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Drug Addiction Disorder Described by Occasional Relapses

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Abstract

The bound drug in the bloodstream may act as a reservoir for the drug. Some drugs accumulate in certain tissues, which can also act as reservoirs of the extra drug. These tissues slowly release the drug into the bloodstream, keeping blood levels of the drug from decreasing rapidly and thereby prolonging the effect of the drug.

Keywords: Drugs; Pharmacodynamics; Therapeutic Plan; Blood levels; Epidemiological factors; ADRs

Introduction

Some drugs, such as those that accumulate in fatty tissues, leave the tissues so slowly that they circulate in the bloodstream for days after a person has stopped taking the drug. Distribution of a given drug may also vary from person to person. For instance, obese people may store large amounts of fat-soluble drug, whereas very thin people may store relatively little. Older people, even when thin, may store large amounts of fat-soluble drugs because the proportion of body fat increases with aging. Alcohol affects the metabolism of many drugs and it facilitates the development of ADRs. Alcohol drug interaction refers to the possibility that alcohol may change the intensity of the development of ADRs making it more toxic or harmful to the patient either in a pharmacokinetic or pharmacodynamics manner [1]. Taking alcohol with certain drugs can cause many ADRs like nausea, vomiting, headaches, drowsiness, fainting, and loss of coordination, hypotension and many other ADRs. Internal bleeding may occur due to severe ulceration if alcohol is taken with NSAIDs by a patient having peptic ulcer or ex-peptic ulcer or gastritis. Chronic alcohol consumption activates enzymes which transform some drugs into toxic chemicals that can damage the liver and other body organs. Alcohol can also magnify the inhibitory effects of sedatives and narcotics at their site of action in the brain.

Discussion

Alcohol might affect the functionality of the liver causing liver cirrhosis and liver hepatitis which in turn affect the ability of this organ to metabolize drugs especially drugs metabolized by the liver and drugs which have first pass metabolism [2]. E.g. the toxicity of beta blockers increases with liver problems. This will lead to drug interactions, because of that physicians and pharmacists must warn patients about the health hazards that might be caused by alcohol drug interaction. Alcohol drug interaction may be more harmful when elderly patients mix them together, as age and alcohol lead to many health problems. Evidences suggest that ethnicity exerts a substantial influence on drug response and action. Drug action varies greatly between individuals. Ethnic background is controlled by genetic factors, which makes the inter-individual differences due to polymorphisms in genes encoding drug metabolizing enzymes, drug transporters, and receptors. Recent development suggests that ADRs may be avoided by individualizing the therapeutic plan according to genetics. David suggested genetics play a crucial part in the willingness of some patients to develop ADR for a specific drug over others. A study on epidemiological risk factors for hypersensitivity reactions to abacavir found the Caucasian race as a risk factor for ADRs. In a recent cohort study evaluated risk factors for ADRs associated with angiotensin-converting enzyme inhibitors involving people who had to discontinue therapy due to ADRs, African Americans were found to be more susceptible to developing ACE-related angioedema than other ethnic groups [3]. Ethnicity is an important demographic variable contributing to inter-individual variability in medication metabolism and response. Some studies are discussing the issue that genetic factors can determine individual susceptibility to both dose-dependent and dose-independent ADRs. Determinants of susceptibility include kinetic factors, such as gene polymorphisms in cytochrome enzymes, and dynamic factors, such as polymorphisms in drug targets. The relative importance of these factors will depend on the nature of the ADRs; however, it is likely that more than one gene will be involved in most instances. Different ethnic groups have different risks for important ADRs to cardiovascular drugs [4]. Ethnic groups may therefore be one determinant of harm of a given treatment in the individual patient, either because it acts as a surrogate measure of genetic makeup or because cultural factors alter the risk. Black patients had a relative risk of angioedema compared with non-black patients, and the risk of intracranial haemorrhage was higher in black patients than non-black patients. These findings may help healthcare providers present more accurate and relevant data to their patients when prescribing cardiovascular therapy [5]. The problem with trials is that the groups of people in trials are not necessary representatives of the general population and if they are, their background might not be specified. It is also documented that the risk of angioedema with blood pressure lowering drugs was three times greater in black patients than non-black patients. The risk of cough was also nearly three times higher in East Asian patients compared with white patients. For thrombolytic therapy, the risk of bleeding increased fold in black patients compared with non-black patients. Another study shows that black patients vs. non-black patients experienced moderate to severe bleeding following thrombolytic therapy; these data come from the global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries. The same study concluded that there was significantly more depression from hydrochlorothiazide for black patients compared with white patients [6]. Non-whites had a higher risk of hospital admission from bleeding after oral

