



Nuts, Seeds and Diverticula

By: W. Grant Thompson, M.D., F.R.C.P.C., Emeritus Professor of Medicine, University of Ottawa, Ontario, Canada

The following question was submitted by a *Digestive Health Matters* reader. If you have a question about your digestive health, please contact us at IFFGD, 700 W. Virginia St., #201, Milwaukee, WI 53204. This information is provided for general information and is not intended to replace your doctor's advice.

From New York – Your Summer 2002 newsletter issue contains a statement that I question, regarding diverticulosis and diverticulitis, in an article by W. Grant Thompson, MD titled “Diverticula, Diverticulosis, Diverticulitis: What’s the Difference?” In the article, Dr. Thompson asserts, “The notion that nuts or seeds may lodge in the diverticula and provoke diverticulitis is probably untrue.”

I have had this condition for twenty-five years. On several occasions the ingestion of nuts and seeds and even shredded coconuts has caused the most acute repeated attacks of diverticulitis, marked with acute pain on the left side, bowel movement distress, and a fever. When nuts, seeds, and offending foods were eliminated from my diet I no longer suffered the diverticulitis symptoms described above – except on one or two occasions when due to careless lapses in such dietary precautions, (such as eating an unidentified mixed dish in a darkly-lit restaurant) the attack recurred.

Moreover, a sibling with the same condition experienced precisely the same symptoms after eating seeds and nuts. Only a diet that scrupulously avoids these foods in her case and in mine has prevented subsequent attacks of this kind. I would appreciate some clarification regarding the role of diet in this syndrome with special reference to the question of seeds, nuts, and any other possible offending foods.

Response from Dr. Thompson – The issues presented by the reader are whether or not nuts or seeds in tomatoes, grapes, etc. ingested by individuals with colonic diverticula can cause acute diverticulitis, and whether or not they should be avoided by those who have had such an attack (secondary prevention). By extension one might wonder if all those with diverticular disease should also avoid these foods (primary prevention).

First of all, we need to remind ourselves of the epidemiological perspective. Briefly, 30 to 50 percent of North Americans and Europeans over 60 years of age have diverticula in their colon that cause no symptoms, and unless they have had a colonoscopy or barium enema (the majority have not) they are unaware of the fact. Diverticulitis on the other hand is a relatively uncommon but serious complication that occurs in susceptible populations at a rate of about 10 to 100 cases per 100,000 people per year. Diverticulosis is uncommon in vegetarians and in many non-European societies and diverticulitis is correspondingly rare. African-Americans have a risk of diverticula development that is similar to their European-American compatriots.

A gut diverticulum (singular) is an outpouching of the wall of the gut to form a sac. *Diverticula* (plural) may occur at any level from esophagus to colon. Most individuals who possess colonic diverticula are unaware of them. The condition of having colonic diverticula is called *diverticulosis*. Diverticula are almost always innocent bystanders, and their presence is termed *uncomplicated diverticular disease*. Despite this, colonic diverticula can occasionally become the source of serious illness. These few may bleed or perforate thus becoming *complicated diverticular disease*. Inflammation occurs when a diverticulum, usually in the left colon, bursts leaking bacteria-rich feces into the abdominal cavity (peritoneum). The resulting *diverticulitis* is usually confined to the surface of the adjacent colon producing an acute, sometimes devastating illness characterized by severe abdominal pain in the left lower part of the abdomen, fever, and prostration.

The epidemiological data has convinced many that diverticula in the colon result from increased intra colonic pressure that occurs in populations consuming a low-fiber (low-residue diet). Thus most textbooks espouse a high-fiber diet for diverticular disease, but not during acute attacks of diverticulitis. The rationale is that dietary fiber

reduces intra colonic pressure and softens the stool, thereby reducing the risk of *fecalith* (hard mass of dried feces) formation.

