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A RETEROSPECTIVE STUDY ON CEREBROVASCULAR ACCIDENT AND CLINICAL PHARMACIST INTERVENTIONS IN THE ASSESSMENT AND MANAGEMENT OF ISCHEMIC STROKE

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ABSTRACT

The need of study was performed to know the route of various complications leading to the ischemic stroke. The main aim of the study was to study about cerebrovascular accident and clinical pharmacist intervention in the assessment and management of Ischemic Stroke. The study was conducted in the Neurology department at BBR tertiary care hospital. This is a retrospective, observational study conducted in a period of 6 months in the Neurology department. In our study we have observed the data and finally concludes that ischemic stroke was caused due to several comorbidities as mentioned above. If they were identified in the initial stages before leading to the ischemic stroke they can be treated. We observed that the management for the ischemic stroke are statins and anti-coagulants are the best choice of drugs either in single or combination therapy along with the conservative management for the comorbidities leading to severity of the condition in ischemic stroke patients. We have observed several drug interactions also which are closely monitored, moderate and severe interactions which can cause other complications resulting in worsening the patient condition. The clinical pharmacist role is more acceptance in the hospital. So, the inclusion of pharmacists with proper clinical practice knowledge could improve the safety care and quality assurance of life in patients with ischemic stroke.

KEYWORDS: Ischemic stroke, Cerebrovascular accident, Clinical pharmacist interventions, Retrospective study.

INTRODUCTION

Stroke is defined as a sudden onset of a neurological deficit caused by an acute focal injury to the central nervous system due to a vascular cause.[1] The incidence of strokes occurring every year worldwide is about 17 million and it is the second leading cause of death after coronary artery disease.[2] It is the fifth leading cause of death in the United States, i.e., killing more than 130,000 Americans each year[3] and affecting 795,000 people annually.[4] It is the third most common cause of disability and reduces mobility in more than half of stroke survivors in ages 65 and over.[4,5] Furthermore, the economic burden of stroke on the nation through health care services, medications, rehabilitation and loss of productivity is around $33 billion annually.[4] By 2020 in developed countries, it is predicted that stroke will be accountable for 6.2% of the total burden of illness.[6] These data put forward the need for controlling risk factors, knowledge of identifying the signs of stroke, timely reperfusion therapies and measures to improve delivery of the aforementioned resources for the best possible outcome.

Ischemic strokes are the most common (≈85%), the rest being hemorrhagic that include cerebral and subarachnoid (≈15%).[7] The Trial of Org10172 (TOAST) is the most commonly used classification that identifies five subtypes in acute ischemic stroke: 1) large artery atherosclerosis 2) cardio-embolism 3) small vessel occlusion 4) stroke of other determined etiology 5) stroke of undetermined etiology.[8]

The events resulting from any subtype of ischemic stroke result in the loss of blood supply, oxygen, nutrients and elimination of metabolic wastes. These resulting changes obstruct normal neuronal functioning.[9] This ultimately results in neuronal death/necrosis from occlusion of the vessel. The brain tissue is exquisitely sensitive to these changes, and the therapeutic window that is needed to prevent reversible ischemia from becoming irreversible infarction[10] is narrow and stresses the phrase “time is brain”. This concept is especially important to minimizing evolving insult and controlling the propagation of ischemic penumbra.[11,12,13] Furthermore from a therapeutic point of view, this crucial time provides a “window of opportunity” in reversing the neurological symptoms either partly or completely through acute interventional approaches, either invasively or non-invasive.[14]

The main aim of the study was to study about cerebrovascular accident and clinical pharmacist intervention in the assessment and management of Ischemic Stroke. The objectives are to reduce the disease causing comorbidities, To prevent the more number of drug interactions, to identify the number of causes leading to the ischemic stroke, to focus on the signs and symptoms of the patients rather than the cure of disease, to reduce the morbidity and mortality rate of diseased people, to provide appropriate treatment to the patients in the initial stages, to prevent disease condition, to improve mental, physical and social conditions of patients to provide good health, to analyses the treatment suggested according to their diseased condition.

