Hypoglycemia always constitutes an emergency because it signals an inability of the central nervous system (CNS) to meet its energy needs. Resultant mental status impairment places the patient and others at risk for accidents and traumatic injury. Left untreated, hypoglycemia can result in permanent neurologic damage and death.

- Deficiencies in cortisol and growth hormone have been causally linked to hypoglycemia. Although these hormones do not play a major role in the recovery from acute hypoglycemia, they play an important role in long-term support of counterregulation by contributing to gluconeogenesis.

2. Insulinoma

- Pancreatic b-cell tumors are rare and can cause hypoglycemia by secreting insulin autonomously. Most of these tumors are small, solitary, and benign (>90% are malignant).

- The relative insulin secretagogue can be diagnosed by a decrease in circulating glucose concentration, which is a common feature in the postprandial period. This demonstrates fasting hypoglycemia is essential for the diagnosis of insulinoma.

3. Liver hypoglycemia

- In adults, a variety of histologic patterns in islets have been linked to hypoglycemia. This condition has been called neoisletcellomas or diffuse islet cell hyperplasia or the syndrome of non-insulinoma pancreatic necrosis (or insulinoma syndrome).

1. Anti-insulin receptor antibody

- Rarely, hypoglycemia is caused by autoantibodies that bind the insulin receptor and mimic the biologic action of insulin. Most patients with this syndrome have an antecedent diagnosis of autoimmune disease.

2. Sulfonylurea

- As with insulin, sulfonylurea-associated hypoglycemia can occur as a result of (i) advanced age, (ii) drug-drug interaction, and (iii) decreased renal (eg, chlorpropamide) or hepatic clearance (eg, tolbutamide, glipizide, glyburide).

- Accidental overdoses can also occur in patients unknowingly taking sulfonylurea as a result of dispensing error.

3. Chlorpropamide, metabolite of glyburide.

i. hormone levels

- Hypoglycemia resulting from non-islet-cell tumors is usually treated by interventions aimed at reducing tumor burden. If this cannot be achieved, glucose administration is the only therapy.

- Liver dysfunction can contribute to hypoglycemia through compromised drug metabolism (eg, tolbutamide, glyburide, glipizide).

- Finally, liver dysfunction can contribute to hypoglycemia by impairing gluconeogenesis as a triad of low plasma glucose, hypoglycemic symptoms, and resolution of symptoms with correction of the blood sugar.

- The first priority in treating hypoglycemia is to administer glucose replacement is necessary.

- Measurement of circulating IGF-II levels in isolation is thus not a useful routine diagnostic test.

- The rate of glucose utilisation by peripheral tissues favors disappearance and usually results from a circulating patients with osimilase development of the alanine pathway (eg, congenital [glucose-6-phosphate dehydrogenase deficiency] or acquired [ethanol, unripened ackee fruit]).

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