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Also found in: Dictionary, Thesaurus, Acronyms, Encyclopedia, Wikipedia.Related to diabetic ketoacidosis: diabetic diet, diabetic coma Diabetic ketoacidosis is a dangerous complication of diabetes mellitus in which the chemical balance of the body becomes far too acidic.Diabetic ketoacidosis (DKA) always results from a severe insulin deficiency. Insulin is the hormone secreted by the body to lower the blood sugar levels when they become too high. Diabetes mellitus is the disease resulting from the inability of the body to produce or respond properly to insulin, required by the body to convert glucose to energy. In childhood diabetes, DKA complications represent the leading cause of death, mostly due to the accumulation of abnormally large amounts of fluid in the brain (cerebral edema). DKA combines three major features: hyperglycemia, meaning excessively high blood sugar kevels; hyperketonemia, meaning an overproduction of ketones by the body; and acidosis, meaning that the blood has become too acidic.Insulin deficiency is responsible for all three conditions: the body glucose goes largely unused since most cells are unable to transport glucose into the cell without the presence of insulin; this condition makes the body use stored fat as an alternative source instead of the unavailable glucose for energy, a process that produces acidic ketones, which build up because they require insulin to be broken down. The presence of excess ketones in the bloodstream in turn causes the blood to become more acidic than the body tissues, which creates a toxic condition.DKA is most commonly seen in individuals with type I diabetes, under 19 years of age and is usually caused by the interruption of their insulin treatment or by acute infection or trauma. A small number of people with type II diabetes also experience ketoacidosis, but this is rare given the fact that type II diabetics still produce some insulin naturally. When DKA occurs in type II patients, it is usually caused by a decrease in food intake and an increased insulin deficiency due to hyperglycemia.Some common DKA symptoms include: high blood sugar levels frequent urination (polyuria) and thirst fatigue and lethargy nausea vomiting abdominal pain fruity odor to breath rapid, deep breathing muscle stiffness or aching coma Diagnosis requires the demonstration of hyperglycemia, hyperketonemia, and acidosis. DKA is established if the patient's urine or blood is strongly positive for glucose and ketones. Normal glucose levels in a non-diabetic person on average range from 80-110 mg/dl. A person with diabetes will typically fluctuate outside those parameters. DKA glucose levels exceed 250 mg/dl and can reach 400 to 800 mg/dL. A low serum bicarbonate level (usually below 15 mEq/L) is also present, indicative of acidosis.A blood test or urinalysis can quickly determine the concentration of glucose in the bloodstream. Test strips are available to patients commercially can submerge in urine to detect the presence or concentration of ketones.Acidosis — A condition that causes the pH of the blood to drop and become more acidic.Diabetes mellitus — Disease characterized by the inability of the body to produce or respond properly to insulin, required by the body to convert glucose to energy.Edema — The presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body.Glucose — The type of sugar found in the blood.Hyperglycemia — Condition characterized by excessively high levels of glucose in the blood, and occurs when the body does not have enough insulin or cannot use the insulin it does have to turn glucose into energy. Hyperglycemia is often indicative of diabetes that is out of control.Hyperketonemia — Condition characterized by an overproduction of ketones by the body.Hypoglycemia — Lower than normal levels of glucose in the blood.Hypokalemia — A deficiency of potassium in the blood.Insulin — A hormone secreted by the pancreas in response to high blood sugar levels that induces hypoglycemia. Insulin regulates the body's use of glucose and the levels of glucose in the blood by acting to open the cells so that they can intake glucose.Ketones — Poisonous acidic chemicals produced by the body when fat instead of glucose is burned for energy. Breakdown of fat occurs when not enough insulin is present to channel glucose into body cells.Lactic acidosis — A serious condition caused by the build up of lactic acid in the blood, causing it to become excessively acidic. Lactic acid is a by-product of glucose metabolism.Metabolism — The sum of all chemical reactions that occur in the body resulting in growth, transformation of foodstuffs into energy, waste elimination, and other bodily functions.Polyuria — Excessive secretion of urine.Type I diabetes — Also called juvenile diabetes. Type I diabetes typically begins early in