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Original Article

Influence of ABCB1 Polymorphisms on Outcomes of Breast Cancer

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ABSTRACT

Background: The importance of genotyping ABCB1 polymorphism genes in predicting the outcome of breast cancer patients receiving anthracycline and taxane-based regimens was reported. However, interethnic variations play a significant role in predicting the response to chemotherapy. **Materials and Methods:** This prospective study was carried out to assess the influence of ABCB1 genotypes on breast cancer response. Genotyping of ABCB1 was carried out by real-time PCR (7300 Applied Biosystems; Life Technologies Corporation, USA) using TaqMan SNP genotyping assays, Response assessments were evaluated as per RECIST criteria version. **Results:** A total of 170 locally advanced breast cancer patients treated with anthracycline-based neoadjuvant chemotherapy were recruited. The genotype frequencies of ABCB11236 homozygous wild-type variant CC, the heterozygous variant CT, and the mutant homozygous variant TT were 23.0%, 44.0%, and 33.0% respectively. The genotype frequencies of ABCB1 1236 homozygous wild-type variant CC, the heterozygous variant CT, and the mutant homozygous variant TT were 28.0%, 42.0%, and 30.0% respectively. Similarly, the ABCB1 2677 wild-type variant GG (18%), TT (26% AA (2% the heterozygous variant GA (7%), GT (45%), TA (2%), and the mutant homozygous variant were observed. **Conclusion:** Our study revealed that ABCB1 3435 C>T polymorphism was associated with response as patients with ABCB1 3435 mutant genotypes had better response. Patients having ABCB1 1236 mutant genotypes (CT & TT) had more risk for neutropenia and vomiting, suggesting the significance of ABCB1 genotypes in predicting response and toxicity in anthracycline-based regimens

Keywords: Breast cancer, ABCB1, Clinical response, Neutropenia, Multidrug-resistant protein Efflux transporter.

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INTRODUCTION

Cancer is characterized by the uncontrolled growth and progression of abnormal cells. Worldwide Cancer is one of the leading causes of significant morbidity and mortality even though tremendous improvement has been achieved concerning its diagnosis and treatment. The available reports show that the estimated number of cases of cancer patients across the globe was 14 million in 2012 resulting in 8 million deaths. The incidence is expected to increase to 24 million cases by 2035.[1] The insights of the human genome have a significant impact on understanding the molecular mechanisms behind most of the cancers. The introduction of advanced diagnostic tools and newer molecular-targeted treatment strategies have benefited certain cancers. Breast cancer continues to be a disease with a significant health burden. In the United States of America, it is expected that 2.5 lakh new cases of invasive breast cancer and sixty-three thousand cases of in situ breast carcinoma to be diagnosed among women.[2] In India, breast cancer accounts for 5-8% of all cancers and reports roughly 1 lakh new cases per year.[3] The response to neoadjuvant chemotherapy is mostly imprecise and may not reflect the pathologic response well.[4] Patients who experienced the full pathological response following neoadjuvant chemotherapy were found to have a longer overall and progression-free survival.[5,6] An important problem arises in the treatment of breast cancer when the response and toxicity experienced by the patients vary considerably. Inter-individual variability in drug response could be either due to genetic or non-genetic factors, and genetics play a prominent role in both the efficacy and toxicity of a drug.[7] Pharmacogenomics