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anticoagulation for deep vein thrombosis. Of few patients treated with ibutilide fumarate injection for the recent onset of atrial fibrillation or atrial flutter, black patients, compared with white patients, developed Torsade de Pointes after treatment. Human leukocyte Antigen genotype is an essential predictor of the susceptibility of drug-induced liver toxicity. HLA genotype is not the main cause of liver toxicity; there are other genotypes like SLCO1B1 which lead to simvastatin myotoxicity. Ten percent of patients who take Carbamazepine suffer from cutaneous adverse reactions. These ADRs have been attributed to the human leukocyte antigen genotype. In a recent study, it has been determined that carrying HLA-B 1502 in Asians was associated with pooled odds ratio for Carbamazepine-induced Stevens Johnson syndrome and toxic epidermal necrolysis. Another recent study showed that patients with ADRs had a higher frequency of CYP1A2 low activity allele combinations and lower CYP levels than patients without ADRs. In Parkinson's disease patients with UDP-glucuronosyl-transferase genotypes are prone to ADRs and to catechol-O-ethyltransferase inhibitors. Smoking, one of the risk factors of many diseases like peptic ulcer, cancer and cardiovascular diseases. It also affects the metabolic process by affecting liver enzymes acting as a potent inducer of the hepatic cytochrome isoenzymes possibly [7]. Many drugs are substrates for hepatic CYP1A2, and their metabolism can be induced in smokers, resulting in a clinically significant decrease in pharmacologic effects. These drug interactions are not caused by nicotine, the cause is tobacco. Because it stimulates the sympathetic nervous system, nicotine can counter the pharmacologic actions of some drugs. More research findings world-wide revealed the smoking-drug interaction, and theophylline, flecainide, insulin, oral contraceptives, beta-blockers, thiothixene and H2 blockers are medicines whose therapeutic responses can be affected by smoking. One clinical study showed that on average insulin-dependent diabetic smokers needed more insulin than non-smokers, and up to 30% more if they smoked heavily [8]. Cigarette smoking increases the rate of heparin clearance, possibly because of the smoking-related activation of thrombosis with increases of heparin binding to antithrombin III. Cutaneous vasoconstriction by nicotine may decrease the rate of insulin absorption after subcutaneous administration. Cigarette smoking also reduces the effect of beta blockers on blood pressure and heart rate. Taking several drugs, whether prescription or over-the counter, contributes to the risk of having an ADR. The number and severity of ADRs increases disproportionately as the number of drugs taken increases. Many definitions are applied for polypharmacy. It is different from scholar to scholar but the basic concept of taking more medications at the same time than are clinically appropriate remains constant. It implies to the prescription of too many medications for a particular patient, with a possibility of increased risk of ADRs. The more the medications that are prescribed the more the possibility of polypharmacy, this does not necessarily mean however those patients should not take many medications [9]. Polypharmacy is a result of many conditions; patients might suffer from more than one disease especially among the elderly. Patients might seek more than one prescriber at the same time for different diseases or acute or chronic conditions. ADRs may occur due to drug interaction, synergism, duplication, additive effect, discontinuation of therapy, changing the dose to save money, skipping some medications and physiological antagonism. One important reason for the development of ADRs from polypharmacy is the inability of some patients especially the elderly to keep track of using their medications regardless of how well the medications may work if given alone. If the patients are not strict enough to take the medications as prescribed, then they will separate from treatment and not take the medication properly [10]. Economic value of the medications may lead to skipping some of them which in turn causes shortage of treatment and the development of adverse events. Prescribing cascade is also a result of polypharmacy in which certain drugs are used to treat the adverse effect of other drugs. This will potentially lead to an endless line of medications used by the patient.

Conclusion

It is possible that symptoms and signs of polypharmacy could be overlooked by confusing them with symptoms of aging or the disease itself. This in turn will result in more medications being taken by the patients. Constipation, diarrhoea, tiredness, weakness, skin rashes, falls, anxiety and many other symptoms could be caused by both diseases and polypharmacy

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Conflict of Interest

None

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