

# Pathophysiology Of Type 1 Diabetes & Chest Infection

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## Introduction

Eric is 25 years old and has been diagnosed with diabetes for ten years and controls his blood glucose. He coughed for the past two weeks. He feels breathless and now spits out significant yellow mucus. A constant cough can be disturbing to anyone, but as far as people with diabetes are concerned, it complicates things. To start with, a diabetic cannot merely go for cough syrup over the counter since it is possible for it to be strong in sugar. Second, coughing as an outcome of a cold exerts extra stress on a diabetic patient, increasing his blood sugar. Thus, treating coughing in people with diabetes involves much more care and consideration. If a cough is the result of an infection, the immune system tries to fight it by distributing substantial amounts of hormones to counter the disease. Although it is effective for patients without diabetes, it can lead to problems for people with diabetes since these hormones inhibit the action of insulin in the system (Anton Y. Peleg, 2006), regardless if it is normal insulin released by the pancreas or if it is externally received for treatment of diabetes, this hormonal interfering will interact with the insulin to raise glucose levels in the blood. The color of the phlegm that Eric coughs indicates pathogens that infect his body. If you spit out massive amounts of phlegm, you may have an infection or allergies. Thick, yellowish phlegm is a symptom of bacterial or viral infection, bronchitis, lower [respiratory tract](#) illness or sinus infection. The yellow mucus suggests that the immune cells begin to act on the site of the disease; as the white blood cells continue to fight the infection, they are picked up by the mucus, giving it a yellowish hue (C. M. Fletcher, 1959). From the above, it can be diagnosed that Eric, who is a type I diabetic, may have symptoms of a chest infection of bronchitis that leads to a wet cough producing thick yellow mucus. The two ills that Eric suffers from and their reciprocal interactions have been analyzed according to their pathological processes, and appropriate pathological care has been suggested.

## Analysis Of Type 1 Diabetes Mellitus

An evaluation of insulin deficiency as a result of loss of function and mass of beta cells is investigated here. The first anomalies of beta cell malfunction, visible before the emergence of illness symptoms, comprise the reduction of pulsatile insulin emission and inhibition of first-stage insulin (FPIR) reaction to glucose within the arterial circulation. Even before and after the start of symptoms, insulin secretion promptly decreases, and that decrease in its function leads to an increase in liver glucose production and decreased glucose uptake by insulin-sensitive tissues, especially muscles and fats (Chunguang Chen, 2017). The blood sugar level rises and spreads in the urine. Withdrawal of glucose

in other tissues triggers the deterioration of structural fats and proteins, resulting in rapid weight loss. The deficiency of insulin following an unmanaged diabetic condition leads to increased production of acid ketone bodies and results in a metabolic ailment called diabetic ketoacidosis, which is an indication of mismanaged type 1 diabetes mellitus. (Brandt, 1999)

## Aetiology Of Type 1 Diabetes Mellitus

Type 1 diabetes is included in the category of diseases that are known as autoimmune diseases. Autoimmune disorders occur when the immune system mistakenly classifies its beneficial cells as an attacking organism. Genetic predisposition is a critical factor as researchers have discovered some genetic regions closely related to type 1 diabetes. While genetics provides hints as to the reason that some people are more risk prone to type 1 diabetes, it does not provide an explanation of the reason why in some people these genes cause type 1 diabetes to develop and why others do not have these genes (Feld, 2002). The researchers suggested that there is a possibility that environmental factors are responsible for triggering the initial development of type 1 diabetes (McCulloch DK, 1991). Viral infection is also described as a cause, as links have been established between type 1 diabetes and various viruses. Research has demonstrated that anti-viruses have been observed at higher rates in pregnant women with other children who are at risk of type 1 diabetes (Christophe M. Farne, 2008).