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helps select the predictive markers of drug efficacy and safety by assessing the genetic bases for interindividual variations in response and toxicity.[8,9] These approaches help in drug safety, treatment efficacy, and personalization of medicine. As the Indian population differs ethnically from the worldwide population, there exists a strong indication for performing pharmacogenetics and population-based pharmacokinetic analysis. Early detection and analysis of predictive biomarkers of breast cancer were important.[10] Various genes influencing the response and risk were reported in south Indian breast cancer patients.[11] P-glycoprotein, an ATP-Binding Cassette (ABC) superfamily transporter, is encoded by MDR1. MDR1 is the most extensively researched resistance-related gene and the first ABC transporter to be identified.[12] The ABC superfamily of proteins is involved in the transport of a wide range of endogenous and exogenous substances. P-gp is involved in the transport of anthracyclines, immunosuppressants, and other compounds.[13] So far, 48 human ABC genes have been documented and divided into 7 subfamilies. The MDR1/ABCB1 gene is situated on chromosome 7 and consists of 28 exons ranging from 49 to 587 bp.[14] Studies on mutations indicate that the MDR1 gene exhibits significant polymorphism and is extensively utilized to investigate the structure-function relationships of P-glycoprotein.[15] The interindividual variability in P-glycoprotein activity and susceptibility to various diseases may be attributed to the extensive polymorphism variants in the MDR1 gene.[16,17] The most widely studied SNPs that are investigated for their clinical effects are ABCB1 C3435T, ABCB1 C1236T, and ABCB1 G2677A/T. Any genetic variations of the MDR1 gene can affect the patient's response to treatment. we have investigated the effect of ABCB1 genotypes on response to anthracycline-based neoadjuvant treatment

MATERIALS AND METHODS

Study population

We conducted this prospective study to evaluate the impact of genotypes on response and toxicity. Before recruiting the patients, we obtained approval from the institutional ethics committee. We recruited all newly diagnosed breast cancer patients receiving doxorubicin or epirubicin-based chemotherapy. We performed fine needle aspiration cytology and core needle biopsy for the diagnosis of breast cancer.

Response assessment

Patients underwent an MRI scan of the breast before the first cycle of chemotherapy and after the completion of chemotherapy. Based on an MRI scan, we assessed the tumor response using the Response Evaluation Criteria in Solid Tumors (RECIST) criteria. We graded the toxicity according to the commonly used criteria for adverse event v6.

DNA extraction and genotyping

We collected 5 mL of blood from the patients in tubes containing Ethylenediaminetetraacetic Acid (EDTA) for DNA extraction. We centrifuged the blood at 3000 g and discarded the supernatant plasma. We separated the leukocytes and extracted DNA using the phenol-chloroform method. We quantified the extracted DNA using a photometer (Eppendorf AG 22331, Germany). We used real-time PCR to genotype ABCB1 using TaqMan assays [Assay id: C__3237198_20]. In order to distinguish between alleles, we employed the 7300 Sequence Detection Software (SDS) version 1.4.

Statistical analysis

The chi-square test tested the observed genotype frequencies for the Hardy-Weinberg equilibrium. We analyzed the associations of polymorphic variants of ABCB1 genotypes with clinicopathological parameters and response to neoadjuvant chemotherapy by calculating the relative risk and 95% Confidence Intervals (CIs) using the 2-tailed Fisher exact test. We performed the statistical analysis of the data using GraphPad InStat 10.2.2 (GraphPad Software Inc., San Diego, CA, USA). $p < 0.05$ was considered significant.

RESULTS

The study recruited a total of 170 patients with locally advanced breast cancer. The median age of the patients was 50 years (23-60). Table 1 summarizes the clinicopathological characteristics across genotypes.

ABCB1 genotyping and tumor response assessment

Table 2 provides the genotype frequencies of ABCB1 C3435T, C1236T, and G2677A/T. We assessed 145 patients, finding that the clinical complete, partial, and non-response rates were 31%, 60%, and 9%, respectively.

Genotyping, response, and toxicity

Table 3 provides the genotype frequencies of ABCB1 3435 C>T, ABCB1 1236 C>T, and ABCB1 2677G>A/T across clinical settings. We kept a close watch on the patients until the end of their treatment to gauge any potential side effects. For patients with severe toxicity, we have agreed to postpone treatment for a maximum of 2 weeks. We investigated the occurrence of blood-related toxicity, the use of additional GCSF prophylaxis, and the decrease in dosage for grades 3 and 4. Additionally, we assessed the occurrence of non-blood-related toxicity, the need for dosage reduction, and the possibility of changing the chemotherapy regimen for these same grades. In our study, hematological toxicities like neutropenia, leucopenia, and anemia accounted for 48.17%. 31.7% and 9.1%, There were reports of non-hematological toxicities such as mucositis (49.0%), nausea (45.1%), vomiting (52.8%), diarrhea (34.14%),