life. Affected individuals have a primary insulin deficiency and must take insulin injections.Type II diabetes — Type II diabetes is the most common form of diabetes and usually appears in middle aged adults. It is often associated with obesity and may be delayed or controlled with diet and exercise.Ketoacidosis is treated under medical supervision and usually in a hospital setting.Basic treatment includes: administering insulin to correct the hyperglycemia and hyperketonemia replacing fluids lost through excessive urination and vomiting intravenously balancing electrolytes to re-establish the chemical equilibrium of the blood and prevent potassium deficiency (hypokalemia) during treatment treatment for any associated bacterial infection With proper medical attention, DKA is almost always successfully treated. The DKA mortality rate is about 10%. Coma on admission adversely affects the prognosis. The major causes of death are circulatory collapse, hypokalemia, infection, and cerebral edema.Once diabetes has been diagnosed, prevention measures to avoid DKA include regular monitoring of blood glucose, administration of insulin, and lifestyle maintenance. Glucose monitoring is especially important during periods of stress, infection, and trauma when glucose concentrations typically increase as a response to these situations. Ketone tests should also be performed during these periods or when glucose is elevated.American Diabetes Association. 1701 North Beauregard Street, Alexandria, VA 22311. (800) DIABETES (800-342-2383). Diabetes Foundation. 120 Wall St., 19th Floor, New York, NY 10005. (800) 533-CURE. Institute of Diabetes and Digestive and Kidney Disorders (NIDDK). 31 Center Drive, MSC 2560, Bethesda, MD 20892-2560. Encyclopedia of Medicine. Copyright 2008 The Gale Group, Inc. All rights reserved.buildup of ketones in blood due to breakdown of stored fats for energy; a complication of diabetes mellitus. Untreated, can lead to coma and death.Farlex Partner Medical Dictionary © Farlex 2012The American Heritage® Medical Dictionary Copyright © 2007, 2004 by Houghton Mifflin Company. Published by Houghton Mifflin Company. All rights reserved. A hyperglycemia-induced clinical crisis most common in type 1 DM Clinical N&V, thirst, diaphoresis, hyperpnea, drowsiness, fever, prostration, coma, possibly death Lab ↑ ↑ ↑ Glucose, often > 33.6 mmol/L–US: > 600 mg/dL, ↑ ketone bodies, relative ↑ in protein, albumin, Ca2+, BR, alk phos, AST, CK, anion gap, acidosis, dehydration, ↓ K+, Na+, phosphate Management Insulin, fluid and electrolyte replacement, treat initiating factors–eg, leukocytosis or hypothermia, avoid complications–eg, hypokalemia, late hypoglycemiaMcGraw-Hill Concise Dictionary of Modern Medicine. © 2002 by The McGraw-Hill Companies, Inc. (DKA) (di-ă-bet'ik kē'tō-as'i-dō'sis) Buildup of ketones in blood due to breakdown of stored fats for energy; a complication of diabetes mellitus. Untreated, can lead to coma and death. Medical Dictionary for the Dental Professions © Farlex 2012 Want to thank TFD for its existence? Tell a friend about us, add a link to this page, or visit the webmaster's page for free fun content. Link to this page: diabetic ketoacidosis Abbreviations DKA: Diabetic ketoacidosis ICU: Intensive care unit PD-1: Antiprogrammed death 1 receptor PD-1L: Programmed death ligand GADA: Glutamic acid decarboxylase autoantibodies IAA: Insulin autoantibodies IA-2A: Islet antigen 2 autoantibodies ICA: Islet cell antibodies ZnT8A: Zinc transporter 8 autoantibodies. Diabetic ketoacidosis (DKA) is an acute metabolic complication of diabetes characterized by hyperglycemia, hyperketonemia, and metabolic acidosis. Hyperglycemia causes an osmotic diuresis with significant fluid and electrolyte loss. DKA occurs mostly in type 1 diabetes mellitus. It causes nausea, vomiting, and abdominal pain and can progress to cerebral edema, coma, and death. DKA is diagnosed by detection of hyperketonemia and anion gap metabolic acidosis in the presence of hyperglycemia. Treatment involves volume expansion, insulin replacement, and prevention of hypokalemia.Test your KnowledgeTake a Quiz! Figure 1 Pathogenesis of DKA and HHS:… Figure 1 Pathogenesis of DKA and HHS: stress, infection, or insufficient insulin. FFA, free fatty… Medical conditionHyperosmolar hyperglycemic stateOther namesHyperosmolar hyperglycemic nonketotic coma (HHNC), hyperosmolar non-ketotic coma (HONK), nonketotic hyperosmolar coma, hyperosmolar hyperglycemic nonketotic syndrome (HHNS) [1]SpecialtyEndocrinologySymptomsSigns of dehydration, altered level of consciousness[2]ComplicationsDisseminated intravascular coagulopathy, mesenteric artery occlusion, rhabdomyolysis[2]Usual onsetDays to weeks[3]DurationFew days[3]Risk factorsInfections, stroke, trauma, certain medications, heart attacks[4]Diagnostic methodBlood tests[2]Differential diagnosisDiabetic ketoacidosis[2]TreatmentIntravenous fluids, insulin, low molecular weight heparin, antibiotics[3]Prognosis~15% risk of death[4]FrequencyRelatively common[2] Hyperosmolar hyperglycemic state (HHS), also known as hyperosmolar non-ketotic state (HONK), is a complication of diabetes mellitus in which high blood sugar results in high osmolarity without significant ketoacidosis.