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TREATMENT



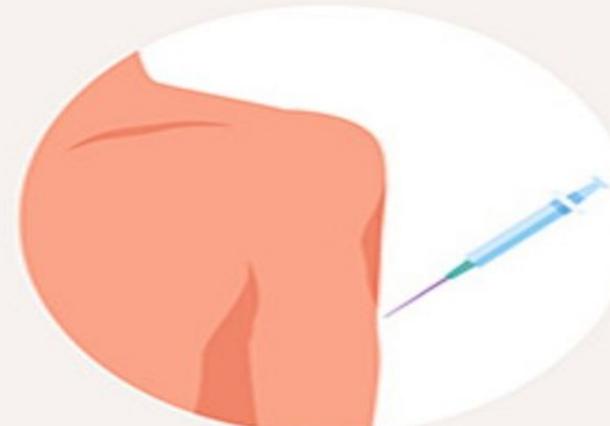
Diabetes
Medications



Blood Sugar
Monitoring

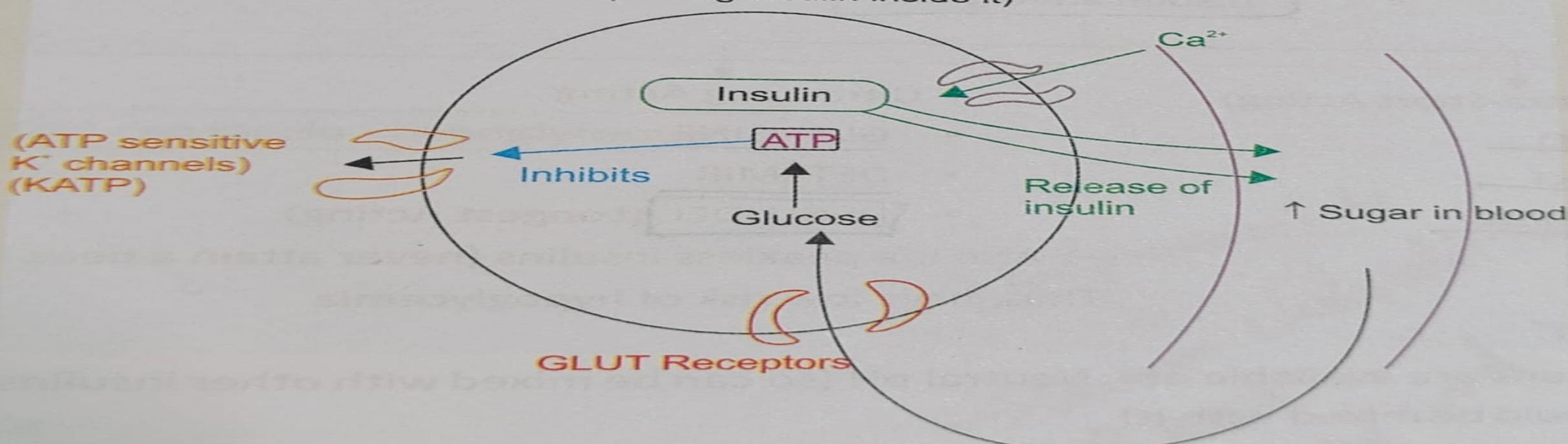


Insulin Pills



Insulin
Injections

Increase in blood glucose
↓
Enter beta cells through GLUT receptors
↓
Glucose is metabolized to form ATP
↓
ATP blocks ATP sensitive K^+ channels
↓
Increased K^+ inside cell leading to slight depolarization
↓
Opening of calcium channels
↓
Influx of calcium leads to depolarization and release of insulin
 β cell (storing insulin inside it)



INSULIN ;

- INDICATIONS ;**

1. All patients of type 1 DM
2. Uncontrolled patients of type 2 DM
3. Diabetes in pregnancy
4. Diabetic ketoacidosis
5. Acute hyperkalemia

ROUTES OF ADMINISTRATION;

- **SUB-CUTANEOUS ROUTE:**

- MC route (because self-administration is possible with this route)
- All insulin preparations can be given by subcutaneous route.

- **SITE OF ADMINISTRATION**

- . Entire abdomen except area around umbilicus(thickness of skin is not uniform, so insulin absorption is affected)

- . Anterior thigh

- . Lateral thigh

- . Arm

. Intravenous route:

- Only REGULAR INSULIN can be given
- So, insulin of choice in diabetic ketoacidosis

. Inhalational route:

Exubera – withdrawn from market because of pulmonary complications.