It is unknown why only a few people with colonic diverticula suffer attacks of diverticulitis. Pathologists observe that an acute attack is associated with a hard ball of fecal matter (fecalith) lodged in one diverticulum. The pressure on the very thin wall of the diverticulum damages the tissue and leads to a small perforation causing an infection that may or may not be localized to the site of the perforation. Some have wondered if the ingestion of particulate matter such as nuts or seeds might favor this process, perhaps by contributing to the fecalith or themselves lodging in the thin-walled outpouching. The suggestion that nuts and seeds be abstained from in diverticular disease rests solely on this premise and on anecdotal reports such as that of the reader.

However, many doubt that this is true and the recommendation to avoid particulate substances would complicate the recommended high-fiber diet. It has been stated that there is no known case of nuts or seeds plugging up a diverticulum and causing an attack of diverticulitis¹. Most of several texts of gastroenterology, medicine, and pathophysiology that are available in our university library do not mention nuts or seeds in their descriptions of the pathogenesis, prevention, or treatment of diverticulosis and diverticulitis.²⁻⁶ One that did⁷ stated there was no evidence to support the role of seeds and nuts in the pathogenesis of diverticulitis. Two medical sites on the Internet support this view.^{1,8} In a survey of 373 fellows of the American College of Colorectal Surgeons,⁹ fifty-three percent believed that avoidance of seeds and nuts was of no value and more were undecided.

In Medical science, it is very difficult to prove a negative. Nevertheless, no data support a role for particulate foods in the pathogenesis of diverticulitis. Lacking a pathological basis the hypothesis that seeds and nuts cause diverticulitis will have to rely for proof on clinical grounds. The clinical trial required to demonstrate such a role would be expensive, lengthy, and very difficult to do. Therefore, it seems that the definitive answer to the reader's question will not be available soon. The decision whether or not to exclude seeds or nuts as primary or secondary prevention of diverticulitis will boil down to common sense and personal choice.

Primary prevention would be difficult. Advising nut and seed abstinence would be impractical for the great majority of people with diverticulosis and no diverticulitis. Even the use of high-fiber diets for primary prevention is a difficult proposition for those who know they have diverticula but no symptoms. For secondary prevention the evidence suggests that a high fiber diet is important and that might include particulate matter other than seeds and nuts. For the reader and his or her sibling who clearly link ingestion of these substances to their attacks of diverticulitis, common sense dictates that they avoid them.

For the rest, in the absence of a similar experience there is no evidence that such a dietary intervention is necessary and it may interfere with the more important consumption of high fiber foods.

References

1. Arai R. Seeds, Sprinkles and diverticulosis. http://www.ivillagehealth.com/experts/digestive/qas/0,,232662_168447,00.html. (*iVillage* 2000;accessed 2003).
2. The gastrointestinal tract. In: Rubin E, Farber JL, editors. *Pathology*. Philadelphia: Lippincott-Raven, 2001:669-755.
3. The gastrointestinal tract. In: Cotran RS, et al, editors. *Robbins pathological basis of disease*. Mosby, 1998:755-829.
4. Diverticular disease. In: Feldman M, et al, editors. *Sleisenger and Fortran's gastrointestinal and liver disease*. Philadelphia: Saunders, 1998:1790-1797.
5. Isselbacher KJ, Epstein A. Diverticular disease. In: Braunwald E, et al, editors. *Harrison's principles of internal medicine*. New York: McGraw-Hill, 2002:1695-1702.
6. Smith AN. Diverticular Disease of the Colon. In: Phillips SF, Pemberton JH, Shorter RG, editors. *The large intestine*. New York: Raven Press, 1991:549-577.
7. Huether SE. Alterations of digestive function. In: McCance KL, Huether SE, editors. *Pathophysiology*. Mosby, 2002:1261-1313.
8. Massey BT. Diverticulosis vs. diverticulitis. www.healthlink.mcw.edu/article/1013634026.html. (*HealthLink* 2002;accessed 2003).
9. Schecter S, Mulvey J, Eisenstat TE. Management of uncomplicated acute diverticulitis: results of a survey. *Dis Colon Rectum* 1999; 42:470-475.

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