history established. The subjects had their buccal swabs obtained in sterile kits, and shipped to the laboratory to be segregated at genomic level.

The pharmacogenomic panel was targeting four major genes that are identified to affect antidepressant response as well as metabolism:

- CYP2D6 and CYP2C19 They are enzymes that mediate hepatic SSRIs and TCAs metabolism; classifications were done by classifying the participants as poor, intermediate, extensive, or ultra-rapid metabolizers.
- SLC6A4 (5-HTTLPR variant): serotonin transporter gene polymorphism that alters the response to SSRI.
- HTR2A (rs7997012 variant): Serotonin receptor gene implicated with developing treatment-emergent side cues and response fluctuation.

In the intervention group, the result of genotyping was sent to the prescribing psychiatrists who could make medication decisions based on metabolic pattern and estimated medication response. Poor metabolizers such as those, were prescribed in small amounts or other agents with favourable metabolic factors.

2.4 Data Analysis Techniques

The data were coded and entered into the IBM SPSS Statistics version 26.0 on Windows. The analyses were done in the following way:

- Descriptive Statistics: This will be utilized to provide summaries on the demographic information, genotype frequencies, as well as the baseline clinical characteristics.
- Independent-Sample t-Tests and ANOVA: This was used in comparing reduction in the HAM-D scores between the control group and the intervention group.
- Paired t-Tests: Two tailed paired t-test used to compare pre and post treatment within a group.
- Logistic Regression Analysis: To estimate the predictive value of single genetic markers (ex. CYP2D6, SLC6A4) of the treatment response (at least a 50 percent cut in HAM-D score).
- Chi-Square Tests: Chi-Square tests have been used to estimate the relationship between categorical values like the genotype as a categorical value and adverse events.

All the statistical tests done were two-tailed and values less than 0.05 are regarded as statistically significant.

3. RESULTS

The result of the clinical trial to test the tolerability and the clinical efficacy of guidance of antidepressant drugs based on the genetic makeup of the patients with Major Depressive Disorder (MDD) is displayed. The findings are presented under four important subsections as follows: (1) demographic and baseline clinical features, (2) genotypic distribution and clinical implications, (3) symptom trend throughout the 12 weeks duration of treatment and (4) treatment-emergent adverse effects. The results are reflected in the use of a series of statistical tables as well as graphical presentation which reveals a serious distinction in the treatment

response and safety between the patients undergoing pharmacogenomic-guided therapy and regular treatment.

3.1 Demographic and Baseline Characteristics

In order to ascertain soundness of comparison of treatments, demographic as well as baseline clinical features of all study participants were considered before the intervention. To facilitate measuring the possible confounding variables to a minimum and isolate the impact of the phg-based treatment choice on treatment outcomes, it is crucial to ensure the comparability of the obtained baseline characteristics of the two groups. Table 1 shows how the demographic and clinical characteristics of the study participants are in the two groups.

Table 1: Demographic and Baseline Clinical Characteristics of Study Participants

Characteristic	**Pharmacogenomic Group (n = 75)**	**Standard Group (n = 75)**	**p-value**
Mean Age (years)	38.4 ± 9.7	38.8 ± 10.6	0.76
Female (%)	61.3%	58.7%	0.71
Mean Baseline HAM-D Score	24.1 ± 3.4	23.9 ± 3.5	0.58
Duration of Illness (months)	18.2 ± 7.9	17.5 ± 8.1	0.48

As received in Table 1 results indicate, there were no significant differences between the pharmacogenomic group and standard treatment group based on the age, sex ratio, baseline severity of depression, or length of disease. This points to the conclusion that there was a good matching of the groups at the baseline stage thus enhancing internal validity of the outcome comparisons that were to follow. The almost identical baseline HAM-D scores (24.1 vs. 23.9) confirm the suggestion that the two groups entered the study with similar statuses regarding the level of depressive symptomatology.

3.2 Genotypic Distribution and Clinical Correlation

This is the section of distribution of clinically significant bearing gene polymorphisms identified by pharmacogenomic profiling of study participants. Determining the prevalence of such polymorphism in the sample population would enable the contextualization of the clinical implication of having a pharmacogenomic-based treatment in the Major Depressive Disorder (MDD). Table 2 has an overview of the frequencies of the major gene variations and their impacts on metabolism or clinical conditions.

