Gastroparesis

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Synonyms

- delayed gastric emptying
- gastric atony
- gastric dysmotility
- gastric stasis
- gastrointestinal autonomic neuropathy
- gastroparesis diabeticorum
- gastropathy
- severe functional dyspepsia

Subdivisions

- diabetic gastroparesis
- idiopathic gastroparesis
- post-surgical gastroparesis

Diseases Overview

Gastroparesis (abbreviated as GP) represents a clinical syndrome characterized by sluggish emptying of solid food (and more rarely, liquid nutrients) from the stomach, which causes persistent digestive symptoms especially nausea and primarily affects young to middle-aged women, but is also known to affect younger children and males. Diagnosis is made based upon a radiographic gastric emptying test. Diabetics and those acquiring gastroparesis for unknown (or, idiopathic) causes represent the two largest groups of gastroparetic patients; however, numerous etiologies (both rare and common) can lead to a gastroparesis syndrome.

Gastroparesis is also known as delayed gastric emptying and is an old term that does not adequately describe all the motor impairments that may occur within the gastroparetic stomach. Furthermore, there is no expert agreement on the use of the term, gastroparesis. Some specialists will reserve the term, gastroparesis, for grossly impaired emptying of the stomach while retaining the label of delayed gastric emptying, or functional dyspepsia (non-ulcer dyspepsia), for less pronounced evidence of impaired emptying. These terms are all very subjective. There is no scientific basis by which to separate functional dyspepsia from classical gastroparesis except by symptom intensity. In both conditions, there is significant overlap in treatment, symptomatology and underlying physiological disturbances of stomach function.

For the most part, the finding of delayed emptying (gastric stasis) provides a "marker" for a gastric motility problem. Regardless, the symptoms generated by the stomach dysmotility greatly impair quality of life for the vast majority of patients and disable about 1 in 10 patients with the condition.

While delayed emptying of the stomach is the clinical feature of gastroparesis, the relationship between the degree of delay in emptying and the intensity of digestive symptoms does not always match. For instance, some diabetics may exhibit pronounced gastric stasis yet suffer very little from the classical gastroparetic symptoms of: nausea, vomiting, reflux, abdominal pain, bloating, fullness, and loss of appetite. Rather, erratic blood-glucose control and life-threatening hypoglycemic episodes may be the only indication of diabetic gastroparesis. In another subset of patients (diabetic and non-diabetic) who suffer from disabling nausea that is to the degree that their ability to eat, sleep or carry out activities of daily living is disrupted gastric emptying may be normal, near normal, or intermittently delayed. In such cases, a gastric neuro-electrical dysfunction, or gastric dysrhythmia (commonly found associated with gastroparesis syndrome), may be at fault.

Therefore, these disorders of functional dyspepsia, gastric dysrhythms, and gastroparesis are all descriptive labels sharing similar symptoms and perhaps representing a similar entity of disordered gastric neuromuscular function. For this reason, a more encompassing term, gastropathy, can be used interchangeably with gastroparesis.

Signs & Symptoms

The digestive symptom profile of nausea, vomiting, abdominal pain, reflux, bloating, a feeling of fullness after a few bites of food (early satiety), and anorexia can vary in patients both in combination and severity. A small percentage of patients who live with poorly managed symptoms despite numerous treatment interventions, and an inability to meet their nutritional needs represent the extreme end of the gastroparesis spectrum of gastric failure. For most, the prevailing symptom experience is persistent nausea that often intensifies a few hours after eating. Nausea may become so intense as to trigger vomiting even after a few sips of water. Vomiting, also commonly reported, typically starts a couple of hours after eating so that the food is still recognizable and undigested. Chronic abdominal pain, which may also occur, is felt to result from visceral neuropathy. The pain, often diffuse, is described as burning, shearing or gnawing in character. The nature of pain may also be complex with

some individuals experiencing acute pain, trigged by eating, layered on top of the chronic pain. This acute, sharp pain may be related to intestinal cramping and/or to spasms in the upper portion of the stomach caused by its failure to relax and "accommodate" the just-eaten food. As well, a gall bladder that is sluggish to empty (paresis) is commonly found in association with a poorly emptying stomach. This may all add to the pain experienced soon after eating. A poorly emptying stomach additionally predisposes patients to regurgitation of solid food, as well as gastric esophageal reflux disease (GERD). The reflux may range from mild through to severe. GERD complications can create esophageal spasm (also called non-cardiac chest pain) and can add to the burden of chronic pain. In severe cases, reflux aspiration pneumonitis compounds the clinical picture.

