SEVEN HILLS TIMES

www.shcptirupati.com

Venkatatramapuram, TIRUPATI

Volume – 3 | Issue No – 6 | November 2019

An Official Publication of Department of Pharmacy Practice
Seven Hills College of Pharmacy, Tirupati,
Andhra Pradesh.

In association with Sri Padmavathi Medical College for Women, Alipiri Road, Tirupati, Chittoor (Dist.,), Andhra Pradesh, India.
www.shcptirupati.com;shcpdic333@gmail.com
Phone : 7730084513, 7702484513

VISION

To emerge as one of the premier pharmacy colleges in the country and produce pharmacy professional of global standards.

MISSION

- To deliver quality academic programs in Pharmacy and empower the students to meet industrial standards.
- To build student community with high ethical standards to undertake R&D in thrust areas of national and • international standards.
- To extend viable outreach programs for the health care need of the society.
- To develop industry institute interaction and foster entrepreneurial spirit among the graduates

BURDEN OF COMORBIDITIES AND THEIR TREATMENT IN PATIENTS WITH ACTIVE TUBERCULOSIS: A PROSPECTIVE OBSERVATIONAL STUDY

B Priyanka
Pharm.D Intern

Background: TB is an infectious disease caused by bacillus "Mycobacterium tuberculosis. "Multiple drug therapy in active tuberculosis patients along with comorbidities may cause several drug related problems. So the study concentrates upon assessing the rate of drug interactions between anti tubercular regimens and comorbidy regimens. Study on tubercular and comorbidy drugs allows us to understand incidence and pattern of drug related problems which can be further used to prevent recurrence of drug related problems thereby improving quality of treatment.

Methodology: The patients who are with active tuberculosis along with other comorbidities were taken and assessed the drug related problems i.e drug interactions and adverse drug effects between antitubercular regimens and comorbid regimens and the obtained continuous variables was summarized as mean and standard deviation and the results (descriptive data) was summarized as numbers and percentages.

Results: A total of 105 tuberculosis patient's data was collected, with a mean age of 43.73% (Age range from 15-75 years. Out of total 105, 68 patients were with several comorbidities like DM, HTN, CAD, Epilepsy, CKD, Asthma, COPD, and HIV. In this study, a total of 162 drug related problems were found in 81 patients. Out of them 79 major drug interactions and 74 moderate drug interactions were found and 9 ATT induced adverse effects were found.

DIFFERENT TYPES OF COMORBIDITIES

COMORBIDITIES	Frequency	Percentage (%)
Hypertension	06	10.34
Hypertension and CAD	05	8.6
Hypertension and CKD	02	3.84
Hypertension and hypothyroidism	01	1.72
Hypertension, Epilepsy and Type-II DM	01	1.72
Hypertension, CAD, and Type-II DM	01	1.72
Hypertension, Type-II DM and hemiparesis	02	3.45
Hypertension and Type-II DM	07	12.06
Diabetes mellitus Type-II	07	12.06
Diabetes mellitus Type-I	01	1.72
Epilepsy	06	10.34
Epilepsy and Hemiplegia	03	5.17
Epilepsy and OCD	01	1.72
Asthma	04	6.89
COPD	03	5.17
Bronchial asthma, COPD overlap syndrome	01	1.72
Hypothyroidism	02	3.45
Chronic kidney disease	01	1.72
Chronic kidney disease and Malnutrition	01	1.72
Ischemic stroke	01	1.72
Rheumatoid arthritis	01	1.72
Human immunodeficiency disease	01	1.72

ADVERSE DRUG REACTIONS

Adverse drug effect	No. of patients	Drugs
Elevation of LFT	4	Isoniazid(H), Rifampicin(R), Pyrazinamide(Z)
Thrombocytopenia	2	Rifampicin(R)
Anemia	3	Isoniazid(H), Rifampicin(R), Pyrazinamide(Z)

DRUG INTERACTIONS

No.of patients on drugs	Percentage (%)	Interacting drugs
01	0.67	H/R ↔Inj. Insulin
01	0.67	R ↔T. Glibenclamide
20	13.41	R ↔T. Amlodipine
02	1.34	H ↔T. Phenytoin
02	1.34	R↔T. Phenytoin
03	2.01	R↔T. Atorvastatin
02	1.34	R↔T. Ivabradine
16	10.73	R↔T. Pantoprazole
02	1.34	R↔T. Ondansetron
14	9.39	R↔Inj. Dexamethasone
02	1.34	R↔T. Clinidipine
01	0.67	R ↔Inj..Dexamethasone
01	0.67	Inj. Streptomycin ↔Inj. Mannitol

CONCLUSION: Comorbidities and their treatment is causing significant burden by leading to treatment delay of failure. Strategies and programs to tackle burden of comorbidities on tuberculosis are to be developed and implemented.