[4][5] Symptoms include signs of dehydration, weakness, leg cramps, vision problems, and an altered level of consciousness.[2] Onset is typically over days to weeks.[3] Complications may include seizures, disseminated intravascular coagulopathy, mesenteric artery occlusion, or rhabdomyolysis.[2] The main risk factor is a history of diabetes mellitus type 2.[4] Occasionally it may occur in those without a prior history of diabetes or those with diabetes mellitus type 1.[3][4] Triggers include infections, stroke, trauma, certain medications, and heart attacks.[4] Diagnosis is based on blood tests finding a blood sugar greater than 30 mmol/L (600 mg/dL), osmolarity greater than 320 mOsm/kg, and a pH above 7.3.[2][3] Initial treatment generally consists of intravenous fluids to manage dehydration, intravenous insulin in those with significant ketones, low molecular weight heparin to decrease the risk of blood clotting, and antibiotics among those in whom there are concerns of infection.[3] The goal is a slow decline in blood sugar levels.[3] Potassium replacement is often required as the metabolic problems are corrected.[3] Efforts to prevent diabetic foot ulcers are also important.[3] It typically takes a few days for the person to return to baseline.[3] While the exact frequency of the condition is unknown, it is relatively common.[2][4] Older people are most commonly affected.[4] The risk of death among those affected is about 15%.[4] It was first described in the 1880s.[4] Symptoms of high blood sugar including increased thirst (polydipsia), increased volume of urination (polyuria), and increased hunger (polyphagia).[6] Symptoms of HHS include: Altered level of consciousness Neurologic signs including: blurred vision, headaches, focal seizures, myoclonic jerking, reversible paralysis[6] Motor abnormalities including flaccidity, depressed reflexes, tremors or fasciculations Hyperviscosity and increased risk of blood clot formation Dehydration[6] Weight loss[6] Nausea, vomiting, and abdominal pain[6] Weakness[6] Low blood pressure with standing[6] The main risk factor is a history of diabetes mellitus type 2.[4] Occasionally it may occur in those without a prior history of diabetes or those with diabetes mellitus type 1.[3][4] Triggers include infections, stroke, trauma, certain medications, and heart attacks.[4] Other risk factors: Lack of sufficient insulin (but enough to prevent ketosis)[6] Poor kidney function[6] Poor fluid intake (dehydration)[6] Older age (50–70 years)[6] Certain medical conditions (cerebral vascular injury, myocardial infarction, sepsis)[6] Certain medications (glucocorticoids, beta-blockers, thiazide diuretics, calcium channel blockers, and phenytoin)[6] HHS is usually precipitated by an infection,[7] myocardial infarction, stroke or another acute illness. A relative insulin deficiency leads to a serum glucose that is usually higher than 33 mmol/L (600 mg/dL), and a resulting serum osmolarity that is greater than 320 mOsm. This leads to excessive urination (more specifically an osmotic diuresis), which, in turn, leads to volume depletion and hemoconcentration that causes a further increase in blood glucose level. Ketosis is absent because the presence of some insulin inhibits hormone-sensitive lipase-mediated fat tissue breakdown.[citation needed] According to the American Diabetes Association, diagnostic features include:[8][9] Plasma glucose level >30 mmol/L (>600 mg/dL) Serum osmolality >320 mOsm/kg Profound dehydration, up to an average of 9L (and therefore substantial thirst (polydipsia)) Serum pH >7.30[9] Bicarbonate >15 mEq/L Small ketonuria (~+ on dipstick) and absent-to-low ketonemia (30 mg/dL (increased)[6] Creatinine > 1.5 mg/dL (increased)[6] Cranial imaging is not used for diagnosis of this condition. However, if an MRI is performed, it may show cortical restricted diffusion with unusual characteristics of reversible T2 hypointensity in the subcortical white matter.[10] The major differential diagnosis is diabetic ketoacidosis (DKA). In contrast to DKA, serum glucose levels in HHS are extremely high, usually greater than 40-50 mmol/L (600 mg/dL).[6] Metabolic acidosis is absent or mild.[6] A temporary state of confusion (delirium) is also more common in HHS than DKA. HHS also tends to affect older people more. DKA may have fruity breath, and rapid and deep breathing.[6] DKA often has serum glucose level greater than 300 mg/dL (HHS is >600 mg/dL).[6] DKA usually occurs in type 1 diabetics whereas HHS is more common in type 2 diabetics.