Table 2: Genotype Frequencies and Predicted Metabolic Phenotypes

Gene	**Polymorphism**	**Frequency (%)**	**Metabolic/Clinical Effect**

CYP2D6	*4, *10	38%	Poor metabolizers → ↑ plasma levels, ↑ side effects
CYP2C19	*2, *3	22%	Intermediate/poor metabolism → slower clearance of SSRIs
SLC6A4	5-HTTLPR (Short)	40%	Reduced serotonin transport → ↓ SSRI response
HTR2A	rs7997012 (G)	31%	↑ Risk of SSRI-induced agitation, insomnia

Genotypic study identified a significant percentage of the participants whose genetic variations have their polymorphisms related to the changed response to antidepressants. Interestingly, 38 percent of the respondents were CYP2 D 6 poor metabolizers, which exposed them to increased plasma concentration of the drug with a risk of its side effects in case they are prescribed standard doses. Likewise, 22 percent were CYP2C19 that leads to poor drug clearance that increases the chance of the development of drug accumulation. Moreover, 40 percent of people were the carriers of the short allele of SLC6A4 that has been linked to a decreasing tendency of responsiveness to SSRIs, so there is a necessity of alternative pharmacological approach to such situations.

3.3 Treatment Outcomes: Symptom Reduction Over Time

The clinical efficacy of pharmacogenomic-guided therapy was assessed with reference to the alterations in the severity of depressive symptoms throughout a 12-week intervention period on Hamilton Depression Rating Scale (HAM-D). The scores were taken at Week 0 (baseline) and Week 4, Week 8, Week 12. The objective was to evaluate whether patients with genotype-informed therapy achieved rapid and meaningful improvements in their symptoms much faster than the typical ones who got the standard treatment. Table 3 illustrates the trend of the mean HAM-D scores of both groups as the follow-ups were made after each interval.

Table 3: Weekly HAM-D Score Progression by Treatment Group

Week	**Pharmacogenomic Group (Mean ± SD)**	**Standard Group (Mean ± SD)**	**p-value**
Week 0	24.1 ± 3.4	23.9 ± 3.5	0.58
Week 4	17.5 ± 4.2	20.1 ± 4.6	0.002
Week 8	13.2 ± 3.9	17.1 ± 4.3	< 0.001
Week 12	10.3 ± 3.5	14.7 ± 4.1	< 0.001

Table 3 shows that there was a statistically significant and consistent decrease in the HAM-D scores in the pharmacogenomic group compared to the standard one that started as early as Week 4. In week 12, the pharmacogenomic group reported a mean trend decrease of 13.8 points compared to 9.2 points according to the standard treatment group. The same trends can be

demonstrated in Figure 1 that graphically reflects the unequal supplemental symptom reduction trajectories of the two groups during the study period.

