Celiac Sprue Disease Essay, Research Paper

What if your doctor told you that eating a certain food say pizza would be devastating to your health? You might not like it, but you’d learn to live with it. But what if it was more than just pizza? What if you were told to avoid all bread, breadcrumbs, and pasta? And dozens of breakfast cereals, canned soups, luncheon meats, and salad dressings? And a wide variety of ice creams, ice cream cones, cookies, cakes, puddings, and pies? And most chewing gum, beer, canned tuna, and hot dogs?

That’s what it’s like for people who have celiac disease. Celiac disease, also known as gluten-sensitive enteropathy, celiac sprue, nontropical sprue, and idiopathic steatorrhea, is a disease whose extensive and variable symptoms challenge physicians to make a correct diagnosis in a timely fashion. The term “celiac sprue” has been applied to a clinical syndrome characterized by signs and symptoms of malabsorption, such as diarrhea and weight loss caused by eating grains. The term “gluten-sensitive enteropathy” more correctly defines the clinical pathologic disease caused by an immune-mediated sensitivity to gluten, a protein found in many cereal grains, principally wheat, barley, rye, and to a lesser degree oats. Most nutritionists agree that gluten is not present in rice, white or sweet potatoes, and corn.

Over 100 years ago a British physician named Samuel Gee described the “coeliac affection.” Dr. Gee observed the syndrome in people of all ages, but especially in children who had chronic diarrhea, weight loss, edema, and a distended abdomen. The led to death unless cured by various diets. Many other physicians followed in the footsteps of this pioneer, prescribing diets based on rice, bananas, and lamb, which often led to symptomatic improvement of these children with celiac sprue.

It wasn’t until the end of World War II that the connection between the consumption of wheat and rye flour and the incidence of celiac sprue was made. Dutch pediatricians noted that during the war, when these flours were in short supply, celiac patients improved and few new cases were seen. After the war, when adequate food supplies were restored to the civilian population, celiac disease reappeared with regularity.

Based on this clinical observation, scientist then determined that a water-insoluble protein component of these grains, gluten, was the substance that damaged the intestine in certain individuals. Not until the 1950s were the characteristic microscopic changes in the lining of the intestine documented. The arrival of techniques to obtain biopsy specimens of the small intestine opened the floodgates of research activity, which taught us much of what we know today about this disease.

Most recently, research has been conducted that describes the immunologic mechanisms causing this type of intestinal injury. The vast majority of patients with celiac sprue possess a particular tissue type, which can be thought of in a similar fashion to each person’s blood type. If one possesses the right, or more correctly, wrong tissue type, there is a likelihood of developing celiac sprue.

The exact mechanism for this phenomenon is unknown, but several theories have been proposed. First, patients with celiac sprue may lack an enzyme necessary to digest toxic fractions of gluten. Second, celiac disease-associated tissue-type antigens are found on the surface of the intestinal cells that face the lumen (the part of the tubular intestine that is exposed to dietary contents). It is believed that these cells may bind with the toxic fractions of gluten. On the surface of the intestinal cell, gluten acts as a foreign substance, eliciting an immune reaction that destroys the intestinal-lining cells. Of course, each individual’s tissue type is inherited. Because of this, if one of a pair of identical twins develops celiac disease, the other invariably will develop it as well.

Approximately 25% of Caucasians, in whom the disease is most common, possess the celiac disease-associated tissue-type antigens, but obviously not all of them develop the disease. Other unknown factors begin the chain reaction of immunologic injury. One possible cause is infection with common agents as viruses. At least one virus has been shown to posses a similar protein composition to toxic fractions of gluten. Theoretically, infection with this virus exposes the immune system to an antigen that is “shared” with the intestinal epithelium, or lining. Long after the virus has disappeared, the immune system continues to attack the body’s own tissue, which produces celiac disease.

Once the immune system is activated in this way, there is progressive destruction of the surface cells that are normally responsible for absorption in the small intestine. The injury is most severe in the proximal intestine (jejunum) and becomes progressively less severe in the more distal intestine (ileum).

