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DIABETTYPE 1

Dr. arefnia

Pediatric Endocrinologist

Assistant Prof. of golestan University Of Medical

Diabetes Mellitus

- type 1 DM (T1D),
- autoimmune destruction of the insulin-producing ß cells (islets) of the pancreas,
- leading to permanent insulin deficiency.

- Type 2 DM (T2D)
- insulin resistance and relative insulin deficiency usually in the context of exogenous obesity.

Definition

glucose abnormalities that may need to be confirmed by repeat testing:

- Fasting serum glucose concentration ≥126 mg/dL
- random venous plasma glucose ≥200 mg/dL with symptoms of hyperglycemia,
- an abnormal oral glucose tolerance test (OGTT) with a 2- hour postprandial serum glucose concentration ≥200 mg/dL
- HgbA1c ≥6.5%

SPORADIC HYPERGLYCEMIA

- can occur in children, usually in the setting of an intercurrent illness.

- When the hyperglycemic episode is clearly related to an illness or other physiological stress, the probability of incipient diabetes is small (<5%).`

PATOPHISIOLOGY

- Insulin deficiency usually first causes postprandial hyperglycemia and then fasting hyperglycemia.
- Ketogenesis is a sign of more complete insulin deficiency.
- Lack of suppression of gluconeogenesis and glycogenolysis further exacerbates hyperglycemia,

.

PATOPHISIOLOGY

- fatty acid oxidation generates the ketone bodies: ß-hydroxybutyrate, acetoacetate, and acetone.

- Protein stores in muscle and fat stores in adipose tissue are metabolized to provide substrates for gluconeogenesis and fatty acid oxidation

PATOPHISIOLOGY

- Glycosuria

  occurs when the serum glucose concentration exceeds the renal threshold for glucose reabsorption (from 160 to 190 mg/dL).

- Glycosuria causes an osmotic diuresis (including obligate loss of sodium, potassium, and other electrolytes) leading to dehydration..

Clinical Manifestations

- Polydipsia

occurs as the patient attempts to compensate for the excess fluid losses.

Weight loss

results from the persistent catabolic state and the loss of calories through glycosuria and ketonuria.

- The classic presentation of T1D includes polyuria, polydipsia, polyphagia, and weight loss

Diabetic Ketoacidosis

- . patients with known T1D if insulin injections are omitted

- during an intercurrent illness when greater insulin requirements are unmet in the presence of elevated concentrations of the counter-regulatory and stress hormones

(glucagon, growth hormone [GH], cortisol, and catecholamines).

Diabetic Ketoacidosis

- the arterial pH is below 7.3,
- the serum bicarbonate level is below 15
- mEq/L,
- Bohbutyrat >3 mmol/l
- ketones are elevated in serum or urine
- BS>200mg/dl

CLINICAL SIGNS OF DKA

- Dehydration,
- tachycardia,
- tachypnea,
- deep sighing respiration,
- breath smells of acetone,
- nausea and/or vomiting,
- abdominal pain,
- blurry vision,
- confusion,
- drowsiness,
- progressive decrease in level of consciousness
- loss of consciousness (coma

PATHOPHYSIOLOGY

- In the absence of adequate insulin secretion, persistent partial hepatic oxidation of fatty acids to ketone bodies occurs.
- Two of these three ketone bodies are organic acids and lead to metabolic acidosis with an elevated anion gap.
- Lactic acid may contribute to the acidosis when severe dehydration results in decreased tissue perfusion.

PATHOPHYSIOLOGY

- Hyperglycemia causes an osmotic diuresis that is initially compensated for by increased fluid intake.
- As the hyperglycemia and diuresis worsen, most patients are unable to maintain the large fluid intake and dehydration occurs.
- Vomiting and increased insensible water losses caused by tachypnea can worsen the dehydration

PATHOPHYSIOLOGY

- As hydrogen ions accumulate as a result of ketoacidosis, intracellular potassium is exchanged for hydrogen ions.

- Serum concentrations of potassium increase initially with acidosis then decrease as serum potassium is cleared by the kidney.

PATHOPHYSIOLOGY

Depending on the duration of ketoacidosis, serum potassium concentrations at diagnosis may be

- ✓ increased,
- ✓ normal,
- ✓ decreased,

but intracellular potassium concentrations are depleted

PATHOPHYSIOLOGY

- . A decreased serum potassium concentration is an ominous sign of total body potassium depletion.

Phosphate depletion also can occur as a result of the increased renal phosphate excretion required for elimination of excess hydrogen ions.

Sodium depletion is also common in ketoacidosis resulting from renal losses of sodium caused by osmotic diuresis and from gastrointestinal losses from vomiting

PATHOPHYSIOLOGY

- Respiratory compensation for acidosis results in tachypnea with deep (Küssmaul) respirations.
- The fruity odor of acetone frequently can be detected on the patient's breath.
- An altered mental status can occur, ranging from disorientation to coma
- .

Pathophysiology

- Abdominal pain occurs frequently and can mimic an acute abdomen. The abdomen may be tender from vomiting or distended secondary to a paralytic ileus.

Pathophysiology

- Once glucose goes below 180 mg/dL (10 mmol/L), the osmotic diuresis stops and rehydration accelerates without further increase in the infusion rate.

LABORATORY

- BUN can be elevated with prerenal azotemia secondary to dehydration.
- The WBC is usually elevated and can be left shifted without implying the presence of infection.
- Fever is unusual and should prompt a search for infectious sources that may have triggered the episode of DKA.

Laboratory

- The serum sodium concentration is an unreliable measure of the degree of ECF contraction for two reasons:
- (1) glucose, largely restricted to the extracellular space, causes osmotic movement of water into the extracellular space thereby causing dilutional hyponatremia,
- (2) the low sodium content of the elevated lipid fraction of the serum in DKA.

Laboratory nelson 2020

- The magnitude of this effect can be calculated as follows: [ ] Na corrected = + [ ] Na measured 1 6. (× − [ ] glucose m 100 g/dL)/100 where [Na]measured = sodium concentration measured by the clinical laboratory and [Na]corrected = corrected sodium concentration

Risk factors for DKA

- Risk factors for DKA in newly diagnosed patients include

- younger age,
- delayed diagnosis,
- lower socioeconomic status,
- residence in a country with a low prevalence of type 1 diabetes mellitus (T1DM)

In children with established diabetes

- The risk of DKA in established type 1 diabetes is 1% to 10% per patient per year

- • Children who omit insulin
- Children with poor metabolic control or previous episodes of DKA •
- Gastroenteritis with persistent vomiting and inability to maintain hydration •
-

In children with established diabetes

- Children with psychiatric disorders, including those with eating disorders •
- Children with difficult or unstable family circumstances (eg, parental abuse) •
- Peripubertal and adolescent girls •
- Binge alcohol consumption
- Children with limited access to medical services

MANAGEMENT OF DKA

- careful replacement of fluid deficits,
- correction of acidosis and hyperglycemia via insulin administration,
- correction of electrolyte imbalances,
- monitoring for complications of treatment.

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MANAGEMENT OF DKA

- There should be documentation on a flow chart of hour-by-hour clinical observations, IV and oral medications, fluids, and laboratory results.

