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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

CYSTIC FIBROSIS INDUCED DIGESTIVE DISEASE

K Karunakaran, Pharm D IV Yr

Background:

Cystic Fibrosis is an inherited Multi organ system disorder affecting children and increasingly in adult . It is most common life shortening genetic disease among whites and major cause of severe chronic lung disease. Disease generally manifest as mucosal obstruction of exocrine gland caused by defective Ion (cl) transport within the epithelial cell, Cystic fibrosis generally thought of as a lung disease because much of the associated morbidity and mortality is related to pulmonary complication Gastrointestinal complication have become cause of morbidity in patients with CF.

In the united State, CF most commonly occur in whites, approximately 1 in 3200 individuals, CF is less common in Hispanics of about 1 in 13,500 individual's ,in Asia 1 in 31,000 individual's and in African American of 1 in 15000.

Effect on GI System:

Gastrointestinal (GI) involvement often presents as meconium Ileus, small-bowel obstruction shortly after birth due to abnormally thick meconium. Older CF patients may develop distal intestinal obstruction syndrome (DIOS) due to fecal Impaction in the terminal ileum and Rectum.

Mal-digestion due to pancreatic enzyme insufficiency is present in 85% to 90% of CF patients. Thick pancreatic secretions and Cellular debris obstruct pancreatic and lead to fibrosis. Volume and concentration of pancreatic enzymes and bicarbonate are reduced, leading to mal-digestion of fat and protein and Subsequent mal-absorption of fat-soluble vitamins (A, D, E, and K).

Pathophysiology:

CF is a disease of exocrine gland epithelial cell where CFTR expression is prevalent. Normally, these cell transport chloride ion through the CFTR chloride channel with water and sodium accompanying the flux across the cell membrane.

In CF the CFTR chloride channel is dysfunctional and usually result in decreased secretion of chloride ion and increased absorption of sodium ion lead to altered viscosity of fluid secreted by exocrine gland and mucosal obstruction.

Symptoms:

• Pulmonary: Chronic cough, sputum production, decreased Exercise tolerance, and recurrent pneumonia and sinusitis. Exacerbations may be marked by increased cough, sputum Changes (darker, thicker), hemoptysis, dyspnea, and fever.

• GI: Numerous large, foul-smelling loose stools (steatorrhea), Flatulence, and abdominal pain. Intestinal obstruction may present as abdominal pain and distention and/or decreased Bowel movements.

• Nutritional: Poor weight gain despite voracious appetite and Hunger. Dry skin, skin rash, and visual disturbances may be noted in vitamin deficiency.

• CFRD: Weight loss, increased thirst, and more frequent Urination.

Signs:

• Obstructive airways disease: Tachypnea, dyspnea, cyanosis, Wheezes, crackles, sterna retractions, digital clubbing, and Barrel chest.

• Failure to thrive: Below age-based normal of both height and weight in children; adults may be near/below ideal body Weight or have a low body mass index (BMI).

• Salty taste to the skin.

• Hepatobiliary disease: Hepatomegaly, splenomegaly, and prolonged bleeding may occur.

• Recurrent pancreatitis (usually in pancreatic-sufficient Patients): Episodic epigastric abdominal pain, persistent

Laboratory findings:

• Hepatobiliary disease: Serum aspartate aminotransferase, Alanine aminotransferase, alkaline phosphatase, glutamyltransferase, and bilirubin may be elevated.

• Leukocytosis with increase in polymorphonuclear (PMN) Leukocytes and bands may occur in acute pulmonary Exacerbations

• Mal-digestion: Decreased serum levels of fat-soluble Vitamins (A, D, E, and K). Decreased vitamin K levels• CFRD: Blood glucose 200 mg/dL (11.1 mmol/L) or higher 2 hours after an oral glucose-tolerance test or fasting hyperglycemia (fasting blood glucose 126 mg/dL [7 mmol/L] or more regardless of the post-glucose challenge level).