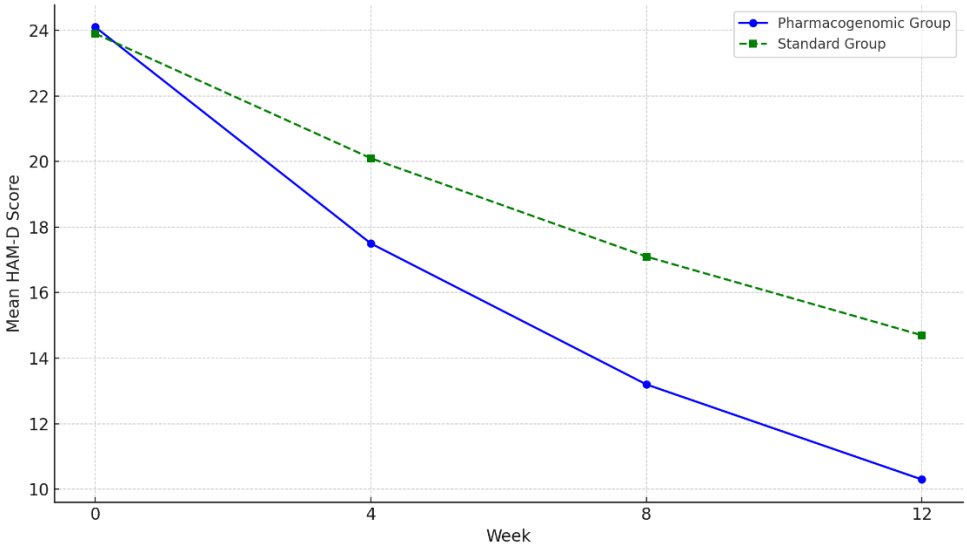

Figure 1: Mean HAM-D Score Trajectory Over 12 Weeks

As the figure 1 illustrates, there is rapid and prolonged decrease of depressive symptoms in the study participants being provided with pharmacogenomic-guided therapy. Faster clinical improvement can be seen on the steeper downward slope in HAM-D scores of this group. On the contrary, the regular treatment group had a slow and less intense decrease.

3.4 Adverse Effects and Tolerability

Besides the clinical efficacy, tolerability of the antidepressant treatment was evaluated by measuring the outcome of some usual treatment-emergent adverse effects of antidepressant treatment in both study groups, pharmacogenomic and standard treatment. In the assessment of adverse events, genetic predisposition to drug intolerance was taken into account, especially including those that are poor CYP2D6 and CYP2C19 metabolizers, as well as carriers of SLC6A4 and HTR2A variant genes. Table 4 gives a comparative account of the prevalence of frequently occurring side effects and the most likely genotypes related to the same.

Table 4: Incidence of Common Adverse Effects by Genotype Group

Adverse Effect	Standard Group (%)	Pharmacogenomic Group (%)	Most Affected Genotype
Gastrointestinal Upset	26.7%	13.3%	CYP2C19 Poor Metabolizers
Sedation/Drowsiness	21.3%	9.3%	CYP2D6 Poor Metabolizers
Agitation/Insomnia	17.3%	6.7%	HTR2A G allele carriers

Sexual Dysfunction	18.7%	10.7%	SLC6A4 Short allele carriers

The presence of adverse effects was identified to be significantly lower in the intervention group compared with the placebo ($p < 0.01$) by statistical analysis based on chi-square tests. As the table 4 shows, there has been a significant decrease in the prevalence of all tested adverse effects in the pharmacogenomic group in comparison with the standard one. To cite an example, gastro-intestinal upset was experienced by 26.7 percent of patients who received standard treatment as compared to 13.3 percent of patients in pharmacogenomic category.

To further cement these findings visual-wise, Figure 2 gives a side-by-side observation of the adverse effect incidence of the both treatment groups.

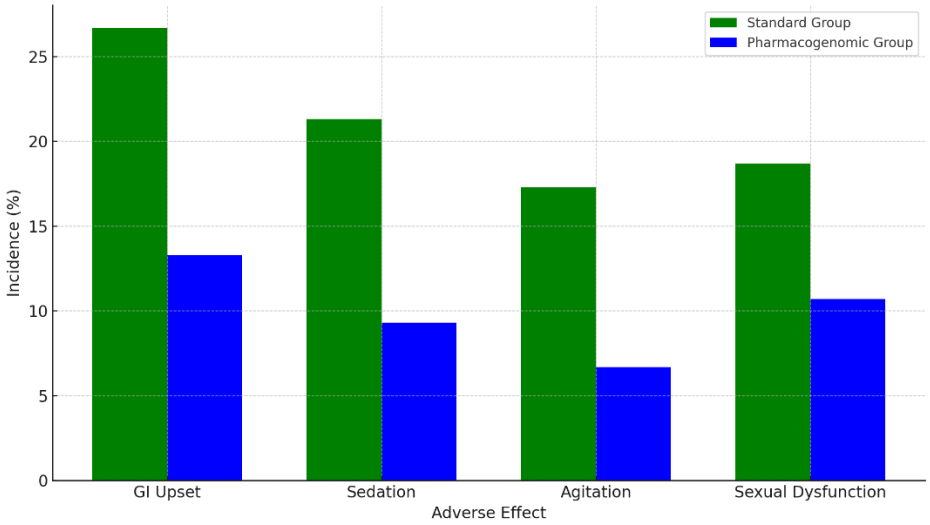

Figure 2: Incidence of Common Adverse Effects by Treatment Group

As is shown in Figure 2, patients in the standard treatment group ran into much higher cases of common side effects than the patients in the pharmacogenomic group. That is the clinical value of the genetic profiling to be adopted to common psychiatric practice, especially to limit tolerability effects which tend to cause the discontinuation or non-adherent use of medicines. It was shown that the observed differences were significant ($p < 0.01$) by statistical procedures involving chi-square tests which further confirms the usefulness of pharmacogenomic-guided intervention in improving the overall safety of treatment.