MANAGEMENT OF DKA

- Experienced nursing staff trained in monitoring and management of DKA in children and adolescents ·

- Written guidelines or, if unavailable, access to online guidelines for DKA management in children ·

Principles of water and salt replacement

- Calculation of fluid deficits using clinical signs is difficult in children with DKA because intravascular volume is better maintained in the hypertonic state.
- For any degree of tachycardia, delayed capillary refill, decreased skin temperature, or orthostatic blood pressure change,

- the child with DKA will be more dehydrated than the child with a normotonic fluid deficit. .

Monitoring should include the following:

- Hourly (or more frequently as indicated) vital signs (heart rate, respiratory rate, blood pressure) ·

- Hourly (or more frequently as indicated) neurological observations (Glasgow coma score; for warning signs and symptoms of cerebral edema

Principles of water and salt replacement

- Resuscitation fluids For patients who are volume depleted but not in shock,
- begin immediately with 0.9% saline to restore the peripheral circulation. The volume administered typically is 10 mL/kg infused over 30 to 60 minutes;
- however, if tissue perfusion is poor the initial fluid bolus is given more rapidly (eg, over 15-30 minutes) and a second fluid bolus may be needed to ensure adequate tissue perfusion.

Principles of water and salt replacement

- In the rare patient with DKA in shock, rapidly restore circulatory volume with isotonic saline in 20 mL/kg boluses infused as quickly as possible through a large bore cannula with reassessment of circulatory status after each bolus.

Principles of water and salt replacement

- Ongoing losses resulting from osmotic diuresis usually do not need to be replaced unless urine output is large or signs of poor perfusion are present.
- Osmotic diuresis is usually minimal when the serum glucose concentration decreases to less than 300 mg/dL.
- To avoid rapid shifts in serum osmolality, 0.9% sodium chloride can be used as the replacement fluid for the initial 4-6 hours, followed by 0.45% sodium chloride.

Principles of water and salt replacement

- The remaining fluid deficit after the initial bolus should be added to maintenance fluid requirements, and the total should be replaced slowly over 36-48 hours

MAINTENANCE REQUIREMENTS

- Average (range) losses per kg 24-hour maintenance requirements Water
- ≤10 kg 100 mL/kg/24 h
- 11-20 kg 1000 mL + 50 mL/kg/24 h for each kg from 11 to 20
- >20 kg 1500 mL + 20 mL/kg/24 h for each kg >20

Principles of water and salt replacement

IV rate mL kg maintenance bolus hr =

85ml/kg +maintenance-bolus:23 h

Principles of water and salt replacement

- Sample calculation for a 30-kg child: 1st hr = 300 mL IV bolus 0.9% NaCl or LR

- 2nd and subsequent =(85ml *30)+1750ml-300ml:23
- =175ml/h

Principles of water and salt replacement

If K <3 mEq/L, give 0.5-1.0 mEq/kg as oral K solution or increase IV K to 80 mEq/L

Oral intake with subcutaneous insulin

No emesis; CO_2 ≥16 mEq/L; normal electrolytes

Principles of water and salt replacement

- *The sodium content of the fluid should be increased if measured serum sodium concentration is low and does not rise appropriately as the plasma glucose concentration* falls.

Principles of water and salt replacement

- The use of large amounts of chloride-rich fluids (combined with preferential renal excretion of ketones over chloride) may be associated with the rapid development of hyperchloremia
- (defined as a ratio of chloride:sodium [Cl^- :Na^+] > 0.79) and hyperchloremic metabolic acidosis.

Principles of water and salt replacement

- When hyperchloremia develops, a persisting base deficit or low bicarbonate concentration can be erroneously interpreted as being due to ongoing ketosis.

- To avoid this misinterpretation, measurement of bedside BOHB levels will prevent any confusion and can demonstrate that ketoacidosis has resolved.

- Hyperchloremic acidosis resolves spontaneously.

Insulin therapy

- Although rehydration alone frequently causes a marked decrease in blood glucose concentration, insulin therapy is essential to restore normal cellular metabolism, to suppress lipolysis and ketogenesis, and to normalize blood glucose concentrations.

- So-called low dose IV insulin administration is safe and effective.

Insulin therapy

- Start insulin infusion at least 1 hour after starting fluid replacement therapy; that is, after the patient has received initial volume expansion
- Correction of insulin deficiency • Dose: 0.05 to 0.1 unit/kg/h (eg, one method is to dilute 50 units regular [soluble] insulin in 50 mL normal saline, 1 unit = 1 mL)

- Route of administration IV

Insulin therapy

- An IV bolus should not be used at the start of therapy; it is unnecessary,
- may increase the risk of cerebral edema,
- can precipitate shock by rapidly decreasing osmotic pressure,
- can exacerbate hypokalemia

Insulin therapy

- The dose of insulin should usually remain at 0.05 to 0.1 unit/kg/h at least until resolution of DKA (pH >7.30, serum bicarbonate >15 mmol/L,

- Monitor venous pH and serum BOHB concentration every 2 hours to ensure steady improvement of biochemical parameters

Insulin therapy

- If the patient shows marked sensitivity to insulin (eg, some young children with DKA, patients with HHS, and some older children with established diabetes),
- the insulin dose may be decreased provided that metabolic acidosis continues to resolve.
- For example, if a young child is receiving 0.05 unit/kg/h, it may be necessary to reduce the insulin dose to 0.03 unit/kg/h to prevent hypoglycemia despite the addition of IV glucose.

INSULIN THERAPY

- For less severe DKA (pH >7.1-7.2), 0.05 U/kg/h (0.03 U/kg/h for age<5years
- • Insulin has an aldosterone-like effect leading to increased urinary potassium excretion.
- High doses administered intravenously for a prolonged period of time may contribute to a decrease in serum potassium concentration due to increased urinary potassium excretion despite potassium administration. •
- Time on IV insulin infusion and dose of insulin should be minimized to avoid severe hypokalemia

INSULIN THERAPY

- During initial volume expansion the plasma glucose concentration falls steeply.

- Thereafter, and after commencing insulin therapy, the plasma glucose concentration typically decreases at a rate of 36 to 90 mg/dl/h, depending on the timing and amount of glucose administration

- Serum glucose concentrations should decrease at a rate no faster than 100 mg/dL/hr.

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Insulin therapy

- To prevent an unduly rapid decrease in plasma glucose concentration and hypoglycemia,
- 5% glucose, initially, should be added to the IV fluid when the plasma glucose falls to approximately (250-300 mg/dL),
- or sooner if the rate of fall is precipitous

Insulin therapy

- It may be necessary to use 10% or even 12.5% dextrose to prevent hypoglycemia while continuing to infuse insulin to correct the metabolic acidosis.
- These glucose concentrations are often necessary to prevent hypoglycemia when insulin is infused at a rate of 0.1 unit/kg/h

Insulin therapy

- • If BG falls very rapidly (>5 mmol/L/h) after initial fluid expansion, consider adding glucose even before plasma glucose has decreased to (300 mg/dL).

Insulin therapy

- . If serum glucose concentrations decrease to less than 200 mg/dL before correction of acidosis, the glucose concentration of the IV fluids should be increased, but the insulin infusion should not be decreased by more than half, and it should never be discontinued before resolution of acidosis

Insulin therapy

Insulin therapy decreases the production of free fatty acids and protein catabolism and enhances glucose usage in target tissues.