Another advanced theory is that infantile vaccinations might have a role in children acquiring type 1 diabetes later in their lives. Some studies suggest that there may be a connection between immunization vaccines such as Hib vaccines, tuberculosis and smallpox, and type 1 diabetes mellitus (Stratton KR, 1994). A compelling indication of the fact that the tuberculosis vaccine is related to a greater occurrence of type 1 diabetes, but other research has also highlighted its contribution to prevention (Massachusetts General Hospital, 2017). Additionally, a link between type 1 diabetes and vitamin D has been established (Kamal as Al-Shoumer, 2015). The researchers found that regions with the maximum occurrence of type 1 diabetes are usually further from the Equator. Research has also shown that those people have a higher occurrence rate of developing type 1 diabetes and have a lower vitamin D content in their system.

Another theory enlists cow's milk to be a cause of developing type 1 diabetes. Research has demonstrated that introducing cow's milk at an early age period was linked to a higher occurrence of type 1 diabetes (Gottlieb, 2000). They theorized that the reason for the risk could be bovine insulin in cow's milk. A contributing factor was also an increase in insulin demand. One study found that a diet excessive in glycemic index foods may hasten the development of type 1 diabetes in infants with signs of self-immunity (Molly M. Lamb, 2008). As children reach adolescence, the growth phase they go through leads to an increase in the quantity of insulin secreted and can cause additional stress on beta cells, which increases the likelihood that the immune system will attack the cells that secrete insulin (Molly M. Lamb, 2015).

# Epidemiology Of Type 1 Diabetes Mellitus

Type 1 diabetes may occur at any time in life, but it usually occurs at an early age with a greater risk during the puberty phase. Its frequency varies with the highest rates in Northern Europe (CC, 2009). Both genders are risk-prone in childhood, but more likely to be affected are men who are at the beginning of adult life (Gale EAM, 2001). The frequency of type 1 diabetes in children is increasing swiftly in populations everywhere, especially in infants under the age of five, with an expanding period of fewer than two decades in European populations (Podar, 2001). The growing frequency of type 1 diabetes indicates a significant environmental influence in factors leading to growth, whereas viruses have also been attributed as a possible cause but remain contested.

Type 1 diabetes has always been the most common in ethnic populations originating from Europe, though it is increasingly observed to have been progressing in other ethnic groups as well. Populations that are genetically related have also demonstrated differences in development: for instance, type 1 diabetes is less widespread in Icelanders of mainly Norwegian origin than it is in people from Norway, whereas children from Finland are at triple in likelihood to be risk-prone than people from Estonia (Podar, 2001). The prevalence of type 1 diabetes rests comparatively lower in ethnicities and populations of non-European origin, though several reports observe an increasing occurrence of the disease elsewhere, too. The incidence in Kuwait, for instance, has become 22.3 out of 100,000 inhabitants. The chances of developing type 1 diabetes peak at puberty and decrease swiftly after that. In populations of older groups, the classification of type 1 diabetes becomes more problematic. A transnational study on male percentages among infants or teenagers under 15 years of age found a small surplus of male patients among European residents and people of European descent, whereas a surplus of female patients was observed in the population of Asian and African origin (Karvonen, 1997). On the other hand, a clear male majority emerged in most surveys of people diagnosed with type 1 diabetes appeared amid the ages of 15 and 40 (Gale EAM, 2001).

There is increasing evidence of increasing incidence since childhood diabetes was an uncommon disease at the beginning of the twentieth century. From the mid-twentieth century, or shortly after that, some populations experienced a resumption of incidence, which continued more or less linearly to this day (Gale EAM, 2001). The present general percentage of upsurge in Europe is around 3-4% each year, while the fastest increase is for children from 0 to 5 (Podar, 2001). However, there are significant intraregional variances, with indications of a swift upsurge in Eastern European regions (Jarosz, 2011). There are also suggestions that the increasing occurrence of diabetes in children may signify a change towards the left side of the starting period instead of signifying an entire overall increase within a whole population's likelihood of developing the disease, and this result is consistent with the improved sensitivity of genes under more tolerant environment (EAM, 2005). The increased prevalence within populations of stable genetic makeup indicates environmental influences as possible factors, but the specific factors currently being considered do not convincingly explain the possible association (Hermann, 2003).