The need of study was performed to know the route of various complications leading to the ischemic stroke. Along with the clinical pharmacist interventions in the assessment and management of ischemic stroke patients. To identify best choice of drugs for the treatment of stroke along with supportive care for the complications causing stroke. The role of clinical pharmacist is to help providing information of evidence based, rational treatment to the ischemic stroke patients.

METHODOLOGY

Study location: The study was conducted in the Neurology department at BBR tertiary care hospital.
Study period: The study will be conducted in a period of 6 months (i.e., October 2019 to March 2020)

Plan of work:

- Collection of data.
- Ethical committee approval.
- Literature review was carried out.
- Analysis of data.
- Report the data.

Study design: This is a retrospective, observational study conducted in a period of 6 months in theNeurology department.

Study population: Patients with ischemic stroke are admitted in the in-patient department of neurologic department of BBR hospital.

Study creteria: The patients with ischemic stroke in the neurology department were enrolled into the study after taking neurologist consent and by considering following inclusion and exclusion criteria.

Inclusion criteria: All the in-patients diagnosed with stroke by a consultant neurologist, in the neurology department were included in this study.

Exclusion criteria:

- Patients who were under day care management.
- Patients who were not willing to participate in the study.
- All out patients in OPD.
- Pediatrics.
- Pregnancy and breast-feeding patients.

Source of data:

- Patient's history records.
- Patient's progress records.
- Treatment charts.

Analysis of data:
Collected data is analysed through Microsoft Excel.

RESULTS

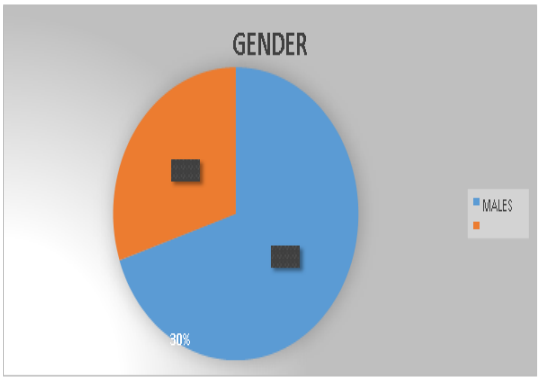

Fig. 1: Representation of patients according to the gender.

In this study patients were segregated into male and female. A total of 136 patients were included in this study, among 136 patients 95 (69.85%) were male and 41 (30.14%) werefemales.

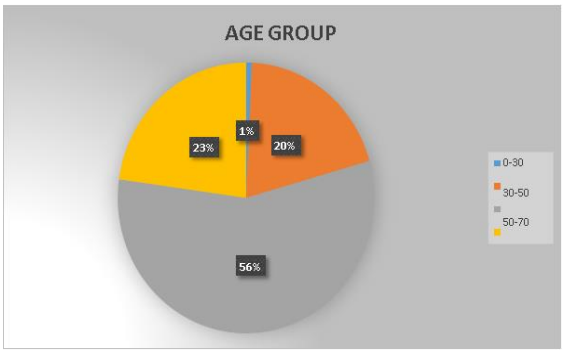

Fig. 2: Representation of patients according to the age group.

In the above-mentioned graph majority of the 77 patients (56%) were 50-70yrs followedby 31 patients (23%) were above 70yrs followed by 27 patients (20%) were 30-50yrs followed by 1 patient (1%) were 0-30yrs.