These processes correct acidosis.

Bicarbonate therapy should be avoided.

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INSULIN THERAPY

- As acidosis is corrected, urine ketone concentrations may appear to rise. βHydroxybutyrate, which is not detected in urine ketone assays, is converted with treatment to what the assay most detects, acetoacetate.
- Hence minute-to-minute urine ketone concentrations are not a required index of the adequacy of therapy

Insulin therapy

- If biochemical parameters of DKA (venous pH, anion gap, BOHB concentration) do not improve,
- reassess the patient,
- review insulin therapy, and consider other possible causes of impaired response to insulin; for example, infection, errors in insulin preparation or route of administration.

Subcutaneous administration insulin regular

- Subcutaneous administration of short-acting insulin (regular) every 4 hours is also a safe and effective alternative to IV insulin infusion in children with pH ≥7.0.

- A suggested starting dose is 0.8 to 1 unit per kg per 24-hours; the calculated 24-hour dose is divided by 6 to provide an insulin dose injected every 4 hours.

Subcutaneous administration insulin regular

- Doses are increased or decreased by 10% to 20% based on the blood glucose level before the next insulin injection. For example, if a child weighs 45 kg: 45 × 0.8 = 36 units; starting dose is 6 units.

Subcutaneous administration insulin

- In circumstances where continuous IV administration is not possible and in patients with uncomplicated DKA,
- hourly or 2-hourly SC rapid-acting insulin analog (insulin lispro or insulin aspart) is safe and may be as effective as IV regular insulin infusion,

- Initial dose SC: 0.3 unit/kg, followed 1 hour later by SC insulin lispro or aspart at 0.1 unit/kg every hour, or 0.15 to 0.20 units/kg every 2 to 3 hours.

Potassium replacement

Intracellular potassium is depleted because of

- transcellular shifts caused by hypertonicity (increased plasma osmolality causes solvent drag in which water and potassium are drawn out of cells),
- acidosis, glycogenolysis and proteolysis secondary to insulin deficiency also cause potassium efflux from cells.
- vomiting as a consequence of osmotic diuresis.
- Volume depletion causes secondary hyperaldosteronism, which promotes urinary potassium excretion.

Potassium replacement

Renal dysfunction, by enhancing hyperglycemia and reducing potassium excretion, contributes to hyperkalemia.

Administration of insulin and the correction of acidosis drive potassium back into the cells, decreasing serum potassium levels.

The serum potassium concentration may decrease abruptly, predisposing the patient to cardiac arrhythmias.

Potassium replacement

If hypokalemic,

start potassium replacement at the time of initial volume expansion and before starting insulin therapy.

start replacing potassium after initial volume expansion and concurrent with starting insulin therapy.

If the patient is hyperkalemic, defer potassium replacement therapy until urine output is documented.

Potassium replacement

- If immediate serum potassium measurements are unavailable, an ECG may help to determine whether the child has hyper- or hypokalemia. hypokalemia.

Prolongation of the PR interval, T-wave flattening and inversion, ST depression, prominent U waves, apparent long QT interval (due to fusion of the T and U waves)

hyperkalemia

Tall, peaked, symmetrical, T waves and shortening of the QT interval.

Potassium replacement

- The starting potassium concentration in the infusate should be 40 mmol/L.

Subsequent potassium replacement therapy should be based on serum potassium measurements. •

If potassium is given with the initial rapid volume expansion, a concentration of 20 mmol/L should be used

The maximum recommended rate of IV potassium replacement is usually 0.5 mmol/kg/h.

Potassium replacement

- • If hypokalemia persists despite a maximum rate of potassium replacement, then the rate of insulin infusion can be reduced. •

- Profound hypokalemia (2.5 mmol/L to reduce the risk of cardiopulmonary and neuromuscular compromise

Treatment

- Serum potassium concentrations can decrease rapidly as insulin and then glucose therapy improves acidosis, and potassium is exchanged for intracellular hydrogen ions.

- When adequate urine output is shown, potassium should be added to the IV fluids. Potassium replacement should be given as 50% potassium chloride and 50% potassium phosphate at a concentration of 20-40 mEq/L

Treatment

- . If the serum potassium level is greater than 6 mEq/L, potassium should not be added to IV fluids until the potassium level decreases.

Phosphate

- Depletion of intracellular phosphate occurs
- result of osmotic diuresis.

- after starting treatment and this is exacerbated by insulin, which promotes entry of phosphate into cells.

- Clinically significant hypophosphatemia may occur if IV therapy without food consumption is prolonged beyond 24 hours.

Phosphate

- Severe hypophosphatemia is uncommon, but can have severe consequences.

- Manifestations depend on the severity and chronicity of the phosphate depletion; patients usually do not have symptoms until plasma phosphate is< 1 mg/dL (0.32 mmol/L)

Phosphate

- Severe hypophosphatemia can occur during treatment of DKA; however, symptoms are uncommon because the hypophosphatemia usually is acute and there typically is no antecedent chronic phosphate deficiency

Clinical manifestations of hypophosphatemia

- Clinical manifestations of hypophosphatemia are largely due to intracellular phosphate depletion. Decreased intracellular ATP levels impair cellular functions that depend on energy-rich phosphate compounds, and a decrease in 2,3-diphosphoglycerate (DPG) level increases the affinity of hemoglobin for oxygen and reduces oxygen release in tissues.

- Many organ systems can be affected.

Clinical manifestations of hypophosphatemia

Metabolic encephalopathy (irritability, paresthesias, confusion, seizures, coma);

impaired myocardial contractility and respiratory failure due to weakness of the diaphragm;

muscle dysfunction with proximal myopathy, dysphagia and ileus;

.

Clinical manifestations of hypophosphatemia

- rare hematologic effects include
- hemolysis,
- decreased phagocytosis and granulocyte chemotaxis,
- defective clot retraction,
- thrombocytopenia.

- Acute hypophosphatemia in a patient with preexisting severe phosphate depletion can lead to rhabdomyolysis

HYPOPHOSPHATEMIA BE TREATED

Severe hypophosphatemia associated with any of the above symptoms should be treated.

Administration of phosphate may induce hypocalcemia.

Potassium phosphate salts may be safely used as an alternative to or combined with potassium chloride or acetate, provided that careful monitoring of serum calcium is performed to avoid hypocalcemia

Hypophosphatemia be treated

- This combination provides phosphate for replacement of deficits, but avoids excess phosphate administration, which may precipitate hypocalcemia

Treatment

- Initial laboratory measurements should include serum glucose, sodium, potassium, chloride, bicarbonate, BUN, creatinine, calcium, phosphate, and magnesium concentrations; arterial or venous pH; and a urinalysis.
- Serum glucose measurement should be repeated every hour during therapy; electrolyte concentrations should be repeated every 2-3 hours.
- Calcium, phosphate, and magnesium concentrations should be measured every 4-6 hours during therapy..

Antibiotics and Bladder catheterization

- Give antibiotics to febrile patients after obtaining appropriate cultures of body fluids. • Bladder catheterization usually is not necessary, but if the child is unconscious or unable to void on demand (eg, infants and very ill young children) the bladder should be catheterized.

DKA andECG

- • If laboratory measurement of serum potassium is delayed, perform an electrocardiogram (ECG) for baseline evaluation of potassium status.

