ISSN 2321 – 7359 EISSN 2321 – 7367

Medical Science
The International Weekly Journal for Medicine

Concomitant use of angiotensin converting enzyme inhibitor ACEI and angiotensin receptor blocker ARB in medication therapy of a co-morbid patient in a tertiary health care facility

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Publication History
Received: 11 April 2014
Accepted: 21 May 2014
Published: 28 May 2014

Citation
Aisha Khan, Iqra Farooq, Meena Mairaj, Atta Abbas. Concomitant use of angiotensin converting enzyme inhibitor ACEI and angiotensin receptor blocker ARB in medication therapy of a co-morbid patient in a tertiary health care facility. *Medical Science*, 2014, 8(31), 67-70

ABSTRACT

Introduction: A stroke is the sudden death of brain cells due to improper blood supply. The result is injury and subsequently death of neurons resulting in abnormal brain function. Blood flow to the brain can be disrupted by either a blockage or rupture of an artery in brain. The present case is of transient ischemic attack TIA due to cerebral vascular accident. Concomitant use of ACEI and ARB was seen in pharmacotherapy of the patient.

Case Presentation: This case is based on a female patient of 42 years who was in good health according to the family. At midnight she developed anxiety and pulsating headache associated with vertigo. The patient has a history of hypertension and asthma so was taken to the hospital where she developed left side weakness and slurring of speech. The main goal was to regain motor activity and to maintain heart and brain conditions to a stable state.

Conclusion: In this case ACE inhibitor and ARB had been given together which can lead to serious adverse effects. Presence of a pharmacist would be beneficial to minimize such medication errors.

Keywords: Concomitant; ACE inhibitors; ARB

1. INTRODUCTION

Cerebral vascular accident CVA usually refers to the more common embolic or transient ischemic stroke. These strokes occur from a blood clot that occurs inside the vessel and prevents blood flow to brain (Stroke; The American

Heart Association AHA). Most CVAs are the result of atherosclerosis, hypertension or a combination of both. Transient ischemic attack TIA is also known as the mini-stoke it does not last longer. The patient is receiving ACE inhibitor and angiotensin II antagonist together in therapy which can lead to serious adverse effect (Transient ischemic attack TIA).

2. CASE PRESENTATION

A 42 yrs old female came in with anxiety and pulsating headache associated with vertigo according to her case file she was diagnosed by of cerebral vascular accident CVA associated with transient ischemic attack. She had a history of hypertension HTN and asthma. After getting initial treatment in medical ICU she was shifted to neurology department since she developed left side weakness and slurring of speech. The patient was recommended to do regular physiotherapy.

3. MANAGEMENT AND MONITORING

The main goal was to diagnose the source of clot or atherosclerosis by computed tomography CT scan and magnetic resonance imaging MRI. The patient was given alpha-choline injections indicated for cognitive disorders caused by head trauma or cerebral surgery, for upper and lower limbs paralysis and it also acts as a cerebral vasodilator in dose of 4 ampoules in 100 n/s BD as a therapy for stroke patients. The goals of therapy in such condition are repair of neuronal membranes via increased synthesis of phosphatidylcholine, repair of damaged cholinergic neurons via potentiation of acetylcholine production, and reduction of free fatty acid buildup at the site of stroke-induced nerve damage (Citicoline Monograph, 2008). A suitable therapy geared towards achieving these objectives is usually initiated. The second most important goal is to improve the motor functions and it is achieved by physiotherapy and speech therapy.

For managing TIA aspirin was given to the patient, indicated for TIA when blood vessels are damaged or are diseased, platelets clump together over the hole or vessel tear to facilitate repair. Cyclo-oxygenase COX activates a chemical known as thromboxane A_2 that causes platelets to stick together to form a plug over the damaged area. The aggregation of platelet plug in association with the clotting process, results in a fibrin clot which stops bleeding and aids repair of the blood vessel (How aspirin works, 2010). It was given in the dose of 75mg. For lowering the risk of hyperlipidemia a lipid lowering agent atrovastatin in dose of 20mg was prescribed. Atorvastatin is a competitive inhibitor of hydroxymethylglutaryl-coenzyme A HMG-CoA reductase, the rate-determining enzyme in cholesterol biosynthesis via the mevalonate pathway. HMG-CoA reductase catalyzes the conversion of HMG-CoA to mevalonate. Atorvastatin acts primarily in the liver. Decreased hepatic cholesterol levels increases hepatic uptake of cholesterol and reduces plasma cholesterol level (Atorvastatin).