myalgia (57.3%), and hand-foot syndrome (30.4%). Four patients died due to toxic effects like febrile neutropenia, severe diarrhea, and mucositis. The study results revealed that ABCB1 1236 C>T polymorphism influences the risk of neutropenia. Patients having ABCB1 1236 mutant genotypes (CT &TT) had 1.4 times more risk for neutropenia (p = 0.04) when compared to wild-type carriers (Table 4). Similarly, ABCB1 1236 wild carriers have a significant risk of vomiting when compared to homozygous and heterozygous carriers (Table 5). There is no association between ABCB1 genotypes and any of the other toxicities mentioned above.

DISCUSSION

Breast cancer is one of the most chemo-responsive cancers, but its therapeutic benefits are poor. In locally advanced breast cancer, patients can now undergo surgery for cancers that were previously inoperable, preoperative systemic chemotherapy is a worthwhile practice. Preoperative chemotherapy is recognized as an effective treatment for patients with locally advanced breast cancer.[18,19] Greater interindividual variations in response were observed for patients undergoing chemotherapy. Although chemotherapy improves disease-free survival and overall survival, identifying patients who do benefit from chemotherapy is challenging because breast cancer is a heterogeneous disease with many subtypes. Preliminary investigation of the resistance mechanism of drugs in cell lines has been explored for various drugs like anthracyclines and platinum drugs.[20] The importance of pharmacogenomics studies on transporters, metabolizing enzymes, and drug targets is well known. This aids in evaluating individuals at risk for unfavorable outcomes and increased toxicity. P-glycoprotein (P-gp) encoded by the ABCB1 gene is involved in the transport of the anthracyclines and taxanes.[21] SNPs reported in the ABCB1 gene can influence efflux transport

Table 1: Baseline characteristics of Study population across genotypes.

	Total n=170	ABCB1 3435 n=170				ABCB1 1236 n=170				ABCB1 2677G/T/A n=170			
	n	CC	CT	TT	Ref	CC	CT	TT	Ref	GG	GA GT AT	AATT	Ref
Menopause status													0.06
Premenopausal	62	17	29	16	0.66	11	30	21	0.53	5	36	20	
Postmenopausal	108	36	46	23		27	49	32		24	58	26	
Tumor Grade													0.90
Grade 1	20	8	7	5	0.39	4	9	7	0.92	3	11	6	
Grade 2/3	150	40	74	36		33	71	46		28	82	40	
Tumor size													0.47
T3	107	35	46	26	0.11	27	48	32	0.50	16	63	28	
T4	63	13	37	13		11	31	21		13	31	18	
Lymph node													0.11
0	44	14	18	12	0.41	10	19	15	0.85	6	21	17	
1 to 3	126	34	67	27		28	60	38		25	73	29	
Stage													0.17
II	39	13	14	12	0.09	8	17	14	0.76	4	21	14	
III	131	32	73	26		29	63	39		24	86	31	
ER status													0.70
Negative	89	25	50	24	0.63	21	38	30	0.56	15	48	26	
Positive	81	23	43	15		17	41	23		14	47	20	
PR status													0.52
Negative	108	31	48	29	0.20	24	50	34	0.99	19	57	32	
Positive	62	17	35	10		14	29	19		10	38	14	
HER2													
Negative	69	17	38	15	0.51	14	36	19	0.46	12	41	16	0.63
Positive	101	31	46	24		24	43	34		17	54	30	

Table 2: ABCB1 genotypes of the study population

Genotype	Genotype frequency in %			Established frequency in South Indian healthy population in %		
ABCB1 1236C>T	CC	CT	TT	CC	CT	TT
	23.0	44.0	33.0	27.0	39.0	34.0
ABCB1 3435 C>T	CC	CT	TT	CC	CT	TT
	28.0	42.0	30.0	18.0	47.0	35.0
ABCB12677G>T/A	GG	GA	GT	GG	GA	GT
	18.0	7.0	45.0	20.0	3.0	44.0
	TA	AA	TT	TA	AA	TT
	26.0	2.0	2.0	30.0	2.0	1.0