Glasgow coma scale or score (GCS)

- Best eye response
- 1No eye opening
- 2. Eyes open to pain
- 3. Eyes open to verbal command
- 4. Eyes open spontaneously

GLASGOW COMA SCALE

- Best verbal response

1. No verbal response

- 2. No words, only incomprehensible sounds; moaning
- 3. Words, but incoherenta
- 4. Confused, disoriented conversationb
- 5. Oriented, normal conversation

GLASGOW COMA SCALE

(non-verbal children)

- 1. No response
- 2. Inconsolable, irritable, restless, cries
- 3. Inconsistently consolable and moans; makes vocal sounds
- 4. Consolable when crying and interacts inappropriately
- 5. Smiles, oriented to sound, follows objects and interacts

Glasgow coma scale

- Best motor response

- 1. No motor response
- 2. Extension to pain (decerebrate posture)
- 3. Flexion to pain (decorticate posture)
- 4. Withdrawal from pain
- 5. Localizes pain
- 6. Obeys commands

Glasgow coma scale

- The GCS consists of three parameters and is scored between 3 and 15; 3 being the worst and 15 the best.

CEREBRAL EDEMA IMPORTANCE

- Cerebral injury is the major cause of mortality and morbidity
- cerebral edema accounts for 60% to 90% of all DKA deaths.
- From 10% to 25% of survivors of cerebral edema have significant residual morbidity.

CEREBRAL EDEMAIMPORTANCE

- Complications Clinically apparent cerebral edema occurs in 1-5% of cases of DKA.
- Cerebral edema is the most serious complication of DKA, with a mortality rate of 20- 80%.
- The pathogenesis of cerebral edema likely involves osmolar shift resulting in fluid accumulation in the intracellular compartment and cell swelling..

CEREBRAL EDEMA PATOPHYSIOLOGY

- Despite much effort to identify the cause of cerebral edema, its pathogenesis is incompletely understood and controversy continues concerning the association between the rate of fluid or sodium administration used in the treatment of DKA and the development of cerebral edema.

CEREBRAL EDEMA

- Neurological and mental status should be assessed at frequent intervals.

- Any complaints of headache or deterioration of mental status should prompt rapid evaluation for possible cerebral edema.

CEREBRAL EDEMA SYMPTOMS

- Indicative symptoms include a decreased sensorium, sudden severe headache, vomiting, change in vital signs (bradycardia, hypertension, apnea), a dilated pupil, ophthalmoplegia, or seizure

CEREBRAL EDEMA symptoms

- change in neurological status (restlessness,
- irritability,
- increased drowsiness, confusion, incontinence),
- specific neurological signs (eg, cranial nerve palsies),
- rising blood pressure,
- decreased oxygen saturation (

CEREBRAL EDEMA symptoms

- One diagnostic criterion, two major criteria, or one major and two minor criteria have a sensitivity of 92% and a false positive rate of only 4%.
- Signs that occur before treatment should not be considered in the diagnosis of cerebral edema.
- Neuroimaging is not required for diagnosis of cerebral edema.

CEREBRAL EDEMA symptoms

- Diagnostic criteria ·

- Abnormal motor or verbal response to pain · Decorticate or decerebrate posture
- · Cranial nerve palsy (especially III, IV, and VI)
  ·
- Abnormal neurogenic respiratory pattern (eg, grunting, tachypnea, Cheyne-Stokes respiration, apneusis)

CEREBRAL EDEMA symptoms

- Major criteria ·
- Altered mentation, confusion, fluctuating level of consciousness ·
- Sustained heart rate deceleration (decrease more than 20 beats per minute) not attributable to improved intravascular volume or sleep state ·
- Age-inappropriate incontinence

CEREBRAL EDEMA

- Minor criteria ·

- Vomiting ·
- Headache ·
- Lethargy or not easily arousable ·
- Diastolic blood pressure >90 mm Hg ·
- Age<5 years

CEREBRAL EDEMA symptoms

- Subclinical cerebral edema is common in patients with DKA, but the factors that exacerbate this process leading to symptomatic brain swelling and possible cerebral herniation are not clearly defined.

CEREBRAL EDEMA and diabetes insipidus,

- The appearance of diabetes insipidus, manifested by increased urine output with a concomitant marked increase in the serum sodium concentration, reflecting loss of free water in the urine, is a sign of cerebral herniation causing interruption of blood flow to the pituitary gland

CEREBRAL EDEMA

- Cerebral edema typically occurs 6-12 hours after therapy for DKA is begun, often following a period of apparent clinical improvement

- rarely, may develop as late as 24 to 48 hours after the start of treatment

CEREBRAL EDEMA risk factor

- . Factors that correlate with increased risk for cerebral edema include
- higher initial BUN concentration,
- lower initial PCO2 ,
- failure of the serum sodium concentration to increase as glucose concentration decreases during treatment,

- treatment with bicarbonate

CEREBRAL EDEMA risk factor

More severe acidosis at presentation ·

- A marked early decrease in serum effective osmolality
- Greater volumes of fluid given in the first 4 hours

- Administration of insulin in the first hour of fluid treat

CEREBRAL EDEMA TREATMENT

- A chart with the reference ranges for blood pressure and heart rate (which vary depending on height, weight, and gender) should be readily available, either in the patient's chart or at the bedside.
- .

CEREBRAL EDEMA Treatment

- Treatment involves the rapid use of IV mannitol, endotracheal intubation, and ventilation and may require the use of a subdural bolt.

CEREBRAL EDEMA Treatment

- Initiate treatment as soon as the condition is suspected. •
- Adjust fluid administration rate as needed to maintain normal blood pressure while avoiding excessive fluid administration that might increase cerebral edema formation.
- Assiduously avoid hypotension that might compromise cerebral perfusion pressure.

Treatment of cerebral edema

- Hyperosmolar agents should be readily available at the bedside. •
- Give mannitol, 0.5 to 1 g/kg IV over 10 to 15 minutes.
- The effect of mannitol should be apparent after ~15 minutes, and is expected to last about 120 minutes.
- If necessary, the dose can be repeated after 30 minutes.

Treatment of cerebral edema

- Hypertonic saline (3%), suggested dose 2.5 to 5 mL/kg over 10 to 15 minutes,
- may be used as an alternative to mannitol,

or in addition to mannitol if there has been no response to mannitol within 15 to 30 minutes.

Hypertonic saline (3%) 2.5 mL/kg is equimolar to mannitol 0.5 g/kg

Treatment of cerebral edema

- Elevate the head of the bed to 30 and keep the head in the midline position. •
- Intubation may be necessary for the patient with impending respiratory failure due to severe neurologic compromise

Treatment of cerebral edema

- cranial imaging may be considered as with any critically ill patient with encephalopathy or acute focal neurologic deficit.

- treatment of the clinically symptomatic patient should not be delayed in order to obtain imaging.