# Pathological Processes Type 1 Diabetes Mellitus

Type 1 diabetes is considered an autoimmune illness, unlike type 2 diabetes. It is also called insulin-dependent diabetes mellitus. Various immune cells, including macrophages, cell cells (DC), B lymphocytes, and Natural Killer Cells (NK), are connected with oxidative stress, inflammation, and other deleterious biological processes that cause damage to pancreatic beta cells (responsible for the Insulin production) (G Mendel, 1989). When the pancreas is injured because of a bacterium, virus, infection, drug, toxin, etc., the immune system does not have enough time to mature and be strong enough to fight the effects. In response to the fact that the pancreas is injured or damaged, the immune system intervenes (as it should) to initiate cell repair and the pancreatic healing process (Aggarwal, 2015). The first phase of the cellular repair process is inflammation, as the body needs more blood and other elements to repair and rebuild damaged tissue.

During this phase, macrophages are activated as part of the period of inflammation and the process of cellular repair. During this time, macrophages recruit other immune cells to help you. Frequently, after the ignition phase, the cellular repair process changes into the proliferation and remodeling phases. Because immune cells are not healthy enough to fight viruses, infections and other causal factors, immune cells, mainly macrophages, send cytokine signals to recruit more cells to immune T cells, TH1, TH2 and TH17 (Pagliuca et al., 2014). Depending on the type of infection, TH2 cells can indirectly recruit B cells to cause antibodies, While macrophages can recruit more macrophages and more T cells. The immune system tries to coordinate and balance the immune response between the TH1 and TH2 cells, as well as the TH17 and Treg cells, but is unable to do so. In most cases, there are many more TH1 and Th17 cells that create a significant imbalance. Also, toxic T lymphocytes are recruited – their primary task is to kill infected cells. However, if the infected cells pass beta cells, then the TC or not cells begin to destroy the beta cell. Other immune cells, such as macrophages, whose mission is to kill invasive pathogens, attack infected or damaged beta cells, causing a further increase in beta cell death.

As the inflammation increases, causing more damage, there is an increase in free radicals, causing an increase in oxidation, which also causes damage to the beta cells. This creates a vicious circle of inflammation, oxidation, cellular injury and cell death (Atkinson, 2012). As the number of damaged beta cells increases, the amount of insulin secretion decreases, causing a rise in levels of blood glucose. This eventually leads to severe hyperglycemia, which usually goes unnoticed until the person starts to feel very tired and urine a lot, which leads to a doctor's appointment. With less than 10% of the remaining insulin production, the patient received insulin to bring the blood sugar level back to a reasonable and safe level.

## Pathophysiological Processes

An overview of the between metabolism is provided. The primary organ responsible for the circulation

of glucose homeostasis in the system is the liver. It does this by storing and absorbing the glucose after meals and secreting it in precise volumes between meals; Insulin regulates both these roles. The effect of insulin on the liver is controlled by the amplitude instead of the frequency at which secretory impulses are produced. Moreover, the secreted quantity is regulated by glucose levels and additional fuels in the blood used by the beta cell (Song, 2000). Glucose can be stored in the form of liver glycogen, which is a fast-circulating glucose source. A large amount of the glucose created by the liver comes from gluconeogenesis, which also occurs in the renal cortex and helps in producing about 10% of total glucose. High levels of insulin stimulate glycogen development and inhibit glucose production through gluconeogenesis. Production of glucose is maintained by the mutual equilibrium between glucagon and insulin; high glucagon and low insulin encourage the release of glucose, whereas low glucagon and higher insulin content promote glucose intake. 140 g of glucose each day is usually produced by the liver, of which 40 to 50% is absorbed by an anaerobic route to water and carbon dioxide through the brain. Glucose mainly provides the energy needed to induce membrane pumps that maintain the nerve membrane potential difference (Rui, 2014).