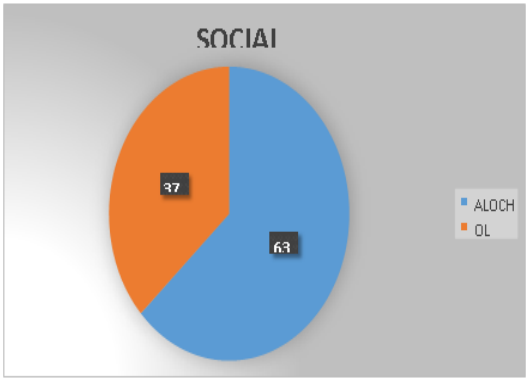

Fig. 3: Representation of patients according to their social habits.

In the above represented graph majority of the 24 patients (63%) were alcohol consumers and the rest 14 patients (37%) were cigarette smokers.

Table 1: Male patients with hypertension, Diabetes and Both.

Age in years	Hypertension (HTN)	Diabetes	th HTN andDiabetes
11-20	-	-	-
21-30	-	-	-
31-40	2	2	1
41-50	6	3	1
51-60	23	15	13
61-70	20	19	15
71-80	8	6	4
81-90	3	2	2
91-100	1	1	1

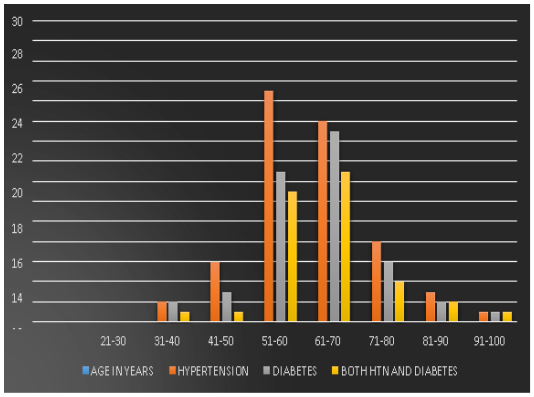

Fig. 4: Representation of m4ale patients with hypertension, Diabetes and Both.

From the above graph representation of male patients with hypertension, diabetes mellitus and both. Out of in which 2 patients with hypertension, 2 patients with diabetes mellitus and 1 patient with both were 31-40yrs followed by 23 patients with hypertension,15 patients with diabetes mellitus and 13 patients are with both were from 51-60yrs followed by 20 patients hypertension,19 patients with diabetes mellitus and 15 patients with both were 61-70years followed by 8 patients with hypertension,6 patients with diabetes mellitus and 4 patients with both were 71-80yrs followed by 6 patients with hypertension, 3 patients with diabetes mellitus and 1 patient with both were 41-50yrs followed by 3 patients with hypertension, 2 patients with diabetes mellitus and 2 patients with both were 81-90yrs followed by 1 patient with hypertension and 1 patient with diabetes mellitus and 1 patient with both were 91-100yrs

Table 2: Female patients with hypertension, Diabetes and Both.

Age in years	Hypertension	Diabetes	th HTN andDiabetes
11-20	-	-	-
21-30	1	1	1
31-40	1	-	-
41-50	2	2	2
51-60	9	6	6
61-70	13	8	8
71-80	12	6	6
81-90	-	-	-
91-100	-	-	-

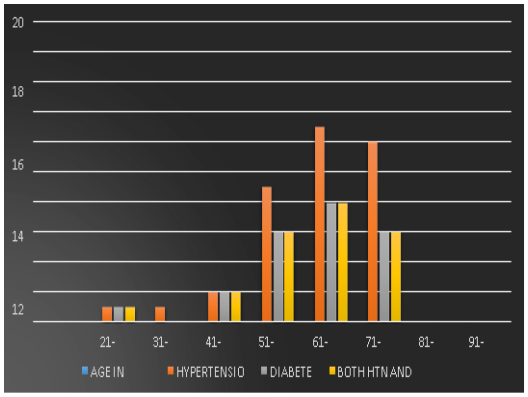

Fig. 5: Representation of female patients with hypertension, Diabetes and Both.

