

HLA-A*31:01 and lamotrigine-induced severe cutaneous adverse drug reactions in a Korean population

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Lamotrigine (LTG) was first synthesized in the early 1980s and approved by the US Food and Drug Administration in 1994. Since then, $it\ has\ been\ used\ worldwide\ as\ an\ effective\ anticonvulsant\ and\ as\ a\ mood\ stabilizer.\ However,\ LTG\ can\ cause\ adverse\ reactions$ including cutaneous adverse drug reactions with approximately 10% probability. Severe cutaneous adverse reactions (SCARs) are the most serious form and include Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug rash with the most serious form and include Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug rash with the most serious form and include Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug rash with the most serious form and include Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug rash with the most serious form and include Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug rash with the most serious form and include Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug rash with the most serious form and the serious form and theosinophilia and systemic symptoms (DRESS). Although SCAR is clinically important because of its high mortality, there are currently no predictive markers for LTG-induced SCAR. Recent advances in pharmacogenomics have found that certain HLA alleles are significantly associated with SCARs caused by drugs, such as carbamazepine and allopurinol.^{2–4} In addition, several studies have described the economic efficiency of genetic screening before prescribing a specific drug.3 Based on these findings, we performed HLA genotyping to identify HLA alleles associated with the risk for LTG-induced SCAR in a Korean population.



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