#Afreeza – short acting – so should be given before every meal

PREPARATIONS OF INSULIN

- Short acting – Regular, semi-lente
- Intermediate acting – NPH(neutral protamine Hagedorn), lente
- Long acting – ultra lente
- Lente (insulin with Zn combination (70% ultra lente+30% semi lente)
- Zn (Stabilizes the hexameric form of insulin to make it long acting)

INSULIN ANALOGUES

- RAPID ACTING (Ultra short acting)
 - Lispro
 - Aspart
 - Glulisine

*ULTRA LONG ACTING

- Glargine
- Detemir
- Degludec(long acting) – also k/a peakless insulin thus, have low risk of hypoglycemia

SIDE EFFECTS

1. Hypoglycemia

- MC side effect
- Most dangerous S/E
- S/E which can be easily prevented
- Advise to patients for prevention

* Do not skip meals

*Keep glucose

2. Hypokalemia

3. Lipoatrophy (results from repeated injections at the same site)

ORAL ANTIDIABETICS DRUGS

Act by increase insulin (insulin secretagogues)

S/E hypoglycemia

>30% functional beta cells should be present

Act by other mechanisms

no hypoglycemia

no such requirement

1. Insulin secretagogues (act by increasing insulin secretion)

These drugs secrete insulin by inhibiting ATP sensitive K⁺ channels.

SULONYLUREAS	MEGLITINIDES
1 st Generations	Nateglinide
Chlorpropamide Tolbutamide	Repaglinide
2 nd Generations	
Glipizide Gliclazide Glibenclamide	

- **Sulphonylureas**

- Cause hypoglycemia
- Cause weight gain

- **Meglitinides**

- Short acting (work for – 1hr)
- Indicated in post prandial hyperglycemia

DRUGS ACTING BY OTHER MECHANISMS

1. AMP kinase stimulators

*Group k/a biguanides

- Metformin
- Phenformin

* Biguanides act by activating an enzyme AMP kinase that result in

- Gluconeogenesis(-)
- Glycogenolysis (-)
- Glycogenesis (+)
- Glycolysis (+)

*These drugs do not release insulin – so do not cause hypoglycemia

*S/E of biguanides

- Megaloblastic anemia (more with metformin)
- Lactic acidosis (more with phenformin)

*Biguanides C/I

- liver disease (gluconeogenesis cannot occur)
- renal disease (lactic acid produced cannot be excreted)
- Indications;
 - Metformin is DOC for T2DM
 - No risk of hypoglycemia
 - Max. reduction in HbA1c
 - M – metformin preferred in
 - O – obese pts
 - S – sulfonylureas are preferred in
 - T – thin patients
- Can cause weight loss or weight neutral
- Metformin is also indicated for POCD

***GLITAZONES**

- Rosiglitazone
- Pioglitazone

MOA

- Act by stimulating PPAR gamma (reversal of insulin resistance)

ADVERSE EFFECT

- Hepatotoxic
- Rosiglitazone & pioglitazone required LFT monitoring
- Predispose to osteoporotic fractures
- Risk of MI
- Risk of urinary bladder carcinoma by pioglitazone

***Alpha glucosidase inhibitors**

- Act by inhibiting the intestinal absorption of carbohydrates

***Drugs**

- Acarbose
- Voglibose

***Flatulence –MC side effect**

- C/I – Inflammatory bowel disease
 - UC
 - Crohn's disease

- **INCRETIN**

- Incretins are normal physiological substance which are released in GIT after food intake that stimulate the release of insulin.
- Most important endogenous incretins are GLP (glucose like peptide) & GIP (Glucose stimulated insulinotropic polypeptide)

- **Functions of incretins ;**

- Increase insulin release
- Decrease gastric motility
- stimulate satiety centre of brain

-GLP is metabolized by DPP-4 & becomes inactive

***GLP analogues**

- Exenatide
- Liraglutide
- Semaglutide

***Advantages**

- Cause weight loss
- Do not cause hypoglycemia

***S/E**

- Acute pancreatitis
- Nausea

- **DPP-4 inhibitors:**

- Sitagliptin
- Vildagliptin
- Linagliptin
- Saxagliptin

***S/E**

- Nasopharyngitis
- Pancreatitis

***C/I**

- Renal failure except linagliptin which is safe

***SGLT-2 inhibitors**

Glucose is freely filtered by glomerulus but the clearance of glucose in urine is negligible.

- Because the reabsorption takes place in PCT by SGLT-2
- SGLT-2 inhibitors stop the reabsorption of glucose in PCT resulting in glucosuria

DRUGS;

- Canagliflozin**
- Dapagliflozin**
- Empagliflozin**

S/E:

-Most common side effect of SGLT-2 inhibitors is Urinary tract infections (urosepsis) & genital tract infections (Fournier's gangrene)

SGLT-2 inhibitors provide cardiovascular benefits thus it has been approved for the treatment of CHF

**MANAGING YOUR
DIABETES
IS NOT A SCIENCE,**

**IT IS AN
ART**

THANK YOU