Treatment of cerebral edema

- primary concern that would warrant neuroimaging is whether the patient has a lesion requiring emergency neurosurgery (eg, intracranial hemorrhage) or a lesion that may necessitate anticoagulation (eg, cerebrovascular thrombosis),

- as suggested by clinical findings of focal or severe, progressive headache, or focal neurologic deficit

Bicarbonate administration

- is not recommended except for treatment of life-threatening hyperkalemia or unusually severe acidosis (vpH<6,9)

- If bicarbonate is considered necessary, cautiously give 1 to 2 mmol/kg over 60 minutes

Bicarbonate administration

- Complications of therapy •
- Cerebral edema •
- Hypokalemia •

Hyperchloremic acidosis •

- Hypoglycemia •
- Inadequate rehydration

Blood pressure and DKA

- Despite their dehydration, patients generally continue to maintain normal blood pressure or even have high blood pressure,

- possibly due to elevated plasma catecholamine concentrations,
- increased release of antidiuretic hormone (ADH) in response to hyperosmolality (which increases blood pressure via V2 receptors), increased osmotic pressure from marked hyperglycemia, or other factors.

URINE OUTPUT IN DKA

- Considerable urine output persists because of glucosuria until extreme volume depletion leads to a critical decrease in renal blood flow and glomerular filtration

DKA AND BS LEVEL

- Consumption of fluids with a high-carbohydrate content (juices or sugar-containing soft drinks) may exacerbate the hyperglycemia.

- though uncommonly, modest hyperglycemia in the setting of severe acidosis may be an indication that the patient has maintained increased water intake and may be only modestly hypovolemic.

DKA AND BS LEVEL

- Rapid emptying of stomach contents containing an abundant quantity of sugar, which occurs as gastroparesis is relieved with therapy, accounts for the rise in plasma glucose concentration observed in some patients

Other complications of DKA

- intracranial thrombosis or infarction,
- acute kidney injury with acute renal failure caused by severe dehydration,
- pancreatitis,
- arrhythmias caused by electrolyte abnormalities, pulmonary edema, bowel ischemia.
- Peripheral edema occurs commonly 24-48 hours after therapy is initiated and may be related to residual elevations in antidiuretic hormone and aldosterone.

Transition to Outpatient Management

- When the acidosis has been corrected and the patient tolerates oral feedings, the IV insulin infusion can be discontinued and a regimen of subcutaneous (SC) insulin injections can be initiated.
- The first SC insulin dose should be given 30-45 minutes before discontinuation of the IV insulin infusion.
- Further adjustment of the insulin dose should be made over the following 2-3 days.

Transition to Outpatient Management

- A patient already diagnosed with T1D may be restarted on the prior doses if they were adequate.
- For a patient with new-onset T1D, typical starting total daily doses are approximately 0.5-0.7 U/kg/24 hr for prepubertal patients
- approximately 0.7- 1 U/kg/24 hr for adolescents, using any number of the available insulin combinations.

Transition to Outpatient Management

- The best and most common choice for making the transition to SC insulin is to begin by giving injections of fast-acting (bolus) insulin (lispro, aspart, or glulisine insulin) with each meal and long-acting (basal) insulin (glargine or detemir) at bedtime.

- An alternative is a fixed mixed split dosing regimen (neutral protamine Hagedorn [NPH] and fastingacting insulin) with two daily injections.

Transition to Outpatient Management

- Demonstration of the ability to administer insulin injections and test glucose concentrations using a glucose meter is necessary before discharge, as is knowledge of hypoglycemia management.
- Meal planning is crucial to control glucose in T1D.
- Nutrition services must be part of the care delivered to the families from diagnosis

HYPOGLYCEMIA

- The adverse effects of hypoglycemia in young children may be significant because the immature CNS may be more susceptible to glycopenia..

Diabetes Control

- Given the benefits of stable, near normal blood glucose concentrations, a goal of HgbA1c <7.5% is set for children of all ages.
- Preprandial blood glucose target concentrations are 90-130 mg/dL and concentrations before bedtime and
- overnight of 90-150 mg/dL. Goals of therapy need to take into account other individual characteristics, such as a past history of severe hypoglycemia and the abilities of the patient and family.

Insulin Regimens

- Insulin Regimens Many types of insulin differ in duration of action and time to peak effect
- These insulins can be used in various combinations, depending on the needs and goals of the individual patient.
- The most commonly used regimen is that of multiple injections of fast-acting insulin given with meals in combination with long-acting basal insulin given at bedtime.

Insulin Regimens

- After the total daily dose of insulin is determined, 30-50% is given as long-acting insulin, and the remainder is given as fast-acting insulin, divided according to the need for corrections of high glucose levels and for meals.

INSULIN SENSITIVITY

- To correct for hyperglycemia, one can determine the insulin sensitivity using the 1,800/1,500 rule: dividing 1,800 (or 1,500) by the total daily dose of insulin to determine how many milligrams per deciliter of glucose will decrease with one unit of insulin

INSULIN:CARBOHYDRATE RATIO

- . is used to calculate insulin for the carbohydrate content of food; 500 divided by the total daily dose determines the number of grams of carbohydrate that requires one unit of insulin.

Blood Glucose Testing

- Blood glucose should be routinely monitored before each meal and at bedtime.

- Hypoglycemia during the night or excessive variability in the morning glucose concentrations should prompt additional testing at 2 or 3 A.M. to ensure that there is no consistent hypoglycemia or hyperglycemia.

Blood Glucose Testing

- During periods of illness or when blood glucose concentrations are higher than 300 mg/dL, urine ketones also should be tested. Continuous glucose monitors, which provide minute-tominute blood glucose concentration information, can be useful in following trends of blood glucose concentrations, but should not be used in calculations of mealtime insulin doses

Symptoms of hypoglycemia

- Neuroglycopenia
- (headache, visual changes, confusion, irritability, or seizures)
- symptoms resulting from the catecholamine response (tremors, tachycardia, diaphoresis, or anxiety).
- .

Treatment hypoglycemia

- Mild episodes can be treated with administration of rapidly absorbed oral glucose (glucose gel or tablets or fruit juices).
- More severe episodes
- seizures or loss of consciousness at home should be treated with glucagon injections.
- IV glucose should be given in hospital settings

۱- ارزیابی فوری

- ارزیابی بالینی برای تایید تشخیص و عامل زمینه ای (مثلاً عفونت) انجام شود
- بیمار توزین شود و از آن برای محاسبات استفاده شود
- شدت کم آبی بصورت دقیق برآورد شود
- سطح هوشیاری برآورد شود (معیار گلاسکو)
- نمونه خون جهت آزمایش قند خون، الکترولیتها،گازهای خون (شامل TCO_2 و سطح بیکربنات)، BUN و کراتنین، هموگلوبین، هماتوکریت، کلسیم، فسفر، منیزیوم گرفته شود. معمولاً علت بالا بودن لکوسیتها بعلت استرس است تا عفونت.
- سطح کتونها در خون و یا ادرار بررسی شود .
- نمونه برای کشت خون، ادرار، حلق در صورت وجود شواهد عفونت گرفته شود
- در صورتیکه جواب پتاسیم دیر آماده می شود برای ارزیابی آن الکتروکاردیوگرام انجام شود

۲- اقدامات محافظتی

- در بیماران دارای کاهش سطح هوشیاری و یا خواب آلود راههای هوایی باز و سوند معده برای جلوگیری از آسپیراسیون گذاشته شود

- یک کاتتر در رگهای محیطی برای نمونه گیری های مکرر تعبیه شود
- مانیتورینگ قلبی برای ارزیابی وضعیت موج T و هیپرکالمی و هیپوکالمی احتمالی انجام شود
- برای بیماران در حال شوک و با وضعیت قلبی – عروقی مختل اکسیژن تجویز شود
- سوند مثانه برای بیماران با سطح هوشیاری پایین و شیرخواران گذاشته شود

۳ – جایگزینی آب و الکترولیت

- برای بیماران دچار کم آبی شدید (در حال شوک) برای افزایش آنی حجم داخل عروقی از نرمال سالین استفاده شود . میزان مایع معمولاً $10^{cc}/kg/hr$ است ولی برحسب وضعیت بیمار می توان تغییر نماید .
- در موارد نادری که بیمار در شوک است و کلاپس شدید قلبی عروقی دارد، میزان $20^{cc}/kg$ بصورت بولوز و انفوزیون آزاد با سرعت هرچه تمام تجویز می شود قبل از تجویز دوز بعدی (درصورت نیاز) وضعیت بیمار ارزیابی گردد.