Other symptoms include belching and bloating-again, developing soon after meal ingestion and lasting for hours-along with visible abdominal distention. The distention and bloating may push up against the diaphragm making breathing uncomfortable. Loss of appetite (anorexia), a conscious avoidance of food in an attempt to moderate digestive symptoms, or food craving that are all frequently reported as well.

Vomiting, which can result in the life-threatening conditions of dehydration and electrolyte imbalances, represents the most troubling of all the digestive symptoms related to gastroparesis. Repeated vomiting usually necessitates a visit to the emergency room. Concomitant spiraling malnourishment accompanies frequent, chronic vomiting and precipitates repeated and prolonged hospital admissions. Approximately 5 to 10% of insulin-dependent diabetics may progress to severe symptomatic gastroparesis.

In the majority of insulin-dependent diabetics, gastroparesis is often overlooked and under-diagnosed, especially in its early stages. The characteristics of poor glucose control and acid reflux are often the only signatures of delayed gastric emptying. The typical picture seen in the diabetic gastroparetic stomach is low blood-glucose levels at bedtime with very high blood-glucose levels by the next morning. This situation is interspersed with days of good blood-sugar control. Some scientific studies have found diabetic gastroparesis to correlate with autonomic neuropathy (diabetic autonomic neuropathy, or DAN and cardiac autonomic neuropathy, or CAN), but not with the duration of diabetes, metabolic control or other chronic complications. It is to be noted that acute hyperglycemia profoundly retards gastric emptying.

If DAN is present, then regaining control of blood glucose can be enormously challenging. For Type 2 diabetics (non-insulin dependent), absorption of oral hypoglycemic agents may be very unpredictable due to the delayed gastric emptying.

Gastroparetic symptoms in most patients show either a pattern of cycling with flare-ups, or daily occurrences persisting for years.

Causes

The etiologies (causes) for gastroparesis are extensive and varied. Reports from one tertiary referral center found that out of their 146 patients with gastroparesis: 36% were idiopathic (unknown causes), 29% were diabetic, 13% were post-surgical, 7.5% had Parkinson's disease and 4.8% had collagen diseases. Any disease of metabolic, neurological (psychiatric, brainstem, autonomic including sympathetic and parasympathetic or enteric), or connective tissue (autoimmune) origin has the potential to disrupt gastric neural circuitry. Regional areas of the stomach may show various degrees of dysfunction, such as: failure of fundic relaxation, weakened postprandial antral contractions, pylorospasm, and / or gastric hyperalgesia; yet, rarely is gastroparesis restricted exclusively to the stomach. If perturbation of stomach function occurs, this will indirectly impact function in many other regions along the GI tract due to the complex enteric reflexes and neuronal relays which exist throughout the gastrointestinal (GI) system. The ability of the GI track to "cross-talk" is essential for coordination of normal digestion. Gastroparesis then is a complex, multifactor, chronic, digestive disease state with possible genetic, physiological, immune, psychological, social and environmental interplays.

Gastroparesis has been documented to occur as a sequel to viral gastroenteritis, slowly resolving over one to two years. However, any upper-gut infection of bacterial, parasitic or viral origin has the potential to disrupt, for prolonged periods of time, gastric motility. In gastroparesis patients, and others with unexplained chronic nausea and vomiting, one recent study reported up to 80% of 121 patients were found to have structural abnormalities of enteric nerves on full-thickness tissue biopsies of the duodenum.