Glucose not needed by the brain is expended by other glucose-demanding tissues like the brush border of the gut or red blood cells absorbed into that surrounding the muscles, influenced by elevated insulin levels. Glucose gets phosphorylated in the cell and is stored in the form of triacylglycerol in fat cells or glycogen in tissue. The consequences of Insulin deficiency include overproduction of glucose by the liver and decreased inhibition of gluconeogenesis (Wilcox, 2005). Tissues whose glucose carriers are not affected by insulin can still be penetrated by glucose in nervous tissues as well as others, however the entry of glucose into insulin-sensitive tissues like muscles and fats is lessened. Plasma glucose increases and surpasses the capacity of the glucose carrier in the proximal kidney tube to reabsorb glucose prior to it entering urine. When glucose crosses the kidney tubes, it presents an osmosis gradient that causes various electrolytes and salts to be lost (Wilcox, 2005). The patient feels more thirst and a greater urge to urinate frequently.

Even in healthy individuals, prolonged fasting can lead to falling Insulin levels, thereby causing the breakdown of non-essential proteins and lipids that serve as a standby fuel for the body. Insulin scarcity in diabetes produces an accelerated fasting condition, and collective loss of fluid and tissue mass results in quick weight loss (Adinortey, 2017). A breakdown of fatty acids in liver mitochondria results in the formation of organic Ketone acidic bodies, a progression efficiently blocked by insulin in low concentrations. A loss of inhibition of this inhibition can occur as a result of extreme insulin deficiency, generating an overproduction of ketone bodies. As a result, the metabolic acidosis that happens leads to intense vomiting and nausea, exacerbating the deficiency of electrolytes and fluid through the kidneys. This chain of events results in a severe metabolic disease called diabetic ketoacidosis, an ailment that was considered fatal before the introduction of insulin (Laffel, 1999).

# Diagnosis Of Bronchitis In Eric

Acute bronchitis is a medical disease triggered by inflammation of the bronchi, bronchioles and trachea. Acute bronchitis is seldom a primary bacterial infection. Indicators of acute bronchitis are typically a productive cough and occasionally subject to severe discomfort through deep coughing or breathing (CC Horner, 2009). In general, the development of acute bronchitis is spontaneously restricted, with broad scarring and a total return to normal function usually observed within 10 to 14 days after beginning of symptoms (Worrall, 2008). Chronic bronchitis is a recurrent degeneration and inflammation of the bronchi that can be linked with an active contamination. Chronic bronchitis patients have more mucus than usual due to a decrease clearance and an increase in production (Victor Kim, 2013). The mechanism through which excess mucus is eliminated is coughing. Chronic bronchitis' incidence has been difficult to assess due to a major medical overlap with asthma plus the response states of respiratory disease (Rubin, 2014). In adults, chronic bronchitis is characterised by a daily productive cough for a minimum of three months in two successive years. Chronic bronchitis is also diagnosed from a range of symptoms. A recurring productive and chronic cough that has crackling, and lasts more than a month (Rubin, 2014). In the case of chronic bronchitis, inhalation treatment should be administered. It can be supplemented with oral corticosteroids if the coughing persists and if patient's history and clinical investigation indicate symptoms of asthmatic bronchitis (J Andrew Woods, 2014).