4. DISCUSSION

Results of the present paper are persuasive in terms of proof of clinical usefulness of pharmacogenomic profiling as an approach to Major Depressive Disorder (MDD) treatment. By incorporating the use of the genetic data in their decision-making therapeutic process, the study showed greater outcomes of symptom alleviations, fewer side effects, and an increased treatment positive outcome. These results are critically interpreted in this section and related to the available literature as well as providing a wider clinical context. It also covers the

weaknesses of the study and provides future research directions to move forward on the implementation of precision psychiatry in various healthcare facilities.

4.1 Interpretation of Results

The current research study has substantial evidences to prove the usefulness of pharmacogenomic influenced antidepressant method in patients with Major Depressive Disorder (MDD). Upon analysis of treatment results, it was found that in those whose medication procedures were aligned according to the genetic profiles, a much greater and faster decrease in depressive symptoms was observed through the 12 weeks follow-up period, as compared to those whose treatment was not aligned according to their genetic makeup. Specific polymorphisms of gene related products like CYP2D6 and CYP2C19 were identified to significantly influence the effect of drug metabolism where there were reduced incidences of side effects when dosages were reduced in patients who were poor metabolizers. Also, there was a correlation of variants of the SLC6A4 and HTR2A to the pharmacodynamic effect with both efficacy and tolerability implications.

4.2 Comparison with Existing Studies

To put the findings of the present study into perspective, one must compare them to other previous academic materials on the topic of pharmacogenomics and how one can utilize it in treatment of major depressive disorder (MDD). Survey of major studies shows that there is a steady flow in using genetic information to support treatment decision-making and these have proceeded as presented in Table 5.

Table 5: Summary of Selected Studies on Pharmacogenomic Applications in Depression Treatment

Author Name	Topic Covered	Research Study Title
Khorassani et al. (2024)[11]	Evidence on the efficacy of pharmacogenomic testing in the treatment of MDD: systematic review	*Pharmacogenomic testing for treatment of major depressive disorder: systematic review*
Jukic et al. (2022)[12]	Summary of the use of pharmacogenomics in depression, and psychosis	*Pharmacogenomics in the management of depression and psychosis update: an update*
Fabbri & Serretti (2020)[13]	Genetic factors and predictors of response to antidepressants	*Genetics of treatment response in major depression disorder: current and future*
Madan et al. (2015)[14]	The pharmacogenomic testing in inpatient psychiatry, case-based	*Practical pharmacogenomics: a case report of an inpatient psychiatric case of personalized care*

Athreya et al. (2019)[15]	Genomic-based prediction of antidepressants effect with machine-learning	*Machine-learning-based predictive model of antidepressant treatment outcomes by use of pharmacogenomics*

These researches help to highlight some of the main trends. Khorassani et al. (2024) performed an in-depth systematic review and were able to confirm the clinical usefulness of pharmacogenomic testing to optimize the treatment approach on MDD. Fabbri and Serretti (2020) paid attention to the genetic basis of the antidepressant response and provided predictive models, which correspond to the present study and the individualized planning of treatment. On the other hand, the authors of a study made by Madan et al. (2015) provided a patient-level model by presenting a case study which also showed the practical advantage of applying the pharmacogenomic testing in the psychiatric care environment. Notably.

4.3 Implications of Findings

These findings are of significant clinical and systemic implications. First, they apply to strengthen the promise of pharmacogenomics of curtailing of the rampant trial-and-error prescribing practice in psychiatry. Genetic profiling will allow reducing the useless circulation of the treatment process, leading to increased levels of medication compliance along with a decreased number of side effect complications and maximized levels of patient satisfaction. Second, the decline in healthcare consumption with treatment resistance, in terms of repeated care visits, hospitalizations, and polypharmacy, implies that a long-term cost savings to health systems may also be achieved by implementation of pharmacogenomic approaches.