The reason for this pattern of distribution is probably due to the fact that gluten is gradually digested and eliminated from the intestinal lumen as the meal literally progresses downstream. The most proximal intestine, called the duodenum, is the primary site for absorption of minerals such as iron, calcium, and magnesium.

Extensive injury to the small bowel results in decreased absorption of nutrients such as carbohydrates, protein, and fat. When these substances are malabsorbed, they act as a laxative, holding water in the intestinal lumen and causing copious diarrhea. Diarrhea also occurs because the immunologic system releases mediators that lead the intestinal lumen to secrete fluid.

As a result of these processes, patients with full-blown celiac sprue have malabsorption of nutrients, minerals, and water. Large quantities of undigested nutrients in the intestine are available for metabolism by bacteria. The bacteria produce gases such as carbon dioxide, methane, and hydrogen, which account for the bloating and gurgling seen and heard in many patients.

Knowing these events it is not difficult to explain the many various manifestations of celiac sprue. However, most people don’t have am easy time with or even access to medical textbooks. To complicate matters, many do not present with classic symptoms, which may vary from mild distention and gas after meals with normal bowel movements to incapacitating malnutrition, diarrhea, and dehydration. Occasionally patients present with signs and symptoms of vitamin and mineral deficiency, such as anemia due to iron, folate, and B12 malabsorption; bone pain and pathologic fractures because of malabsorption of calcium and vitamin D; and growth retardation in children. Some of the more subtle signs and symptoms of celiac sprue include bleeding tendencies, muscle spasms, nerve damage, infertility, impotence, and spontaneous abortion.

Although many individuals with celiac sprue develop diarrhea and bloating as young children when they begin to eat cereals, the diagnosis is often missed when inadvertent but therapeutic dietary manipulations, such as elimination diets, are instituted. However, symptoms may improve spontaneously, and patients may appear to “outgrow” the disease until they are in their 20s or 30s. Occasionally latent celiac sprue is activated by a metabolic stress, such as infection, pregnancy, or surgery. Interestingly, about + of adult-onset sprue patients have a history of diarrhea during childhood.

A doctor who suspects sprue may ask the patient to collect stool samples to determine whether there is malabsorption of nutrients. Several tests can suggest the diagnosis of celiac sprue. Routine blood testing usually shows anemia and low levels of carotene, cholesterol, iron, calcium, and magnesium. Measurement of blood clotting may be abnormally prolonged as a result of malabsorption of the fat-soluble vitamin K. Occasionally physicians order a xylose tolerance test. For this, patients drink a solution of xylose, a type of sugar, and its absorption is quantified by measuring its concentration in the blood or urine.

More recently, blood tests have been developed that measure antibodies directed against gliadin, the toxic fraction of gluten, as well as against other body constituents, such as smooth muscle cells. Unfortunately, these antibodies may be present in healthy individuals and absent in people that have celiac sprue.

The treatment of celiac sprue is straighfoward, at least to the physician. Many patients are told “Just follow a gluten-free diet and you’ll be feeling great in a couple of weeks.” Unfortunately, many otherwise excellent diagnosticians do not understand the implications, complexities, difficulties, and frustrations encountered in this simple order.

Having a brother who was diagnosed with this disease this past summer, I understand more than someone else may how hard it is to follow the diet needed. Yet no one can comprehend how difficult it is except the patient. My brother, who attends Ridgewood High School, eats lunch at school just like everyone else here does. The only difference between his menu and others including pizza, pasta, and sandwiches, is that he may have a type of cold cut (turkey, roast beef, etc.) just rolled together. At home when he wants an after-school snack, he doesn’t even look in the drawer with the chips and the coffee cakes; instead, he reaches for his rice crackers and cheese. When going out to eat, there is a card we have that instructs the chef everything he can and can’t have. Although this is a disease that will stick with him for the rest of his life, he has been doing a great job keeping a positive attitude about the whole thing.