• Hepatobiliary disease: Serum aspartate aminotransferase, may Result in elevated prothrombin time (PT)

• CFRD: Blood glucose 200 mg/dL (11.1 mmol/L) or higher 2 hours after an oral glucose-tolerance test or fasting Hyperglycemia (fasting blood glucose 126 mg/dL [7 mmol/L] .

Gastrointestinal Therapy:

Pancreatic Enzyme Replacement:

This is the mainstay of GI therapy. Most enzyme products available in the United States are formulated as capsules containing enteric-coated Microspheres or micro tablets that escape enzyme inactivation by gastric acid and promote dissolution in the more alkaline duodenum. Capsules may be opened and the micro beads swallowed with food (for infants and young Children), as long as they are not chewed or mixed with alkaline or hot foods (which denature enzymes). Although products may contain similar enzyme ratios, they are not bioequivalent and cannot be interchanged. Pancreatic enzymes are initiated at 1000 units/kg/meal of Lipase component (because fats are most difficult to digest) with Half-doses given for snacks. Enzymes should be taken at the Beginning or divided throughout the meal and must be given with any fat-containing snack. Infants are typically started at 1500 to 2500 units of lipase/120 mL with breast milk

COMMON PANCREATIC ENZYME REPLACEMENT PRODUCTS

1 CREON 3000 (contain lipase 3000 unit, amylase 15000 unit, protease 9500 unit)

2 PANCREAZE MT 2a

3 PERTZYE 4000

4 VIOKACE

Liver Disease

For Liver disease Ursodiol at 20 mg/kg/day in two divided doses May slow progression of liver disease. It improves bile flow and May displace toxic bile acids that accumulate in a cholestatic liver, Stimulate bicarbonate secretion into the bile, offer a cytoprotective Effect, and reduce elevated liver enzymes.

Intestinal Obstruction Treatment of DIOS consists of enteral Administration of polyethylene glycol (PEG) electrolyte solutions. Severe presentations may require surgical resection. Enemas May also be used to facilitate stool clearance. IV fluids are often required to correct dehydration due to vomiting or decreased Oral intake. Re-evaluation of enzyme adherence and dosing is Essential to prevent recurrence, and some patients may require Daily PEG administration

Gastrointestinal Function

• Monitor short- and long-term nutritional status through Evaluation of height, weight, and BMI. Ideally, parameters should be near the normal (50^{th} percentile) for non-CF Patients.

• Evaluate the patient's stool patterns. Steatorrhea indicates suboptimal enzyme replacement or noncompliance. Infants should have two to three well formed stools daily, whereas older children and adults may have one or two stools daily

• Monitor efficacy of vitamin supplementation through yearly Serum vitamin levels. Obtain levels more frequently if treating a deficiency.

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SCOPING REVIEW OF COCHLEAR IMPLANTATION IN SUSAC'S SYNDROME

K Hema Sruthi, Pharm D IV yr

Introduction:

Susac syndrome(s) is a microangiopathy that affects the brain, retina and cochlea, classically presenting with the clinical trial of encephalopathy, branch retinal artery occlusion and sensorineural hearing loss (SNHL) first described by John susac in 1979. He first reported two cases of brain and retinal vasculopathy in female patient with good response to corticosteroids in 1979. It is a rare syndrome and, until now slightly more than 300 cases have been reported in the literature. Susac syndrome is frequently seen in females of 20-40 years of age. It usually presents with severe headache and behavioural changes progressive cognitive decline, apathy and later by hearing loss, tinnitus and segmental visual loss. The clinical trial of sub acute encephalopathy, BRAO and hearing loss is due to a precapillary arteriolar angiopathy of unknown origin, but most evidences are in favour of an immune mediated endotheliopathy and immunosuppressive therapy is the main mode of treatment.