در صورتیکه اقدام به رگ گیری ناموفق است از تزریق داخل استخوان استفاده شود

- جایگزینی مایعات حداقل برای ٤ تا ٦ ساعت اول با نرمال سالین انجام شود . بعد از آن سرم حداقل معادل نیم نرمال سدیم به همراه پتاسیم (کلراید، فسفات، استات)تجویز می شود
- جایگزینی مایعات بر مبنای ٤٨ ساعت برنامه ریزی شود (خوراکی یا تزریقی)
- مثالی از جبران مایعات نگهدانده و ١٠ درصد کم آبی در عرض ٤٨ ساعت :

وزن (کیلوگرم)	سرعت انفوزیون (ML/kg/h)
٤-٩	٦
١٠-١٩	٥
٢٠-٣٩	٤
٤٠-٥٩	٣/٥
٦٠-٨٠	٣

برای نمونه در یک کودک ٦ ساله با وزن ٢٠کیلوگرم 80^{cc} در ساعت یا ^{cc} ١٩٢٠ درعرض ٢٤ ساعت برای دو روز متوالی مورد نیاز است .

٤- درمان با انسولین

- انسولین ۱ تا ۲ ساعت بعد از مایع درمانی شروع شود . برای مثال بعد از انفوزیون سرم اولیه که با هدف افزایش حجم داخل عروقی تجویز می شود.
- کمبود انسولین بیمار باید جبران شود.
- دوز بولوز اولیه در شروع توصیه نمی شود و از تجویز آن باید اجتناب شود.
- انسولین به میزان 0.1 u/kg/hr تا اصلاح کتواسیدوز PH بالای ۷/۳ و بیکربنات بالای ۱۵ و یا بسته شدن شکاف آنیونی ادامه یابد این مدت خیلی طولانی تر از مدت زمان لازم برای اصلاح هیپرگلسیمی بیمار است.
- هرگاه مشخص شود که بیمار به انسولین حساس است مانند شیرخواران و یا افراد مبتلا به کمای هیپر اسمولار دوز انسولین 0.05 U/kg/hr تقلیل می یابد تا اسیدوز بیمار اصلاح شود.
- در ساعتهای اولیه درمان قند سریع پایین می آید ولی بعد از آن قند هر ساعت ۱۰۰-۴۰ میلی گرم در دسی لیتر اُفت می کند و شدت آن به زمان شروع و میزان سرم قندی بستگی دارد.
- باهدف جلوگیری از افت شدید قند خون و احتمال هیپوگلیسمی سرم قندی به میزان ۵ درصد دکستروز به محض رسیدن قند خون به ۲۵۰-۳۰۰ میلی گرم در دسیلیتر شروع می شود.
- گاهی لازم است برای اصلاح اسیدوزو تدوام انسولین درمانی از سرم قندی با غلظت ۱۰ یا ۱۲/۵ درصد استفاده شود.

۵- جایگزینی پتاسیم

- صرف نظر از میزان پتاسیم خون جایگزینی پتاسیم ضروری است
- اگر بیمار هیپوکالمیک است پتاسیم را همزمان با مایع درمانی اولیه و قبل از شروع انسولین شروع نمایید. در غیر اینصورت پتاسیم بعد از مایع درمانی اولیه و همزمان با شروع انسولین آغاز شود. اگر بیمار هیپرکالمیک است شروع پتاسیم را تاحصول اطمینان از برقراری جریان ادرار به تاخیر بیندازید
- اگر اندازه گیری سریع پتاسیم مقدور نیست یک نوار قلب می تواند کمک کننده باشد
- صاف شدن موج T، پهن شدن فاصله QT و ظهور موج U بیانگر هیپوکالمی هستند
- موج T بلند و کم شدن فاصله QT علایم هیپرکالمی هستند

۹- ادم مغز

- علایم هشدار دهنده ادم مغز شامل :
- سردرد و کاهش ضربان قلب
- تغیر در وضعیت عصبی (بیقراری، تحریک پذیری، خواب آلودگی،بی اختیاری)
- علایم عصبی اختصاصی (مردمکها بدون پاسخ، فلج اعصاب مغزی)

- افزایش فشار خون
- کاهش اشباء اکسیژن

درمان ادم مغز

- به محض حدس بروز ادم مغز درمان شروع گردد
- حجم مایعات تجویزی به ۱/۳ تقلیل یابد
- مانیتول به میزان 0.5-1 g/kg بصورت وریدی در عرض ۲۰ دقیقه انفوزیون و بین ۰/۵ تا ۲ ساعت بعد در صورت پاسخ نگرفتن تکرار شود
- جایگزین مانیتول در مواردیکه پاسخ به آن دیده نشده است سالین هیپرتونیک (۳٪) به میزان $5^{cc}/kg$ در عرض ۳۰ دقیقه است

همیشه از در دسترس بودن مانیتول و سالین هیپرتونیک بر بالین بیمار اطمینان حاصل کنید

- وضعیت سربیمار را بالاتر قرار دهیم

۱۰- مونیتورینگ بالینی و بیوشیمیایی

- کنترل هر ساعت (و یا بیشتر در شرایط لازم) علایم حیاتی (ضربان قلب، تعداد تنفس و فشار خون)
- کنترل وضعیت هوشیاری (گلاسکو) بیمار هر ساعت (و یا بیشتر در شرایط ضروری) برای علایم هشداری بروز ادم مغز
- میزان انسولین تجویز شده هر ساعت
- کنترل هر ساعت میزان مایعات تجویز شده
- چک قند خون هر ساعت با گلوکومتر

- تستهای آزمایشگاهی : سدیم، پتاسیم، قند خون، RUN، کلسیم، منیزیوم، فسفر، CBC، VBG هر ۲ تا ٤ ساعت (و یا بیشتر در مواقع ضروری) چک شوند
- کتون خون و یا ادرار هر ۲ ساعت تاوقتیکه منفی شوند چک شود

Moderate and severe DKA

- Shock with hemodynamic compromise is rare in pediatric DKA.
- Clinical estimates of the volume deficit are subjective and inaccurate
- therefore, in moderate DKA assume 5% to 7% and in severe DKA 7% to 10% dehydration.

