A presentation on individualized treatment of pancreatic disease based on specific disease etiology offered a glimpse of the future at the 2015 American College of Gastroenterology Scientific Session on Sunday, October 18, 2015.

It is not trivial for anyone of us to say that we are unique. As a corollary, each and every patient should be approached utilizing the same philosophy. Treating all individuals for a particular illness in exactly the same way is destined to fail. Treating any disease without tailoring the therapy to a patient’s specific needs would not be good medicine. A novel framework—one that identifies data specific to every whole, unique person (disease modeling)—is being studied as a potentially successful future approach to care.

On Sunday, October 18, 2015 at the 2015 American College of Gastroenterology Annual Scientific Meeting, Dr Tyler Stevens, assistant professor and interventional pancreatologist from the department of gastroenterology at the Cleveland Clinic in Cleveland, Ohio discussed what a personalized approach to the diagnosis and treatment of pancreatic disease would look like and what the immediate and more long-term benefits are.

Pancreatitits, like diabetes and heart disease, is not the result of a single gene or mutation. Rather, it the result of a complex interplay among multiple genes whose phenotypes are modulated by lifestyle and the environment. This complexity explains why only a minority of patients who drink alcohol to excess get pancreatitis and also why individuals who smoke are more likely to develop pancreatic calcifications and cancer.

Chronic pancreatitis is a complex inflammatory disorder, the final common pathway of myriad etiologies. The disease is suspected when there are pancreatic calcifications on imaging, beading of the main pancreatic duct, ectatic side ductal branches, or an abnormal secretin stimulation test. Classification of the etiologies of pancreatitis is based on the TIGAR-O system which stands for Toxic-metabolic, obstructive, Idiopathic, Genetic, Autoimmune, Recurrent and severe acute pancreatitis. Five major susceptibility genes (CFTR, SPINK1, PRSS1, CTRC, CASR) have been linked to chronic pancreatitis. Thus, the specific etiology of pancreatitis in any individual will be unique and so may lead to more precise, focused treatment. For example, main pancreatic duct obstruction from a tumor, stones or pancreatic divisum should be treated by endoscopic retrograde cholangiopancreatography, lithotripsy, or surgery. CFTR mutations causing low hydrostatic duct pressure can be treated with duct resistance reduction, or in the future, with CFTR protein processing medications currently under investigation. Acinar cell hyperstimulation (the result of mutations in SPINK1) and calcium dysregulation can be treated with calcium channel blockers or the definitive treatment of hyperparathyroidism for hypercalcemia. Similarly, IgG4-related autoimmune pancreatitis can be treated with B cell inhibitors and or corticosteroids.

Patients with chronic pancreatitis require a multidisciplinary approach that is also tailored to an individual’s specific symptoms, for example diarrhea related to exocrine dysfunction; diabetes secondary to endocrine dysfunction; pain; and depression. Interventions should target lifestyle changes with abstinence from alcohol and smoking, pain management with a menu of visceral nerve interventions (celiac and splanchnic blocks, splanchnic RFA), and psychiatric counseling.

A personalized approach might also save money by eliminating unnecessary tests—and by preventing unnecessary cholecystectomies. Blaming the gall bladder when a genetic mutation is responsible—then performing surgery—is the wrong approach. Although personalized pancreatic medicine is in its infancy, tailored regimens based on disease modeling have unlimited potential. Success will finally allow targeted therapy for patients with pancreatic pathology before they experience intractable pain syndromes requiring narcotics for relief.
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