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Drug-induced hypersensitivity reactions and their associated predictors using spontaneous reported adrs from the Tanzania Medicines and Medical Devices Authority (TMDA) vigflow database 2017-2018- Elias Musa Bukundi- Muhimbili university of Health

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Hypersensitivity reactions are public health problem which contribute to 10% to 20% of hospitalization. The objective of this analysis was to determine the prevalence, signal and risk factors for drug-induced hypersensitivity from spontaneous reported at TMDA Vig flow database from January 2017 to December 2018. A secondary data analysis of 321 spontaneous reported ADRs cases were analyzed. Predictors of drug induced hypersensitivity was identified using multivariate logistics analysis. Drug induced hypersensitivity statistical association (Signals) was determined using reporting odds ratio (ROR).Mapping of the geographical distribution of the reported ADRs was done using QGIS.The prevalance of drug-induced hypersensitivity was 39.56%, the independent predictors for drug-induced hypersensitivity were reports from southern highland zones (OR=7.29), oral route of administration (OR=37.50), reports from other health professional (R=6.508), age group between 15-28 years (OR=0.180), having a non-serious adverse reaction (R=3.97) and being recovered from ADRs at the time of reporting (R=4.076). A signal associated with drug-hypersensitivity was detected in Isoniazid tabs, cotrimoxazole tabs, Artemether lumefantrine tabs, RHZE and antiprotozoal ATC group of drugs. Mara region, Kagera region, Njombe region, Katavi region, Simiyu region, Song we region and Mtwara region did not report any ADRs in 2017 to 2018. More attention should be given to patients aged 15-28, those who use drug by oral route, non-serious adverse reaction, living the Southern highland zone, those using anti TB drugs, ALU, Antibiotics and antiprotozoal drug.

Drug-induced hypersensitivity reactions represent a major concern for clinicians, patients, regulators and drug developers. Severe hypersensitivity is associated with high morbidity and mortality, it cannot be predicted from the known pharmacology of the drug and it is usually detected post-marketing when a large number of patients have been exposed to a particular drug. Recent success in developing clinically useful genetic tests that have allowed us to predict the risk of abacavir-induced hypersensitivity has helped to pave the path for a pharmacogenetic approach. However, the loop from identifying a genetic association to improving clinical outcome is still lacking for many drugs. In this commentary, we discuss the progress of hypersensitivity pharmacogenomics over the last decade and point out what remains to be done in the future. The current efforts of the international community are focused on the development of consortia, which aim to standardize disease phenotypes, but also to collect larger numbers of well-pheno typed patients and to pool biological samples through these collaborations. In addition, it is necessary to advance our knowledge of hypersensitivity mechanisms through functional studies, which will lead to the development of predictive and diagnostic tests. Drug treatment often leads to adverse events (AE). Some of these are so-called medication errors which occur due to the handling of the drug, rather than due to the drug itself. Adverse drug reactions (ADR), colloquially called “side effects,” are adverse events that are due to the inherent biological effects of the drug. These, in turn, are divided into pharmacologically mediated ADR (type A) and hypersensitivity reactions. Type A reactions can occasionally be therapeutically useful or even lead to new indications: for example, minoxidil causes hair growth, and sildenafil has a beneficial effect on erectile dysfunction. Drug-induced liver damage is a well-known kind of type A reaction that can be caused, e.g., by an overdose of acetaminophen, whereas flucloxacillin-associated liver damage is an HLA-associated type B reaction. Type A reactions are generally dose-dependent, while type B reactions are generally considered to be independent of the dose once a low threshold dose has been exceeded.

Both classic immunological (allergic) and non-allergic hypersensitivity reactions involve activation of the immune system or of its effector pathways, such as inflammatory reactions (Table 1, Figure Hypersensitivity reactions are clinically categorized as either immediate (arising less than one hour after exposure) or late (arising more than one hour after exposure). The classic allergic reactions are divided into four types, in the scheme of Coombs and Gell; types I and IV are the ones most

commonly encountered. A drug may trigger very different kinds of hypersensitivity reactions across individuals, or even in the same individual. Penicillins, for example, may induce non-allergic hypersensitivity, as well as allergies of types I–IV. These different kinds of reaction can also arise simultaneously. Topical penicillin preparations are no longer on the market because of the high risk of contact allergy (10%). Hypersensitivity reactions may be provoked by variants in genes being involved in the synthesis or degradation of inflammatory mediators such as bradykinin, histamine, prostaglandins, or leukotrienes, or in the activity of the corresponding receptors. The most prominent example is an asthma attack induced by a non steroidal anti-inflammatory drug such as diclofenac. Another, potentially dangerous reaction of this type is angioedema induced by ACE inhibitors. The latter reaction is associated with a genetic variant of plasma amino peptidase.