انواع انسولین : (Insulin Types)

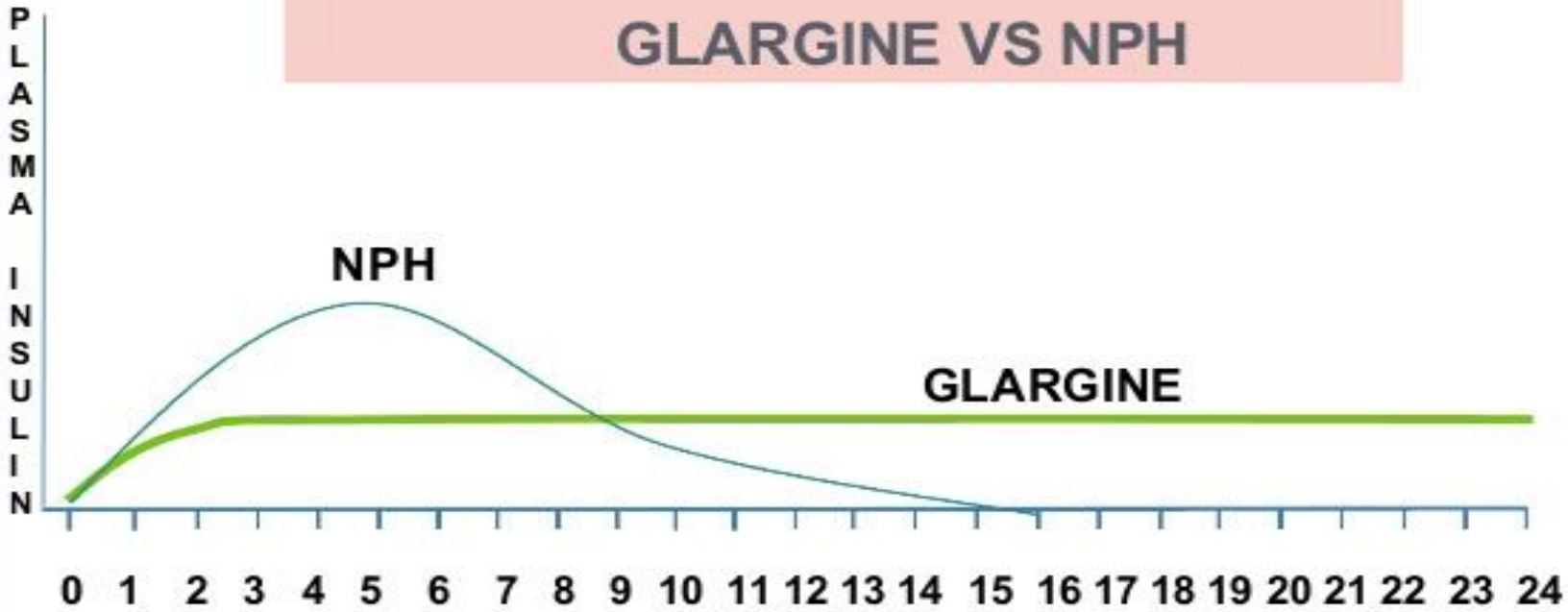
	Type	Onset (hour)	Peak (hour)	Duration (hour)
انسولین سریع الاثر	Aspart/Lispro	0.25 hr	1 hr	3-4 hr
انسولین کوتاه اثر	Regular	0.5 - 1	2-4	6-8
انسولین متوسط الاثر	NPH	1-3	6-8	12-16
انسولین طولانی اثر	Glargine	1	No peak	11-26

CLASSIFICATION

Insulin and analogue preparations

Short acting	Long acting
- Regular	- NPH
- Lispro	- Glargine
- Aspart	- Detemir
- Glulisine	- Degludec

GLARGINE VS NPH



Important information

- Do not shake the insulin as this damages the insulin?
- After first usage, an insulin vial should be discarded after 3 months if kept at 2-8 C or 4 weeks if kept at room temperature.
- Intermediate-acting **and** short-acting/rapid-acting insulin, can be combined in one Syringe.
- Use 4mm needle for injection of Insulin SC.

Insulin syringe: (سرنگ های انسولین)

۳ نوع سرنگ انسولین در بازار موجود است:

۱- سرنگ انسولین ۱۰۰ واحدی (هر خط کوچک سرنگ انسولین ۱۰۰ واحدی معادل ۲ واحد است).

۲- سرنگ انسولین ۵۰ واحدی (هر خط کوچک سرنگ انسولین ۵۰ واحدی معادل یک واحد است).

۳- سرنگ انسولین ۳۰ واحدی (هر خط کوچک سرنگ انسولین ۳۰ واحدی معادل نیم واحد است).

ADA(انجمن دیابت امریکا) توصیه می کند که قندخون در یک فرد دیابتی حداقل ۴ بار در شبانه روز اندازه گیری شود:

- قند ناشتا
- ۲ ساعت بعد از صبحانه

- ۲ ساعت بعد از ناهار
- ۲ ساعت بعد از شام

* همچنین قند شبانه (ساعت 3AM) حداقل ۳ تا ۴ بار در ماه باید اندازه گیری شود.

۵- معاینه محل هایی که بیمار انسولین تزریق می کند. (جهت بررسی لیپودیستروفی و لیپوهیپرتروفی که ممکن است در اثر عدم چرخش محل انسولین ایجاد شود)

۶- معاینه پای بیمار (به صورت سالیانه در کودکان بالای ۱۰ سال انجام می شود)

۷- معاینه چشم ها (در صورت لزوم جهت بررسی رتینوپاتی دیابتی، بیمار را جهت معاینه ته چشم به متخصصین چشم ارجاع می دهیم)

۳- در زمان تشخیص همه بچه های مبتلا به دیابت تیپ ا بایستی از نظر بیماری سلیاک screen شوند.

برای این منظور total IgA و Anti TTG Ab (IgA) یا anti endomysial Ab چک می شود.

- اگر تست های سلیاک + شدند بیمار جهت management لازم بایستی به یک متخصص گوارش کودکان ارجاع داده شود.
- اگر تست های سلیاک – شدند بیمار باید یک سال در میان از نظر سلیاک بررسی شود.

۷- در هر ویزیت درمانگاهی برای یک بیمار دیابتی باید جدول قندهای اور را ببینیم و مقادیر انسولین بیمار را براساس آن ها تنظیم کنیم.

- اگر قندهای ناشتای بیمار بالا باشد، میزان NPH عصر را زیاد می کنیم.
- اگر قندهای ۲ ساعت پس از صبحانه بیمار بالا باشد، میزان انسولین رگولار صبح را اضافه می کنیم.
- اگر قندهای ۲ ساعت پس از ناهار بیمار بالا باشد، میزان انسولین رگولار ظهر را اضافه می کنیم.
- اگر قندهای ۲ ساعت بعد از شام بیمار بالا باشد، میزان انسولین رگولار عصر (قبل از شام) را اضافه می کنیم.

پدیدهٔ Somogyi :

اگر در یک بیمار دیابتی قندهای ناشتای بالا داشته باشیم و با افزودن مقادیر NPH شب باز هم قندهای ناشتا اصلاح نشوند بایستی از بیمار قند 3AM چک کنیم. اگر قند نیمه شب بیمار زیر 70 بود بایستی به فکر پدیدهٔ Somogyi بیفتیم.