Table 3: Association of ABCB1 polymorphism with clinical response

Genotype	Responders n (%)		Non-responders *n* (%)		*p*-value RR (95%C.I.)
	CR *n*=45	PR *n*=88	SD *n*=8	PD *n*=4	
ABCB1 3435 CC	14	20	5	2	
CT	19	40	2	1	
TT	12	28	1	1	
CC	34 (25.5)		7 (58)		0.0381
CT+TT	99 (74.5)		5 (42)		0.8712 (0.9925-1.328)
ABCB1 1236 CC	12	16	1	1	
CT	16	46	4	1	
TT	17	26	3	2	
CC	28 (21.0)		2 (17)		0.904
CT+TT	103 (79.0)		10 (83)		0.98 (0.8732-1.092)
ABCB1 2799 GG	7	13	2	1	
GT	24	39	3	1	
TT	10	25	2	2	
GA	4	6	0	0	
AA	0	2	1	0	
AT	1	3	0	0	
GG	20 (15)		3 (25)		0.4056
GT+TT+GA+ AA+AT	113 (85)		9 (75)		0.9388 (0.7952-1.108)

and expression of P-gp.[22] In Our study we have explored the impact of ABCB1 SNPs (3435C>T, 1236C>T, and 2677G>T/A) with a response to treatment. ABCB1 3435C>T polymorphism was found to be significantly associated with therapeutic response in patients receiving anthracyclines and taxane-based regimens. In our study, the patients having ABCB1 3435 mutant genotypes (homozygous and heterozygous) responded better than wild CC genotypes (p=0.03). Our results agreed with a study that reported significantly better response in heterozygous CT, homozygous TT, and combined genotypes of CT & TT of ABCB1 C3435T compared to wild CC genotype.[23] Kafka et al reported patients having the ABCB1 3435 TT genotype achieved a complete response to chemotherapy.[24] Ashariati *et al.*,[25] reported that patients with the ABCB1 3435TT genotype had better clinical responses than the CT genotype. The prognostic significance of ABCB1 3435C>T polymorphism in taxane response was also reported.[26,27] Our findings support that ABCB1 3435 T allele carriers respond better to anthracyclines and taxanes-based regimens. Some of the studies reported patients with the ABCB1 3435 CC genotype had better responses and the ABCB1 3435

TT genotype had the worst prognosis, which is contradictory to our study.[28] Wu *et al.*,[23] also reported only ABCB1 3435 TT had a better clinical response and the other two polymorphisms were not associated with the response which is in agreement with our study. Studies have reported the influence of polymorphic variants of ABCB1 in hematological and nonhematological toxicities.[29,30] However, some studies have reported no association of ABCB1 genotypes with chemotherapy toxicities.[31] Our results indicated that patients having ABCB1 1236 homozygous and heterozygous variants had 1.4 times more risk for neutropenia (p=0.04). Similarly, a study reported the patients harboring the ABCB1 1236 C>T variant to have hematological toxicity in breast cancer patients which is in agreement with our studies.[30] There was no correlation between the 1236C>T ABCB1 polymorphism and severe toxicities caused by chemotherapeutic drugs in another study.[32] A study reported from China revealed a lack of association of polymorphic variants of ABCB1 with hematologic toxicities in breast cancer patients.[33] Some of the contradictory findings reported in other studies may be due to differences in chemotherapy regimens and ethnicity of the study population. Our study results showed that ABCB1 1236 wild carriers have a significant risk of vomiting when compared to homozygous and heterozygous carriers. Zoto *et al.*,[34] T showed that the ABCB1 435TT, C1236TT, and 2677TT genotypes, and their combination, were associated with a much superior clinical anti-emetic response to 5-HT3 antagonists after chemotherapy. Indonesian patients harboring the CTG haplotype of the ABCB1 gene had Grade 3 and 4 chemotherapy-induced nausea and vomiting than other haplotypes in breast cancer patients on doxorubicin-based therapy.[35] In Our study antiemetics were administered prior to chemotherapy. Our findings indicate that the ABCB1 1236TT genotype was associated with a reduced risk of vomiting, suggesting the role of the ABCB1 transporter in response and toxicity. However, some studies have reported no association of ABCB1 genotypes with nausea and vomiting.[36,37] Further Randomized control trials based on genotypes of candidate genes involved in chemotherapy-induced nausea and vomiting are to be explored for translating the research into clinical practice. One of the limitations of our study was the non-responders reported in our study were only 8% as the patients received a combination of anthracyclines and taxanes-based regimens. The influence of polymorphic variants in the pharmacokinetics of doxorubicin should be further explored.