REFERENCE:

1) Central TB division: TB India report 2018, Revised national TB control programme annual status report (https://tbcindia.gov.in>index1)

2) World Health Organization. (2017) Global tuberculosis report (WHO). Geneva, Switzerland (http://www.who.int/tb/publications/global_report/en/.)

A CASE REPORT ON FRIEDREICH ATAXIA IN A TERTIARY CARE TEACHING HOSPITAL

Meda Manjusai, B.Abishekar Reddy, D.Manjusha Reddy

INTRODUCTION:

FA was described in five papers by Nicholaus Friedreich over the period of 1863-1877 as FA is a debilitating, life-shortening, degenerative neuromuscular disorder. FA is a progressive neurodegenerative disorder affecting both children and adults. FA is usually manifested before adolescence (3). The mean age of onset in classical FA is between 10 and 16 years, late onset and very late onset FA develop after the ages of 25 and 40 years respectively . There is no sex predilection and it can affect both males and females . It is caused by severely reduced levels of frataxin followed by mutations in frataxin gene as a result of large GAA triplet repeat expansion within the first intron of the frataxin gene

CASE REPORT:

A female patient of age 19 years with chief complaints of weakness in both lower limbs, difficulty in walking since 3 ½ years and mild swelling while walking since 3 years, difficulty in climbing stairs. The patient is unable to stand and walk without support. The patient had scoliosis to the right side. Unlike usual presentation of friedreich ataxia, this patient had no difficulty in hearing, no visual abnormalities. Examination showed that the vitals are stable. Diagnosis of friedreich ataxia was done on the basis of patient history and examination as genetic conformation facilities were not available. Cranial nerves and fundus were normal. ECG [electro cardiogram] showed the T-wave abnormality with possible anterolateral ischemia. Thyroid function test, blood sugar profile, liver function tests, urine examination and complete blood profile were within normal limits. Patient was rehabilitated with physiotherapy to provide flexibility, strength and range of movement of muscles and aerobic exercises were practiced to avoid fatigue. The patient is on Cap.Rejunex (OD).

DISCUSSION: Friedreich ataxia is a highly disabling disease. Neurological features characteristically include progressive gait and limb ataxia, dysarthria, weakness, ocular fixation instability and deep sensory loss. Non- neurological involvement includes hypertrophic cardiomyopathy, diabetes mellitus and skeletal deformations such as kyphoscoliosis, pes cavus and pes equinovarus. Musculoskeletal complications are common in FRDA and scoliosis affects most patients. Bracing has not been shown to affect prognosis, it may help delay surgical correction in young children . Scoliosis has a prevalence of 63% to 100% in patients with FA. Males and females are equally affected. Scoliosis develops within a few years of onset of ataxia. FA is associated with high incidence of diabetes mellitus. 23% of FA patients were found to have diabetes and 4 developed diabetic ketosis terminally. Clinically apparent diabetes is seen in approximately 18% of affected individuals, while impaired glucose tolerance is present in upto 39% of FA patients . Younger age at onset and longer disease duration increase the risk of DM . The prevalence for diabetes among FA patients varies between 8 to 49%. Manifestation of diabetes is usually a late event in the course of FA (mean 15 years after onset). So the patients with FA should be regularly reassessed for DM and counselled regarding life style modifications. Ptosis is present in about 10% of patients. Vision is impaired in one fifth of patients. FA may even lead to blindness in late stages. Hearing problems are common and may worsen over time. Foot deformities may significantly interfere with mobility in 55 to 90% patients . There were no such complications in our patient. Over 20 potential therapeutics have been tested in clinical trials in FA patients, but no drug has been approved for this disease . No effective treatment for FA is available so far. Gene therapy and protein replacement strategies for FA are promising approaches for the future . Restoring reduced frataxin levels is an appropriate approach for slowing down or stopping FA. Physiotherapy provides an important means of maintaining balance, flexibility, strength and accuracy of limb movements. Aerobic exercises may help to improve weakness and fatigue. Rehabilitation may help counteract the effects of ataxia, weakness and spasticity in FRDA patients (4).