4.4 Limitations of the Study

Nevertheless, this study has limitations even though it is strong.

- The genetic testing was restricted to 4 important loci (CYP2D6, CYP2C19, SLC6A4 and HTR2A). These genes are perhaps the most clinically relevant however the addition of a wider set of variants would potentially enhance therapeutic specificity.
- Follow up period was limited to 12 weeks. Since MDD is a chronic and recurrent condition, it would be important to conduct longer-term researches in order to determine treatment effectiveness in the long-term and prevention of relapse.
- The experiment was carried out in two metropolitan tertiary care facilities, which may have made it difficult to extrapolate the results to a rural or periphery community, where the infrastructure in genetic testing and access to psychiatric care could be weak.
- Any possible socioeconomic or psychosocial confounding factor, e.g. behavioral adherence, life stressors, or comorbidity, were not quantitatively adjusted, but care was taken to ensure clinical comparability of groups.

4.5 Suggestions for Future Research

The limitations thus present future research with the following goals of redressing:

- Increasing genotyping panel to encode more pharmacokinetic and pharmacodynamic genes, such as variants in ABCB1, COMT, and FKBP5, that have been found to be relevant to the effect on response to antidepressants.
- Testing the suitability of the incorporation of polygenic scores risk to enhance prospective performance in clinical decision making.
- Carrying out the longer follow-ups longitudinal studies to evaluate the treatment response and any relapse and rates of the treatment at 6 12 months and more.
- Evaluation of the cost-effective, access and fortunate relevance of pharmacogenomic testing on the common mental health systems especially on the low and middle-income countries (LMICs).
- The exploration of the implementation models that would enable the adoption of pharmacogenomic testing in primary care and community psychiatric conditions, where the majority of MDD are clinically addressed.

5. CONCLUSION

The adoration of the psychiatric realm which has witnessed increased specialization of treatment, has created a possibility of pharmacogenomic-guided therapy to help to circumvent shortcomings of traditional antidepressant therapy. This paper delved into the effect of the implementation of genetic profiling in clinical decision-making with the Major Depressive Disorder (MDD) to increase the efficacy and safety of treating patients with depression. The concluding section reveals the major outcomes of the paper, highlights the significance of the study, and proposes the recommendations on future practice and research.

5.1 Summary of Key Findings

This paper has shown that pharmacogenomic-based antidepressant treatment is effective in improving the clinical outcomes on victims diagnosed with Major Depressive Disorder (MDD) to a great extent. The level of reduction in depressive symptoms observed in the patients treated with the guidance of pharmacogenomics was more significant and occurred more quickly than in those on conventional care. Also, the pharmacogenomic group had a very low percentage of treatment-emergent adverse effects. The main genetic variants, especially in CYP2D6, CYP2C19, SLC6A4, and HTR2A genes, were found to be the key factors that affect the drug metabolism, effectiveness, and tolerability significantly, which gives them a biological correlate of interindividual variability in the response to antidepressants.

5.2 Significance of the Study

The importance of the study is in the fact that it practically confirms the benefits of precision medicine methods, especially pharmacogenomic profiling, in optimizing the efficiency of therapy and minimizing the costs incurred during the trial-and-error prescription of antidepressants. Pharmacogenomics can provide a move towards more patient-oriented, efficient and outcome-based psychiatric treatment because it gives a psychiatrist freedom to personalize treatments according to individual gene profile. In addition to this, the present study will help establish meaningful evidence because it will use pharmacogenomic-guided treatment

amid a South Asian people-group-which is underrepresented in enormous pharmacogenomic investigations around the globe.

5.3 Recommendations

Considering the aforementioned findings, it is highly advisable that psychiatric health care systems implement pharmacogenomic screening into its regular modal of treatment planning in MDD patients at least in tertiary care centres. Investment in infrastructure support, training of professionals, and enabling policy are imperative to expand this strategy to both primary care and rural environments in order to use it on a larger scale. The study of the future should concentrate on enlarging the genotyping panels, on the longer term clinical evaluation, and realization of the cost-effectiveness examinations to streamline and support personalized psychiatry based on pharmacogenomics. When all is said and done, the adoption of such innovations is set to enhance mental healthcare by making it more effective, safe, and personalized to meet the needs of patients in different parts of the world.