Epidemiology:

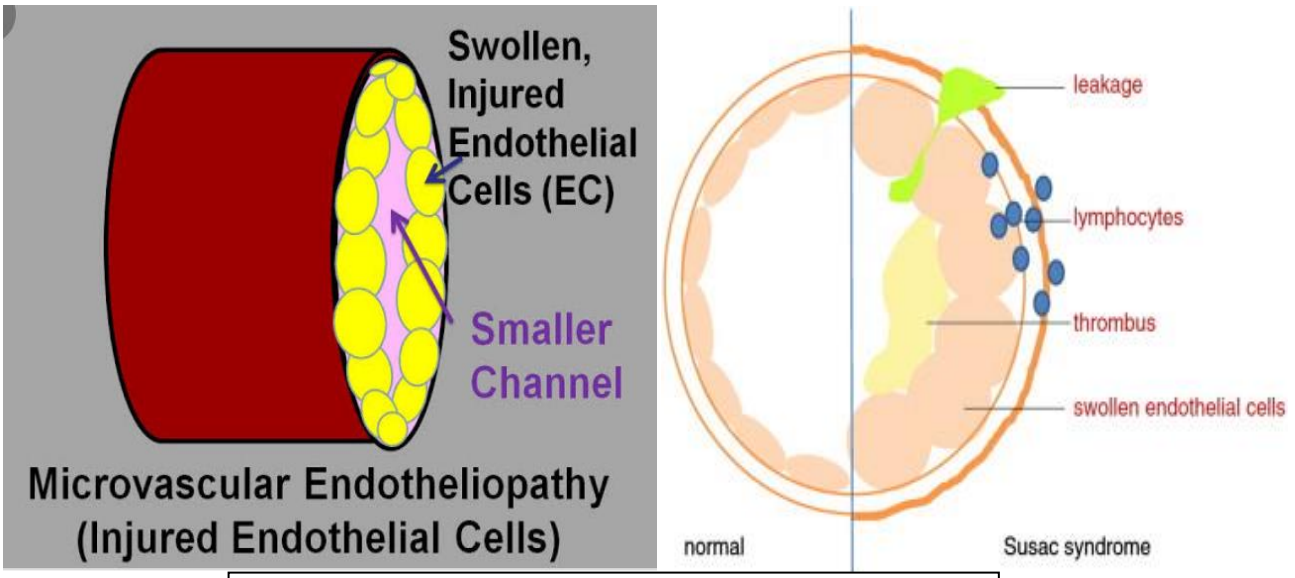
Susac syndrome is frequently seen in women with female to male ratio of 3:1, but most of the patients are 20- 40 years old. The overall incidence of susac syndrome is unknown and there are limited case reports (more than 300 cases) of this syndrome in the literature.

Clinical presentation:

Usually, there is no complete clinical triad at presentation. The encephalopathic syndrome may be heralded by severe and sometimes migrainous headache. Encephalopathy begins with behavioural abnormalities such as aggressiveness, paranoia, depression, and then memory loss with confabulation, viscuospatial deficits, diminished attention and concentration, disorientation, lethargy and apathy may occur. This abnormal mental status sometimes wax and wane. During this period, urinary incontinence and generalised seizure may happen. In many of the reported cases of susac syndrome, sudden SNHL in association with peripheral vertigo, nystagmus and tinnitus occur after the encephalopathic features. The low and medium frequency SNHL occurs as a complication of cochlear apex arteriolar micro infarctions, may be permanent, and if severe, needs cochlear implantation. The clinical presentation of susac syndrome in BRAO which means occlusion of some branches of the retinal artery due to endothelial injury. BRAO can lead to bilateral vision loss or remain asymptomatic, depending on the location of retinal involvement, BRAO can also cause photopia, black spots and scintillating scotomas, ischemic retinal whitening resulting from BRAO is the most common finding on fundoscopy. Other findings are cotton wool spots, tiny peripheral haemorrhages, and oedematous retina and glass plaques. Glass plaques are first described by DON Glass in patients with idiopathic BRAO, are yellow - white lipid deposits at the mid segment of the retinal arterioles. These are caused by slow extravasations of blood lipids into the arteriolar wall at the sites of arteriolar wall damage glass plaques are typically located at the mid-segment of the retinal arterioles

Pathogenesis:

It is a RARE IMMUNE-MEDIATED DISEASE. Susac syndrome is an uncommon immune- mediated disease affecting the eyes, ears and brain in a triad of vision loss, hearing loss and encephalopathy.