Table 4: Association of ABCB1 polymorphism with neutropenia

Genotype	Toxicity n=164		p-value RR (95%C.I.)
	No toxicity (n=85)	Any grade neutropenia (n=79)	
ABCB1 1236 CC	25	12	
CT	33	44	
TT	27	23	
CC	25	12	0.04
CT+TT	60	67	1.43 (1.071 to 1.910)
ABCB1 3435 CC CT	26	21	
TT	40	39	
	19	19	
CC	26	21	0.60
CT+TT	59	58	1.09 (0.8017 to 1.501)
ABCB1 2799 GG	16	11	
GT	34	40	
TT	22	20	
GA	6	2	
AA	3	1	
AT	3	5	
GG	16	13	0.83
GT+TT+GA+ AA+AT	69	66	1.07 (0.7476 to 1.559

Table 5: Association of ABCB1 polymorphism with nausea

Genotype	Toxicity *n*=164		*p*-value RR (95%C.I.)
	No toxicity (*n*=78)	Any grade Nausea (*n*=92)	
ABCB1 1236 CC	13	28	
CT	42	42	
TT	23	22	
CC	13	28	0.04
CT+TT	65	64	0.62 (0.3890 to 1.018)
ABCB1 3435 CC	22	25	1.0
CT	38	37	1.02
TT	18	30	(0.7166 to 1.475)
CC	22	25	
CT+TT	56	67	
ABCB1 2799 GG	17	12	
GT	35	43	
TT	19	23	
GA	3	8	
AA	1	3	
AT	3	3	
GG	17	12	0.15
GT+TT+GA+ AA+AT	61	70	1.35 (0.9457 to 1.941)

CONCLUSION

Our results showed that patients harboring homozygous and heterozygous variants of ABCB1were more likely to achieve a complete clinical response and the risk of neutropenia in patients harboring variants of ABCB1 suggesting the role of ABCB1 in both response and toxicity in south Indian breast cancer patients. In Our population, RCT based on ABCB1 genotypes on anthracyclines response and toxicity is to be carried out before integrating into clinical practice.

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ABBREVIATIONS

ABCB1: ATP-binding cassette (ABC) superfamily; **PCR:** Polymerase Chain Reaction; **RECIST:** Response evaluation criteria in solid tumors; **MRI:** Magnetic resonance imaging; **MDR1:** Multidrug resistance1; **CI:** Confidence Interval; **5-HT:** 5-Hydroxy tryptamine; **EDTA:** Ethylenediaminetetraacetic acid; **RT-PCR:** Real-time polymerase chain reaction: **GCSF:** Growth Colony Stimulating Factor; **P-gp:** P-glycoprotein.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The JIPMER Ethics Committee approved this study. All participants provided informed written consent before their inclusion in the study, and the research was carried out in accordance with the Helsinki Declaration.

SUMMARY

This report comprises of influence of ABCB1 polymorphism on the response and toxicity of breast cancer patients receiving anthracyclines-based chemotherapy. Interethicity played a significant role in determining the response of drugs in various populations so the study explored it. The method of genotyping carried out was by using Realtime PCR. The study revealed that the polymorphic variant of ABCB1 3435C>T and ABCB1 1236C>T affected response and toxicity respectively in South Indian breast cancer patients.

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