From the above graph representation of female patients with hypertension, diabetes mellitus and both 1 patient with hypertension, 1 patient with diabetes mellitus and 1 patient with both were 21-30yrs followed by 1 patient with hypertension were 31-40yrs followed by 2 patients with hypertension, 2 patients with diabetes mellitus 2 patients with both were 41-50yrs followed by 9 patients with hypertension, 6 patients with diabetes mellitus and 6 patients withboth were 51-60yrs followed by 13 patients with hypertension, 8 patients with diabetes mellitus and 8 patients with both were 61-70yrs followed by 12 patients with hypertension, 6 patients with diabetes mellitus and 6 patients with both were 71-80yrs

Table 3: Patients with different comorbidities.

Comorbidites	Number ofpatients	Percentage
Cad	11	12%
Stent	6	6%
Asthma	4	4%
monarykoch's	4	4%
Hypotyroidism	9	9%
Seizures	15	16%
Dysarthria	4	4%
Cva	34	36%
Recurrentstroke	9	9%

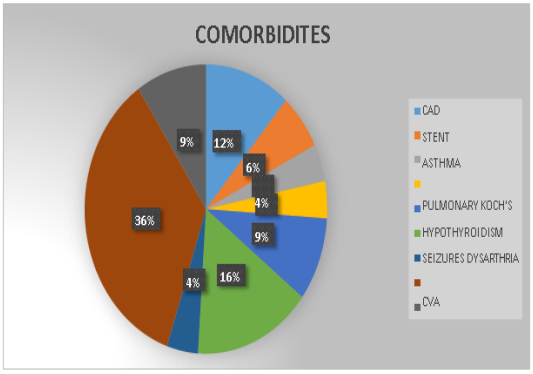

Fig. 6: Representation of patients according to comorbidities.

In the above demonstrated graph patients were divided according to comorbidites like CAD with 11 patients (12%), Stent with 6 patients (6%), Asthma with 4 patients (4%) Hypothyroidism with 9 patients (9%), Seizures with 15 patients (16%), Dysarthria with 4 patients (4%), CVA with 34 patients (36%), Recurrent ischemic stroke with 9 patients (9%)

Treatment

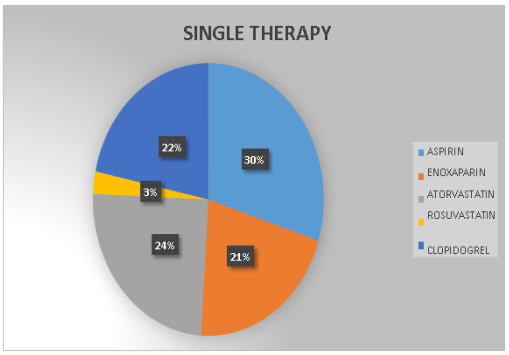

Fig. 7: Representation of patients with single therapy.

In the above single drug regimen the most prescribed drugs was aspirin-113(30%), followed by Atorvastatin-93(25%), followed by clopidogrel-82 (22%), followed by enoxaparin-80 (21%), followed by rosuvastatin-10 (3%).

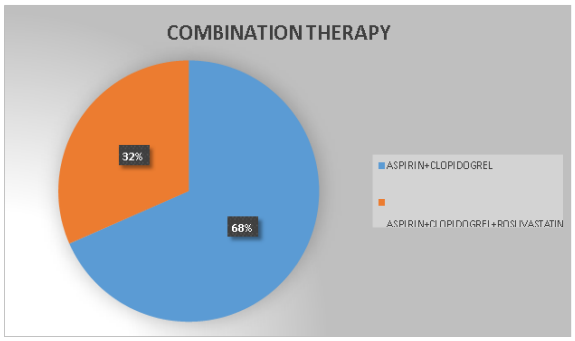

Fig. 8: Representation of patients with combination therapy.