The patient was given valsartan which is an angiotensin II receptor antagonist 40mg for HTN. Valsartan is an ARB that selectively inhibits the binding of angiotensin II to AT_1, which is found in many tissues such as vascular smooth muscle and the adrenal glands. This effectively inhibits the AT_1-mediated vasoconstriction and aldosterone secreting effects of angiotensin II and results in a decrease in vascular resistance and blood pressure. Valsartan is selective for AT_1 and has virtually no affinity for AT_2. Inhibition of aldosterone secretion inhibits sodium and water re-absorption in the kidneys while decreasing potassium excretion (Valsartan (a)). An angiotensin converting enzyme inhibitor ACEI i.e. captopril which is in dose of 25mg was also prescribed. Captopril prevents the conversion of angiotensin I to angiotensin II (a potent vasoconstrictor) through inhibition of ACE by competing with physiologic substrate angiotensin I for active site of ACE; inhibition of ACE initially results in decreased plasma angiotensin II concentrations and consequently, blood pressure may be reduced in part through decreased vasoconstriction, increases rennin activity, and decreases aldosterone secretion. In addition, it increases renal blood flow (Captopril (a)). For the management of asthma the patient was given tablet montelukast 10mg, a leukotrine receptor antagonist and nebulizer ipratropium bromide. Due to lack of movement and impaired speech the patient was is in depression therefore so she was given a selective serotonin reuptake inhibitor SSRI escitalopram 10mg.

4. DISCUSSION

The management of this case requires multi dimensional therapy. Apart from the drug therapy patient requires regular physiotherapy and sessions of speech therapy as she had developed left side weakness and slurring of speech. She also needed to be devised a continuous care plan and therapeutic lifestyle changes were required to lower her blood pressure BP and to maintain her lipid levels (Atta Abbas, 2014). Here the patient requires continuous monitoring of brain activity and as she has TIA blood cholesterol level and the blood pressure should also be monitored as the patient has a history of hyper tension. Patient is also given bronchodilators to manage asthma. The drug therapy should be continued with strict compliance and regular follow ups are required to check the recovery. It is evident clinically that CVA that leads to TIA is due to a blockage of blood vessel via arterial embolism, a clot that travel to the brain and that particular area get infected and the function of that specific area is then compromised.

In this case, all the medications given to the patient for the therapy were appropriate, however concomitant use of ACEI and ARB can cause major adverse effect and damage to the patient (http://eurheartj.oxfordjournals.org/content/26/22/2361.full.pdf). The patient was prescribed tablet valsartan 40mg which is an angiotensin II antagonist for HTN and captopril 25mg which is and ACE inhibitor for HTN, the two drugs if given together can lead to serious drug interaction and adverse effects (Norman M Kaplan). Valsartan may increase the level of potassium in the blood (Captopril (b)). If this medicine is taken with ACE inhibitor that can increase blood potassium level and the effect is enhanced. As captopril blocks the action of ACE, it reduces the production of angiotensin II. This means that the blood vessels are allowed to relax and widen. The overall effect of this is a drop in blood pressure; hence captopril can be used to lower high blood pressure (Varsartan (b)).

ACE inhibitors which inhibit the conversion of angiotensin I to angiotensin II block the kinase activity resulting in increase in bradykinin levels that can lead to dry cough angioedema and increase in serum creatinine level and potassium which can lead to renal insufficiency. When the serum creatinine concentration is more than 30%and serum potassium value increases to more than 5.6mmol/L the therapy should be discontinued immediately (Drug interaction report of valsartan and captopril). The concomitant use of ACE inhibitor and ARB for hypertension is not safe and not tolerated. Moreover, the combination of these drugs can lead to adverse effects. This is encored by the NICE guidelines of 2011 which stipulates not to combine an ACE inhibitor with an ARB to treat hypertension (Clinical management of primary hypertension in adults). This is the dilemma of the health care system and the flaws in the health practices (Sundus Kirmani et al. 2014). Had there been a clinical pharmacist in the health care team, the issue of concomitant use of drug would have been resolved (Omar Qadeer and Atta Abbas, 2014).

5. CONCLUSION

The concomitant use of ACEI and ARB pose a threat to the patient and inclusion of a clinical pharmacist in the health care management team is the need of the hour since it is a pharmacist who can easily identify and rectify any medication error in the pharmacotherapy of patients.

ACKNOWLEDGEMENT

The team of authors express their gratitude to the patient and the staff for consenting and accessing the medical information.

STATEMENT OF CONSENT

The medical information was documented after obtaining the patient consent.

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