تعریف: در پدیدهٔ Somogyi در اثر overtreatment با انسولین، قند نیمه شب بیمار افت می کند. هیپوگلیسمی شبانه باعث آزاد شدن Counter regulatory hormones می شود، آزاد شدن این هورمون ها باعث لیپولیز، گلوکونئوژنز و گلیکوژنولیز می شود که حاصل آن یک Rebound hyperglycemia است که با افزایش قندهای ناشتا خود را نشان می دهد.

- برای درمان پدیدهٔ Somogyi چه می کنیم؟
- به بیمار توصیه می کنیم شب هنگام (موقع خواب) یک وعده غذای سبک (مثلاً نان و پنیر یا مقداری مغزی جات) مصرف کند.
- می توان دوز NPH عصر یا شب بیمار را کم کنیم.
- می توانیم توصیه کنیم انسولین Regular را قبل از شام و انسولین NPH را موقع خواب تزریق کند.

پدیدهٔ Dawn چیست؟

- اگر در بیماران دیابتی قندهای ناشتای بالا داشته باشیم و قندهای نیمه شب بیمار (ساعت 2 تا 3AM) هم بالا باشد (200<) بایستی به فکر پدیدهٔ Dawnبیفتیم.

این پدیده در اثر افزایش ترشح شبانه هورمون هایی نظیر GH و کورتیزول و گلوکاگون و اپی نفرین ایجاد می شود. این امر منجر به افزایش مقاومت به انسولین می شود. بنابراین قندهای شبانه و قندهای ناشتای بیمار بالا می رود.برای درمان پدیدهٔ Dawn دوز انسولین عصر یا شبانه بیمار را افزایش دهید.

Emergency Treatment of Hypoglycemia •

give a fast acting carbohydrate i.e. :
3-4 (15 g) glucose tablets or o

15 ml (1 tbsp.) sugar dissolved in ½ cup water

175 ml (3/4 c) juice or

15 ml (1 tbsp.) of honey
HYPOGLYCEMIA IS LIFE THREATENING IF LEFT UNTREATED….
DO NOT give food or drink if the student is unconscious, having a seizure or is unable to swallow:

give glucagon if delegated

مراقبتهای توصیه شده :

کودکان و نوجوانان مبتلا به DKA باید در مراکز دارای تجربه کافی در زمینه درمان بیماران و امکانات برای سنجش علایم حیاتی، وضعیت عصبی و آزمایشگاه مجهز و به دفعات مکرر پاسخگو بستری شوند.

- مراحل تزريق
- 1- قبل از هر ترزيق بايد دستها را كاملاً بشوييد
- (محل تزريق انسولين نيز بايد تميز باشد)
- ،شيشه انسولين را به آرامي 2NPH- در صورت استفاده از انسولين بين دو كف دست بغلتانيد،ولي شيشه را تكان ندهيد.
- 3- در پوش پلاستيكي شيشه هاي انسولين را با پنبه الكلي تميز نماييد.

- به همان ميزاني كه انسولين لازم داريد، هوا وارد شيشه كنيد . اگر از دو نوع انسولين به صورت مخلوط استفاده مي كنيد،ابتدا هوا وارد هر دو شيشه نماييد.
- 5-شيشه را برگردانيد و ميزان انسولين لازم را وارد سرنگ كنيد. در صورت ترزيق دو نوع انسولين ،ابتدا انسولين كريستال را در سرنگ NPH بكشيدسپس انسولين ، وارد سرنگ نماييد.
- 6- بعد از كشيدن انسولين به داخل سرنگ ، حبا بهاي هوا را خارج كنيد(حبا بهاي هوا به تنهايي خطرناك نيستند اما مقدار انسولين تزريقي با وجود حبا بهاي هوا كاهش مي يابد) چند ضربه به بدنه سرنگ وارد كنيد تا حبا بها به سطح آمده،آنگاه با فشار مختصري به پيستون حبابها را خارج نماييد .
- 7- زاويه تزريق در افراد چاق و لاغر متفاوت است . در افراد چاق در ناحيه پيشين رانها و بازوها ابتدا بايد پوست بالا آورده شود و با زاويه 90سر سوزن وارد گردد. اما در افراد لاغر اين زوايه در بازوها45 است . تغيير زوايه تزريق بايد با توجه به قطر عضله محل ترزيق باشد .

- سر سوزن رابه سرعت و با ملاميت وارد پوست كنيد.
- اگر بعد از بيرون كشيدن سوزن از پوست و پايان ترزيق، ناحيه دردناك شده يا مايعي روشن از محل ترزيق خارج گرديد، محل ترزيق را به مدت چند ثانيه فشار دهيد .
- **مشكلات ناشي از تزريق انسولين چيست ؟**
- كاهش قند خون (هيپوگليسمي)ممكن است در افرادي كه انسولين بيش از حد مورد نياز دريافت كرد هاند يا مقدار خيلي كم مواد غذايي مصرف نموده اند و يا مدت زمان زيادي ورزش كرده اند رخ دهد.
- لازم است علامتهاي افت قند خون را شناخته و به سرعت براي درمان آن اقدام كنيد.نشانه هاي افت قند خون در افرا مختلف متفاوت بوده و شامل عرق ريزش ، سردرد، سر گيجه ، تپش قلب، لرزش، لكنت زبان ، دو بيني ، عصبانيت و تغيير ناگهاني خلق مي باشد. اگر دچار افت قند خون شده ولي هوشيار هستيد ، مي توانيد 2/1 ليوان شير يا دو قطعه بيسكويت مصرف كنيد. اگر دچار لرزش شديد اندام شده و احساس سر گيجه داشتيد مي توانيد 2/1 ليوان آب ميوه يا نوشابه 5-6 عدد شكلات و يا 1-2 قاشق چايخوري شكر يا عسل مصرف كنيد. در هر حال علامتهاي افت قند خون را جدي بگيرند و به سرعت آن را درمان كنيد.

- فواصل تزريق در يك عضو را رعايت كنيد. فاصله هر تزريق با تزريق بعدي بايد 1-5/1 سانتي متر باشد.
- دماي انسولين تزريقي بايد مشابه دماي اتاق باشد (30 دقيقه قبل از تزريق ، شيشه انسولين را از يخچال خارج كنيد).
- از نبود حباب در داخل سرنگ انسولين اطمينان حاصل كنيد ، سپس تزريق را انجام دهيد.
- اگر پيش از تزريق ، از الكل استفاده مي كنيد(ماليدن الكل در محل تزريق در صورت تميز بودن پوست ضرورت ندارد) بايد صبر كنيد تا الكل كاملاً از سطح پوست تبخير شود، سپس تزريق را انجام دهيد.
- هنگام ورود سر سوزن، عضله محل تزريق را شل كنيد .
- راستا و جهت و زوايه سر سوزن را بعد از ورود و همچنين هنگام خروج از پوست تغيير ندهيد .
- اگر سر سوزن كند شده است ، دوباره از اين سرنگ استفاده نكنيد .
- سعي كنيد همواره انسولين را در يك ساعت مشخص از شبانه روز تزريق نماييد.
- مخلوط انسولين NPHو كريستال داخل سرنگ را حداكثر تا 15-5 دقيقه بعد از مخلوط كردن، تزريق نماييد.
- اگر يك نوع انسولين داخل سرنگ كشيد هايد ، انسولين داخل سرنگ را مي توان تا يك هفته داخل يخچال نگهداري كرد.


با تشکر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