Pathogenesis of SUSAC Syndrome

Treatment:

There is no general consensus on treatment of susac syndrome and because of its rarity. Based on their clinical experience and case reports guiding current recommendations. Phases of treatment can be divided into initial therapy, maintenance therapy, and therapy for relapses. Treatment directed at the CNS squeal of this disease will usually adequately treat retinal and vestibule-cochlear manifestations of susac syndrome.. The most commonly recommended initial therapy is intravenous methylprednisolone 1g daily for 3-10 days followed by high dose prednisolone, 1mg/kg/day up to 80 mg PO daily tapered over a period of weeks, in addition to IVIG tapered over six months. Common maintenance therapies include cyclophosphamide, mycophenolate mofetil, tacrolimus, and rituximab, with varying use depending on severity of disease, commonly continued for a period of two years. Relapses may be treated with additional pulses of IV methylprednisolone and, in severe cases, plasma exchange. Monitoring of disease is recommended with interval MRI brain, fluoroscopic angiography, and audiogram. Intratympanic steroid injection for SNHL has been performed, with mixed results reported in case studies. One of our presented patients had no improvement with three rounds of IT dexamethasone. Rehabilitation of hearing loss should be addressed with amplification when appropriate and an understanding of the potential for progression of SNHL. Cochlear implantation has provided significant speech perception benefit in all published cases.

Conclusion:

A rare microvasculopathy that affects the brain, retina, and inner ear, susac syndrome presents both a diagnostic and therapeutic challenge that requires multidisciplinary evaluation and treatment. The paucity of cases and absence of prospective studies in susac syndrome result in a reliance on clinical experience and expert opinion. The SNHL seen in susac syndrome presents with variable severity and progression. In cases of significant bilateral loss with limited benefit from hearing aid amplification, cochlear implantation has been shown to successfully improve speech understanding in all published cases so far and is a responsible means of rural rehabilitation in the setting of susac syndrome.

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JEMPERIL (dostarlimab-gxly) INJECTION – A NEWLY APPROVED DRUG FOR`` ENDOMETRIAL CANCER"

B.Dinesh, Pharm-D 3rd Year

BRAND NAME : **JEMPERIL**

GENERIC NAME : **Dostarlimab- gxly**

MOLECULAR FORMULA : **C_{6420} H_{9832} N_{1690} O_{2014} S_{44}**

DRUG CLASS : **Antineoplastic**

MANUFACTURING COMPANY : **GlaxoSmithKline**

DATE OF APPROVAL : **April 22, 2021**

Dosage Form & Strength :

Injection: 500mg/10mL (50mg/mL) clear to slightly opalescent, colourless to yellow solution in a single-dose vial for intravenous infusion.

Indication :

Jemperil is indicated for the treatment of adult patients with mismatch repair deficient (**dMMR**) recurrent or advanced **endometrial cancer.**

Mechanism Of Action :

Dostarlimab is a monoclonal antibody targeted against Programmed death receptor – 1(**PD-1**) - it binds to the receptor and prevents interactions with **PD-L1** and **PD-L2**, thus allowing the anti-tumor immune response to proceed unimpeded.

Adverse Drug Reaction :

Fatigue/Asthenia, Nausea, Diarrhea, Constipation, Vomiting, Anemia, Urinary tract infection, Myalgia, Cough, Puritius, Decreased Appetite.