In combination therapy the most prescribed combination drugs were Aspirin + Clopidogrel – 13 (68%) followed by Aspirin + Clopidogrel + Rosuvastatin- 6 (32%) in eachcase respectively

Drug interactions

Minor:

- Aspirin + Glimepiride: The effect of Glimepiride is increased by Aspirin by plasma protein binding competition by replacing it from plasma protein binding site or by impeding its metabolism.
- Fosphenytoin + Acetaminophen: Hydantoins may elevate the possible hepatotoxicityof acetaminophen and decline its pharmacological effects. The mechanism may be allied to the evocation of acetaminophen metabolism by following increase in hepatotoxic metabolites.
- Escitalopram + Metoprolol: Escitalopram may elevate the plasma concentration of Metoprolol. The

mechanism may be allied to inhibition of CYP2D6 metabolism. Elevated levels may decrease the beta blocker cardioselectivity.

- Clopidogrel + Fosphenytoin: The metabolism of Fosphenytoin is decreased by Clopidogrel as it inhibits the CYP2C9 isoenzymes, there by leading to toxicity of thesubstrate drug.
- Clonazepam + Acetaminophen: Clonazepam metabolism is enhanced when given with Acetaminophen. CYP3A4 isoenzyme is involved in the metabolism of Clonazepam which is capable of causing an incline in the plasma Clonazepam levels by 30-38%.

Monitor closely:

- Fosphenytoin + Atorvastatin: The serum concentration of Atorvastatin and its metabolites are decreased when co-administered with Fosphenytoin. The efficacy ofAtorvastatin is also reduced. It occurs due to potentiated metabolism of Atorvastatinas it is a substrate of CYP3A4 and Fosphenytoin is a potent inducer of CYP3A4.
- Enoxaparin + Aspirin: Aspirin increases the anticoagulant activity of Enoxaparin due to additive pharmacological actions.
- Levodopa + Hydrochlorothiazide: The risk of severity of hypotension and orthostatic hypotension can be increased when Hydrochlorothiazide is given along with Levodopa.
- Levodopa + Propranolol: Co-administration of Levodopa and Propranolol may resultin additive risk of experiencing decreased blood pressure or severe orthostatic hypotension.
- Metoprolol + Telmisartan: The risk of severe hypokalemia can be increased when Metoprolol is combined with Telmisartan.
- Propranolol + Glimepiride: The therapeutic efficacy of Glimepiride can be increased when used along with Propranolol.
- Aspirin + Propranolol: The serum concentration of Aspirin is decreased when given along with Propranolol. The efficacy of Propranolol is decreased.
- Escitalopram + Clopidogrel: The risk of bleeding can be increased when Escitalopram is combined with Clopidogrel. It inhibits the re-uptake of serotonin into platelets in much the same way it inhibits re-uptake of pre-synaptic neurons, this decrease in available platelet serotonin can result in diminished clotting response and increased bleeding. It also effects clotting ability through action on the glycogen IIb/IIIa surfacereceptor or up regulation of glycogen synthase kinase 3-beta on platelets.
- Aspirin + Telmisartan: The risk of renal failure, hyperkalemia and hypertension can be increased when Telmisartan is combined with Aspirin. NSAIDs produce vasoconstriction particularly when the renin-angiotensin system is inhibited. This can raise the blood pressure and cause renal dysfunction or acute kidney injury. This renal dysfunction can result in increased potassium retention and ultimately hyperkalemia.
- Fosphenytoin + Escitalopram: The metabolism of Fosphenytoin can be decreased when given along with Escitalopram. As these are substrates of the CYP2C19 enzyme compete for metabolism by this enzyme, causing increased exposure to either CYP2C19 substrate drug.
- Pantoprazole + Clopidogrel: The serum concentration of active metabolites of Clopidogrel can be reduced when it is used along with Pantoprazole leading in loss ofefficacy.
- Telmisartan + Labetalol: The metabolism of Telmisartan can be decreased when combined with Labetalol. This occurs due to inhibition of CYP2C19.
- Telmisartan + Atorvastatin: Telmisartan may decrease the excretion rate of Atorvastatin which could result in higher serum level. This is duet to competition forthe BSEP transporter with bile salts.