Examples of gastroparesis include insulin-dependent diabetes, post-vagotomy (surgical), anorexia nervosa and bulimia, chronic liver or renal failure, and chronic pancreatitis. Gastroparesis may also be induced by medications, or associated with total parenteral nutrition or related to bone marrow and other organ transplants. Additional causes include paraneoplastic syndrome, mitochondrial disorders, abnormal gastric pacemaker activity (gastric dysrhythmias), visceral neuropathies, (for example: Guillain-Barre syndrome) and visceral myopathies (for example: systemic scleroderma)

[For more information on any of the listed disorders, choose the specific name as your search term in the Rare Disease Database.]

A great deal of research into the enteric nervous system has begun to identify specific cellular and neuronal abnormalities associated in gastroparesis such as:

+ Loss of ICC (interstitial cells of Cajal)*

+ Loss of neuronal nitric oxide synthase (nNOS)

+ Enteric neuronal degeneration (visceral neuropathy)

+ Smooth muscle disease (myopathy), often related to collagen diseases

*Interstitial cells of Cajal (ICC) are critical components for GI motility. Various types of ICCs have been identified. They are involved in the generation and propagation of electrical rhythmic activity (hence, they are known as the "pacemaker" cells) and serve as well to bridge communication, and amplify the signals from neuronal inputs to mechanical smooth muscle action (transduction).

So far, there is no consensus on how to classify histopathological findings.

Other significant serological abnormalities have been documented in diabetic, post-surgical and idiopathic cases of gastroparesis. In one case study a high prevalence (89%) of acquired and congenital hypercoagulable defects was documented, which would predispose patients to arterial and venous clots. Coagulation evaluation would then be prudent in patients with severe gastroparesis, especially in high-risk thrombophilic situations such as hospitalization, prolonged intravenous access, and surgery.

Affected Populations

Gastroparesis demonstrates a gender bias affecting more women than men. Approximately 80% of idiopathic cases are women. Idiopathic gastroparesis may be linked to an as yet-to-be-elucidated enteric autoimmune disease. The prevalence of delayed gastric emptying in Type 1 diabetics has been reported to be 50% and in type 2 diabetics, reports range from 30% to 50%. Post surgical gastroparesis is a recognized as inadvertent vagal nerve damage or entrapment following upper abdominal surgery, examples are: fundoplication for the treatment of GERD, bariatric surgery, peptic ulcer surgery, anterior approach for spinal surgery (scoliosis), heart, lung transplant, or pancreatic surgery.

Related Disorders

A stomach motor disturbance known as "dumping syndrome" whereby food or liquids empty too quickly from the stomach can present with similar symptoms as are found in gastroparesis. Other disorders that may clinically present as gastroparesis (gastritis, gastric ulcers, pyloric stenosis, celiac disease, and GI obstructions) need to be ruled out.

Diagnosis

A diagnosis of gastroparesis is made based upon a thorough clinical evaluation, a detailed patient history, and a variety of specialized tests. Tests may first be performed to rule out other causes of delayed gastric emptying such as obstruction of the gastrointestinal tract. Additional tests are then performed to confirm a diagnosis of gastroparesis.

Tests to rule out other causes of delayed gastric emptying include routine blood tests, an upper gastrointestinal endoscopy, a barium gastrointestinal series with small-bowel-follow-through, and an abdominal ultrasound. During an upper GI endoscopy, a thin, flexible tube (endoscope) is run down the throat to the stomach and the small intestines. The tube has a tiny camera attached to it that allows a physician to search for abnormalities and obstructions within the gastrointestinal tract. During an ultrasound, reflected sound waves create an image of the abdomen.

An upper endosocpic procedure may lead to a serendipitous diagnosis of gastroparesis through the discovery of identifiable food within the stomach after the pre-procedure overnight fast.

The radionuclide (scintigraphy) solid-phase gastric emptying test (GET), the gold standard for diagnosing gastroparesis, can now be measured using only four images: baseline, 1-hour, 2-hour, and 4-hour. The GET, a non-invasive test, is widely available and accessible. The test involves eating food that contains a small amount of radioactive material (radioisotope). This tiny dose of radiation can be seen on a gamma camera (much like an X-ray machine), but is not dangerous. The scans allow a physician to determine the rate at which food leaves the stomach. Many other methods are now being employed to track gastric emptying times; for instance a gastric breath test (not in common use in North America) and a new encapsulated recording device, called SmartPill has the ability to measure gastric pH, GI luminal pressures, and determine gastric and intestinal transit time.