CONCLUSION: There is a need to identify the drugs that are likely to be effective for the treatment of FA. Early identification of the patients who are at risk of developing FA with related complications and providing proper therapeutic intervention by physiotherapy, aerobic exercises and rehabilitation at an earlier stage of the disease can reduce the disease progression.

REFERENCES:

1. Martin B D, Robert W, Susan M F. Friedreich ataxia: an overview. J Med Genet, 2000;37:1-8.

2. Ashley M, Jennifer F, Susan P et al. Impact of diabetes in the Friedreich ataxia clinical outcome measures study. Annals of Clinical and Translational Neurology, 2017;4(9):622-631.

DRUG PROFILE- DIROXIMEL FUMARATE

V .Vishnu Priya,
Pharm D Intern

Approved Date : October, 2019
Brand Name : Vumerity
Generic Name : Diroximel fumarate
Manufacturing Company : Biogen
Dosage forms, Strength: VUMERITY is available as hard, delayed-release capsules containing 231 mg of diroximel fumarate. The capsules have a white cap and a white body, printed with "DRF 231 mg" in black ink on the body.
Molecular Formula : C11H13NO6
Molecular Weight : 255.22

Mechanism
The mechanism by which diroximelfumarate exerts its therapeutic effect in multiplesclerosis is unknown.MMF, the active metabolite of diroximelfumarate, has been shown to activate the nuclear factor (erythroid-derived 2) like 2 (Nrf2) pathway invitro and invivo in animals and humans. The Nrf2 pathway is involved in the cellular response to oxidative stress. MMF has been identified as a nicotinic acid receptor agonist in invitro.

Adverse drug reactions:
The following important adverse reactions are described elsewhere in labeling:
- Anaphylaxis and Progressive Multifocal Leukoencephalopathy
- Lymphopenia
- Liver Injury
- Flushing

Storage and handling: Store at 20°C to 25°C (68°F to 77°F) Excursions permitted to 15°C to 30°C (59°F to 86°F)

Contraindications:
VUMERITY is contraindicated in patients with known hypersensitivity to diroximelfumarate, dimethyl fumarate, or toany of the excipients of VUMERITY. Reactions may include anaphylaxis and angioedema

Indication and usage:
VUMERITY is indicated for the treatment of relapsing forms of Multiple Sclerosis to include clinically isolated syndrome, relapsing –remitting disease and active secondary progressive diseases in adults

Pharmacokinetics:

Absorption

Peak plasma time	: 2.5-3 hr
Peak plasma concentration	: 2.11 mg/L
Mean steady-state AUC	: 8.32 mg·hr/mL

Distribution

The apparent volume of distribution for MMF is between 72 L and 83 L in healthy subjects after administration of VUMERITY. Human plasma protein binding of MMF is 27-45% and independent of concentration.

Protein bound : 27-45%

Metabolism

Extensively metabolized by esterases, which are ubiquitous in the gastrointestinal tract, blood, and tissues, to the major active metabolite, MMF, before it reaches the systemic circulation.

Further metabolism of MMF occurs through the tricarboxylic acid (TCA) cycle, with no involvement of the CYP450 system

Fumaric and citric acid, and glucose are the major metabolites of MMF in plasma

Esterase metabolism of diroximel fumarate also produces HES, an inactive major metabolite

Elimination:

MMF is mainly eliminated as carbon dioxide in the expired air with only trace amounts (less than 0.3% of the total dose) recovered in urine. The terminal half-life of MMF is approximately 1 hour, and accumulation of MMF does not occur with multiple doses of VUMERITY. HES is mainly eliminated in urine (58-63% of the dose was excreted as HES in urine). HES: Urine (58-63% of the dose)

Departmental Activities in November- 2019

Activities	Patient Counselling	Drug Information services	Adverse Drug Reactions	Medication Errors
Number	1058	157	12	02

Perfect Clicks

Campus Drive

Faculty attended a Two days work shop on Sencitizing Higher education Institutions in SVU

Workshop on Pharmacovigilance and Reporting of ADRs in SPMCW-SVIMS

World Diabetic Day

Swami Vivekananda Rathayathra in the occasion of 125th year celebrations of Chicago Speech