[6] DKA is characterized by a rapid onset, and HHS occurs gradually over a few days.[6] DKA also is characterized by ketosis due to the breakdown of fat for energy.[6] Both DKA and HHS may show symptoms of dehydration, increased thirst, increased urination, increased hunger, weight loss, nausea, vomiting, abdominal pain, blurred vision, headaches, weakness, and low blood pressure with standing.[6] The JBDS HHS care pathway[11] comprises 3 main themes to consider when managing a patient with HHS: clinical assessment and monitoring interventions assessments and prevention of harm To streamline management, there are 5 phases of therapy from the time of recognition of the condition to resolution: 0–60 min 1–6 hours 6–12 hours 12–24 hours 24–72 hours[11] Treatment of HHS begins with reestablishing tissue perfusion using intravenous fluids. People with HHS can be dehydrated by 8 to 12 liters. Attempts to correct this usually take place over 24 hours with initial rates of normal saline often in the range of 1 L/h for the first few hours or until the condition stabilizes.[12] Potassium replacement is often required as the metabolic problems are corrected.[3] It is generally replaced at a rate 10 mEq per hour as long as there is adequate urinary output.[13] Insulin is given to reduce blood glucose concentration; however, as it also causes the movement of potassium into cells, serum potassium levels must be sufficiently high or dangerously low blood potassium levels may result. Once potassium levels have been verified to be greater than 3.3 mEq/L, then an insulin infusion of 0.1 units/kg/hr is started.[14] The goal for resolution is a blood glucose of less than 200 mg/dL.[6] ^ "Hyperosmolar Hyperglycemic Nonketotic Syndrome (HHNS)". American Diabetes Association. Archived from the original on 2 July 2012. 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PMID 36370077. ^ Tintinalli, Judith E.; Kelen, Gabor D.; Stapczynski, J. Stephan (2004). Emergency Medicine: A Comprehensive Study Guide (6th ed.). McGraw-Hill Prof Med/Tech. p. 1309. ISBN 978-0-07-138875-7. Archived from the original on 24 March 2017. ^ Tintinalli, Kelen & Stapczynski 2004, p. 1320 ^ Tintinalli, Kelen & Stapczynski 2004, p. 1310 Retrieved from " "DKA" redirects here. For other uses, see DKA (disambiguation). Medical conditionDiabetic ketoacidosisSpecialtyEndocrinologySymptomsVomiting, abdominal pain, deep gasping breathing, increased urination, confusion, a specific smell[1]ComplicationsCerebral edema[2]Usual onsetRelatively rapid[1]CausesShortage of insulin[3]Risk factorsUsually type 1 diabetes, less often other types[1]Diagnostic methodHigh blood sugar, low blood pH, high ketoacid levels[1]Differential diagnosisHyperosmolar nonketotic state, alcoholic ketoacidosis, uremia, salicylate toxicity[4]TreatmentIntravenous fluids, insulin, potassium[1]Frequency4–25% of people with type 1 diabetes per year[1][5] Diabetic ketoacidosis (DKA) is a potentially life-threatening complication of diabetes mellitus.[1] Signs and symptoms may include vomiting, abdominal pain, deep gasping breathing, increased urination, weakness, confusion and occasionally loss of consciousness.[1] A person's breath may develop a specific "fruity" or acetone smell.[1] The onset of symptoms is usually rapid.[1] People without a previous diagnosis of diabetes may develop DKA as the first obvious symptom.[1] DKA happens most often in those with type 1 diabetes but can also occur in those with other types of diabetes under certain circumstances.[1] Triggers may include infection, not taking insulin correctly, stroke and certain medications such as steroids.[1] DKA results from a shortage of insulin; in response, the body switches to burning fatty acids, which produces acidic ketone bodies.[3] DKA is typically diagnosed when testing finds high blood sugar, low blood pH and keto acids in either the blood or urine.[1] The primary treatment of DKA is with intravenous fluids and insulin.[1] Depending on the severity, insulin may be given intravenously or by injection under the skin.[3] Usually, potassium is also needed to prevent the development of low blood potassium.[1] Throughout treatment, blood glucose and potassium levels should be regularly checked.[1] Underlying causes for the DKA should be identified.[6] In those with severely low blood pH who are critically ill, sodium bicarbonate may be given; however, its use is of unclear benefit and typically not recommended.[1][6] Rates of DKA vary around the world.[5] Each year, about 4% of type 1 diabetics in the United Kingdom develop DKA, versus 25% of type 1 diabetics in Malaysia.[1][5] DKA was first described in 1886 and continued to be a universally fatal condition until introduction of insulin therapy in the 1920s.[7] With adequate and timely treatment, the risk of death is between