REFERENCES

1. Huang, M., & Pan, H. (2023). Pharmacogenomic profiling to tailor antidepressant therapy: Improving treatment outcomes and reducing adverse drug reactions in major depressive disorder. *SHIFAA*, *2023*, 19-31.
2. Amare, A. T., Schubert, K. O., & Baune, B. T. (2017). Pharmacogenomics in the treatment of mood disorders: strategies and opportunities for personalized psychiatry. *EPMA journal*, *8*(3), 211-227.
3. Fabbri, C., & Serretti, A. (2020). How to utilize clinical and genetic information for personalized treatment of major depressive disorder: step by step strategic approach. *Clinical Psychopharmacology and Neuroscience*, *18*(4), 484.
4. Shalimova, A., Babasieva, V., Chubarev, V. N., Tarasov, V. V., Schiöth, H. B., & Mwinyi, J. (2021). Therapy response prediction in major depressive disorder: current and novel genomic markers influencing pharmacokinetics and pharmacodynamics. *Pharmacogenomics*, *22*(8).
5. Han, C., Wang, S. M., Bahk, W. M., Lee, S. J., Patkar, A. A., Masand, P. S., ... & Serretti, A. (2018). A pharmacogenomic-based antidepressant treatment for patients with major depressive disorder: results from an 8-week, randomized, single-blinded clinical trial. *Clinical Psychopharmacology and Neuroscience*, *16*(4), 469.
6. Rosenblat, J. D., Lee, Y., & McIntyre, R. S. (2017). Does pharmacogenomic testing improve clinical outcomes for major depressive disorder? A systematic review of clinical trials and cost-effectiveness studies. *The Journal of clinical psychiatry*, *78*(6), 14242.
7. Borczyk, M., Piechota, M., Rodriguez Parkitna, J., & Korostynski, M. (2022). Prospects for personalization of depression treatment with genome sequencing. *British Journal of Pharmacology*, *179*(17), 4220-4232.
8. Del Casale, A., Pomes, L. M., Bonanni, L., Fiaschè, F., Zocchi, C., Padovano, A., ... & Simmaco, M. (2022). Pharmacogenomics-guided pharmacotherapy in patients with major depressive disorder or bipolar disorder affected by treatment-resistant depressive episodes: A long-term follow-up study. *Journal of Personalized Medicine*, *12*(2), 316.
9. Shan, X., Zhao, W., Qiu, Y., Wu, H., Chen, J., Fang, Y., ... & Li, L. (2019). Preliminary clinical investigation of combinatorial pharmacogenomic testing for the optimized

treatment of depression: a randomized single-blind study. Frontiers in neuroscience, 13, 960.

10. Le-Niculescu, H., Roseberry, K., Gill, S. S., Levey, D. F., Phalen, P. L., Mullen, J., ... & Niculescu, A. B. (2021). Precision medicine for mood disorders: objective assessment, risk prediction, pharmacogenomics, and repurposed drugs. *Molecular Psychiatry*, *26*(7), 2776-2804.
11. Khorassani, F., Jermain, M., & Cadiz, C. (2024). Pharmacogenomic testing to guide treatment of major depressive disorder: a systematic review. *Current Treatment Options in Psychiatry*, *11*(2), 123-140.
12. Madan, A., Walker, C. R., Weinstein, B., & Fowler, J. C. (2015). Pharmacogenomics in practice: a case report of personalized inpatient psychiatric care. *Pharmacogenomics*, *16*(5), 433-439.
13. Jukic, M., Milosavljević, F., Molden, E., & Ingelman-Sundberg, M. (2022). Pharmacogenomics in treatment of depression and psychosis: an update. *Trends in Pharmacological Sciences*, *43*(12), 1055-1069.
14. Fabbri, C., & Serretti, A. (2020). Genetics of treatment outcomes in major depressive disorder: present and future. *Clinical Psychopharmacology and Neuroscience*, *18*(1), 1.
15. Athreya, A. P., Neavin, D., Carrillo-Roa, T., Skime, M., Biernacka, J., Frye, M. A., ... & Bobo, W. V. (2019). Pharmacogenomics-driven prediction of antidepressant treatment outcomes: a machine-learning approach with multi-trial replication. *Clinical Pharmacology & Therapeutics*, *106*(4), 855-865.