## Pathophysiology Of Bronchitis

Acute bronchitis causes phlegm production and cough, which usually results from infection in the upper respiratory tract. This happens through the inflammatory reaction of the mucous membranes in the bronchial passages of the lungs. A single virus or often a combination of viruses are responsible for a majority of these infections (Brodzinski H, 2009) (Miron D, 2010). An airway that suffers from such a reaction quickly responds by cough and bronchospasm, that is followed by inflammation, oedema and greater mucus production. Mucociliary clearance is a vital and key inner defence mechanism that guards the lungs from the damaging effects of inhaled allergens, pollutants or pathogens. (Voynow, 2009). A typical feature of chronic respiratory disease is mucociliary dysfunction. The mucociliary apparatus contains 3 functional systems: the eyelashes, a protective mucus layer, and a layer of airway fluid (ASL) surfaces that function collectively to eliminate any inhaled particles from the lungs. The role of exposure to irritants, especially smoke from cigarettes and airborne particles, in persistent bronchitis and asthma becomes plain. Air particles of nitrogen dioxide and carbon dioxide have been linked with symptoms of chronic bronchitis in asthmatic children (McConnell R, 2003). A persistent insult of the respiratory epithelium, such as frequent aspiration or recurring viral infection, may lead to childhood chronic bronchitis (A, 2007). As a result of damage to the mucous membranes of the airways, chronic infections on usually isolated airway organisms can also happen. The usual bacterial agent causing lower respiratory tract infections in infants of all ages is streptococcal pneumonia (PURUSHOTHAMA V. Dasaraju, 196). Children with

tracheostomy are often colonised by some flora, that includes gamma-hemolytic streptococci and alpha-hemolytic streptococci. Children who are prone to oropharyngeal aspiration, mainly those with weakened airway protection mechanisms, can be infected with streptococcus orally anaerobic strains. (ml Barnett, 2014)

## Etiology Of Bronchitis

Acute bronchitis is usually instigated by respiratory infections; 90% of the time usually they are viral, with about 10% bacterial. Repeated attacks of acute bronchitis, that irritate and weaken bronchial passages over time also can possibly lead to chronic bronchitis. Another common cause is Industrial pollution; The main responsibility, however, is a strong exposure to long-time cigarette smoke. Viral infections can include: Influenza, Adenovirus Influenza, Rhinovirus, Respiratory Syncytial Virus, Human Bocavirus (Bond N, 2008). Coxsackievirus, herpes simplex virus (Schildgen O, 2008). Air contaminants, especially those associated with tobacco and passive smoke, also can lead to bronchiolitis incident (Koehoorn M, 2008). One study suggested that the XPC DNA repair gene is associated with the stimulation of the pathogenesis of bronchial airway pollution in children (Ghosh R, 2016). Other reasons include: chronic aspiration, allergies, fungal infection or gastroesophageal reflux diseases

## Epidemiology Of Bronchitis

Data presented in the summary of the National Outpatient Care Survey of 1991 revealed that 2 774 000 visits to children under 15 diagnosed with bronchitis (US Department of Health and Social Services, 1994). 14 million Americans have been identified to have chronic bronchitis each year since 1996. Bronchitis, both chronic and acute, is widespread all over the world. It is among the top five causes for visiting doctors in children. The occurrence of bronchitis among British students would be 20.7%. A general increase in hospital stays for the lower respiratory tract infection from 1996 to 2000 amid German children is consistent with the data of infants from the United Kingdom, United States, and Sweden (Weigl, 2005). The frequency of children suffering from bronchitis in this German survey group was 28%. Differences in population prevalence were noted in patients with chronic bronchitis. The occurrence of acute bronchitis is the same in men and women. The lack of a definitive diagnostic criteria makes it challenging to study the impact of chronic bronchitis because of its significant overlap with asthma. However, recently, the incidence of chronic bronchitis has been observed to be constantly higher in women than in men (MeiLan K. Han, 2007). Acute (usually wheezing) bronchitis most commonly occurs in children under two years, with a second incidence peak observed in children between 9 and 15 years of age. The effects of chronic bronchitis are more common in people over the age of 45 (AlexisFerré, 2012).