Severe-Use alternative:

- Fosphenytoin + Pantoprazole: The metabolism of Fosphenytoin can be decreased when combined with Pantoprazole as CYP2C19 inhibitors may significantly increase the exposure to CYP2C19 substrates.
- Fosphenytoin + Clopidogrel: The metabolism of Fosphenytoin can be decreased when given along with Clopidogrel. The serum concentration of Fosphenytoin is increased as a result of inhibition of CYP2C9 activity.
- Labetalol + Metoprolol: The metabolism of Labetalol can be decreased when combined with Metoprolol. This leads to increased serum concentration as well as risk of adverse effects. The metabolism of drug is affected as Metoprolol is a moderate CYP2D6 inhibitor and Labetalol is metabolized by CYP2D6.
- Escitalopram + Ondansetron: The risk of serotonin syndrome can be increased when Ondansetron is given along with Escitalopram. Escitalopram increases serotonergic activity in the CNS through inhibition of serotonin re-uptake by serotonin transporter protein. This leads to development of serotonin syndrome.
- Metoprolol + Propranolol: The metabolism of Propranolol can be decreased when combined with Metoprolol. Metoprolol is a moderate CYP2D6 inhibitor and Propranolol is metabolized by CYP2D6. The administration of both the drugs may decrease the metabolism of Propranolol leading to increased serum concentration and increased risk of adverse effects.

DISCUSSION

Clinical pharmacists are the early emerging ones to perform roles and responsibilities for providing a better care for patients in the hospitals. A proper medical staff

with proper clinical practice should be present to observe the patients in the hospital.

We have carried out a study regarding assessment and management of ischemic stroke. Our main goal is to discuss regarding the comorbidities and therapy given to the patients. Although so many healthcare workers are present in the hospital to take care of stroke patients but we were supposed to observe and monitor the patients and observe their responses who were undergone treatment or surgery in the hospital. In the hospital, clinical pharmacists act as a secondary role of physicians in order to take the own decisions to provide proper safety and management regarding the patient's conditions in the hospital.

In our retrospective and observational study, we collected data from the hospital and observed the ischemic stroke patients who were suffering with various complications. From the collected data we have observed that ischemic stroke is occurring due to the several comorbidities like Hypertension, Diabetes Mellitus, Alcohol, Smoking, CVA, CVD, Asthma, Pulmonary Koch's, Hypothyroidism, seizures. We have collected and observed the various factors causing ischemic stroke. We observed that the management for the ischemic stroke are statins and anti-coagulants are the best choice of drugs either in single or combination therapy. We have also observed the drug interactions which are monitor closely, moderate and severe interactions which can lead to other complications ending up severity of diseased condition.

A proper medical staff should be present to provide a proper relevant information for better counsel of the patients. An improper clinical practice may also impact the risk assessments in the patients. So, that proper training or practice is necessary to develop the skills of pharmacists to take care the patients in the hospital in order to minimize the risk of drug interactions in the ischemic stroke patients.

CONCLUSION

In our study we have observed the data and finally concludes that ischemic stroke was caused due to several comorbidities as mentioned above. If they were identified in the initial stages before leading to the ischemic stroke they can be treated. We observed that the management for the ischemic stroke are statins and anti-coagulants are the best choice of drugs either in single or combination therapy along with the conservative management for the comorbidites leading to severity of the condition in ischemic stroke patients. We have observed several drug interactions also which are closely monitored, moderate and severe interactions which can cause other complications resulting in worsening the patient condition. The clinical pharmacist role is more acceptance in the hospital. So, the inclusion of pharmacists with proper clinical practice knowledge could improve the safety care and quality assurance of life in patients with ischemic stroke.

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