Drug Interaction :

The severity of adverse effects can be increased when **Anthrax immune globulin human** is combined with Dostarlimab. The severity of adverse effects can be increased when **Asfotase alfa** is combined with Dostarlimab

Pharmacokinetics :

Absorption :

During the first cycle, and administered at 500mg intravenously every 3 weeks, the mean C_{max} and $AUC_{0\text{-}tau}$ of dostarlimab-gxly are 171 mcg/mL and 35,730 mcg.h/mL, respectively. When administered at 1000mg every 6 weeks, the mean C_{max} and $AUC_{0\text{-}tau}$ are 309 mcg/mL and 95,820 mcg.h/mL, respectively.

Distribution :

The mean (%CV) volume of distribution of dostarlimab-gxly at steady state is 5.3 L (12%).

Metabolism :

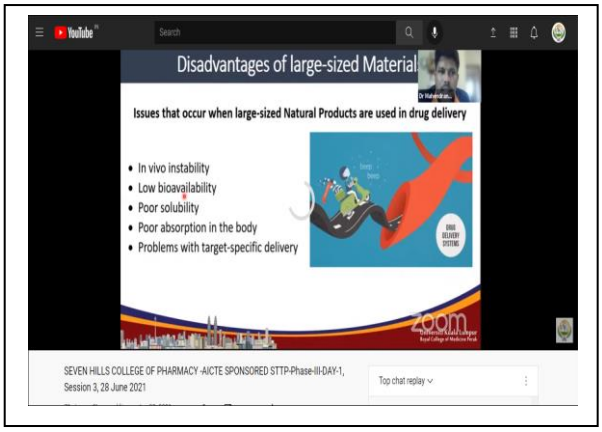
Dostarlimab-gxly is expected to be metabolized into small peptides and amino acids by catabolic pathways.

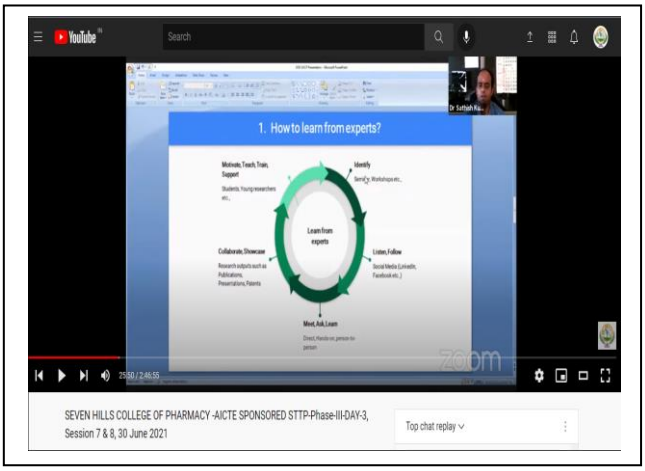
Elimination :

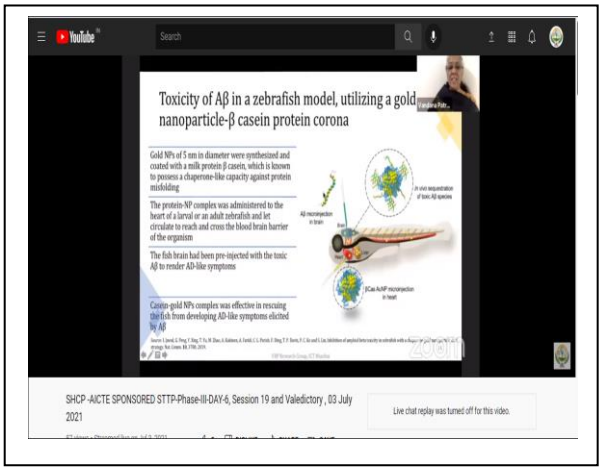
The mean terminal elimination half-life of dostarlimab-gxly is 25.4 days and its mean (%CV) clearance is 0.007 L/h (31%) at steady state.

Perfect Click

Yoga Day Celebrations-2021







Glimpses of AICTE Sponsored STTP Phase III organized of SHCP