# Pathology Of Bronchitis

Acute bronchitis is typically produced by a viral infection. Syncytial respiratory, influenza and coronavirus viruses are the most common causes in patients less than one-year-old (John S. Tregoning, 2010). In patients aged 1 to 10, the influenza virus, viruses, syncytial respiratory virus and rhinovirus dominate the causes of acute bronchitis. Patients over the age of 10 have the most common reasons of the influenza virus, syncytial respiratory illness, and adenovirus (John S. Tregoning, 2010). Acute bronchitis was first described as inflammation of the bronchial mucous membranes. Among the complex events leading to the disease is an infectious or non-infectious trigger. This leads to a lesion of the bronchial epithelium, which leads to an inflammatory reaction with hyperreactivity of the respiratory tract and mucus production (B Moldoveanu, 2009). The repair of the bronchial wall takes several weeks. The patient will always cough during these weeks. Half of the patients with acute bronchitis will continue to cough for more than two weeks, and in a quarter of the patients, it will take more than a month (Michael D Shields, 2013). Chronic bronchitis in children may be the result of excessive inflammation or continuous exposure to allergens or irritants. This leads to bronchospasm and cough. Later, the respiratory tract will be inflamed with oedema and it is a phlegm production. This mucus production can accumulate, which covers the bronchial tubes, and in turn obstructs the bronchioles (Christopher M. Evans, 2010).

## Treatment And Patient Care

Respiratory tract infections in diabetics are associated with increased mortality. People with diabetes are four times more likely to die from pneumonia or flu than non-diabetic people (Juliana Casqueiro, 2012). If a person with diabetes has a cough and cold that lasts for more than a week, chronically high blood sugar levels can lead to other complications, such as ketoacidosis, where too much acid accumulates in the blood. It is even more critical for people with diabetes to cope with both their cold and cough symptoms without waiting for them to disappear (Steele, 2014). Like all pharmaceutical formulations, over-the-counter cough syrups contain some active ingredients and some inactive materials that help to give a tasty and cosmetic product. Active and Inactive Ingredients in Classic Cough Syrup can potentially affect blood sugar levels or other critical functions in a diabetic patient. Sugar is the primary inactive ingredient in most cough syrups and, when absorbed in the blood, directly causes a significant increase in blood sugar.

Alcohol consumption can lead to diabetic complications. While many cough syrups also contain alcohol, it can also affect the body's metabolic pathways to raise blood sugar levels. When it comes to active ingredients in cough syrup, the drugs commonly used are dextromethorphan and guaifenesin. These two are considered safe for people with diabetes at prescribed doses. However, most cough syrup can also help other medications like acetaminophen and ibuprofen to relieve pain – these two drugs can have toxic effects on people with diabetes who have kidney complications (Jan Marquard, 2015). Also, ibuprofen also tends to increase the glycemic impact of anti-diabetic medications (HÖRL,

2010). Decongestants and antihistamines in cough syrup can also interfere with the way the body metabolizes sugar, insulin and anti-diabetic medicines in people with diabetes. Conventional sugar-based cough lozenges are also prohibited for people with diabetes. Honey-containing herbal pellets may also affect blood glucose levels (Michael Malone, 2017). Pharmacists can help patients with diabetes by informing you about the availability of these specialized coughs and cold products and guiding them to the appropriate range of over-the-counter products.

Before recommending the use of over-the-counter outcomes for people with diabetes, pharmacists must review the history of allergy and patient history and the drug profile to prevent drug interactions and contraindications and consider whether self-medication is appropriate. In addition, pharmacists should always advise patients on the correct use of these products and remind patients to always follow and avoid the manufacturer's instructions and warnings on the product label. Use of medicines containing sugar or alcohol, if possible. It is essential that pharmacists remind patients that the use of certain medications can affect their blood sugar levels (Shah, 2010). For example, the use of a decongestant, such as pseudoephedrine, should be used with caution in diabetic patients as it may increase blood sugar. Non-pharmacological measures that can alleviate the discomfort of symptoms associated with a cough or cold, such as the use of vaporizers or humidifiers, rest, adequate hydration and relief of congestion, may also be suggested as an alternative (Katherine A. Safka, 2015).

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