Other diagnostic tests for GP can include electrogastrography (EGG). This is often referred to as the EKG of the stomach. EGG can serve as a screening tool and is complementary to the gastric emptying test. The EGG is capable of detecting specific gastric electrical rhythm abnormalities and indirectly gives an indication of the integrity of the stomach's ICC network. This test is generally done using a non-invasive method with cutaneous (skin) leads. A less common method of administering an EGG is a direct, invasive means via mucosa (either during endoscopy or serosa recordings can be done at the time of gastric electrical stimulation placement for the treatment of nausea and vomiting).

Many patients with symptoms of gastroparesis often have related nutritional deficiencies and disorders. Nutritional laboratory measurements are important. Laboratory tests to include are albumin, pre-albumin, hemoglobin A1C (on all diabetic patients), ferritin, B-12, and 25-hydroxy vitamin D.

Standard Therapies

Treatment

Treating the underlying cause of gastroparesis (such as tightening up blood glucose control in diabetes) is usually the first step in treating individuals with gastroparesis. The specific therapeutic techniques used depend upon several factors, including the severity of the disorder. Therapies that are used to treat individuals with gastroparesis include non-pharmacological steps, dietary modification, medications that stimulate gastric emptying (prokinetics), medications that reduce vomiting (antiemetics), medications for controlling pain and intestinal spasms, and surgery.

Some researchers have proposed a classification system to help determine appropriate treatment options. Grade 1, or mild gastroparesis, is characterized by symptoms that come and go and can easily be controlled by dietary modification and by avoiding medications that slow gastric emptying. Grade 2, or compensated gastroparesis, is characterized by moderately severe symptoms. Individuals with Grade 2 gastroparesis are treated with medications that stimulate gastric emptying and medications

that reduce vomiting; such individuals require hospitalization only infrequently. Grade 3, or gastric failure, is characterized by individuals who do not respond to medications used to treat gastroparesis. These individuals cannot maintain proper nutrition or hydration. Required therapies may include intravenous fluids and medications and nutrition, or surgery. Individuals with Grade 3 gastroparesis often require hospitalization.

Non-pharmacological interventions include: liquid vitamin supplements (including optimal levels of vitamin D), discontinuation of smoking and alcohol use, learning techniques of deep relaxation, the use of acupuncture or acupoint stimulation, and reviewing all medications and supplements with a pharmacist to insure current regimen is not contributing to delayed gastric emptying. Other non-pharmacological therapies for GP include autonomic retraining; often related to autogenic feedback training developed by NASA for space motion sickness, autonomic retraining has shown promise in selected patients.

Dietary changes include eating five to six small meals each day and avoiding high-insoluble fiber and high-fat foods, both of which can slow down stomach emptying. Lactose intolerance is common in gastroparesis, so avoidance of dairy is helpful, but fermented milk (yogurt) is fine. Some individuals do better with liquid or pureed foods while avoiding difficult-to-digest solid foods. Liquid nutritional supplements are also an excellent source of additional calories and proteins to make up for nutritional deficiencies resulting from a poor appetite.

Medications that stimulate gastric emptying are called prokinetics. They are the primary treatment for individuals with gastroparesis. First-line medical treatment begins with the dopamine blockers. Of the two drugs, metoclopromide (Reglan) and domperidone (Motilitum), domperidone has a much better safety profile. Metoclopromide has serious neurological side effects, including irreversible tartive dyskinesia. Domperidone is now available in the United States through the FDA's Investigational New Drug protocol. Next are the motilin agonists, such as erythromycin. Unfortunately, motilin agonists have a number of potential drug interactions and lose effectiveness over time. Cholinergic agonists, either direct-acting or via 5HT receptors, such as bethanecol (Urecholine) and cisapride (Propulsid), have been limited by the side-effect profile (especially, in the case of cisapride, for possible drug interactions) or are not used widely. Tegaserod is no longer available for the treatment of gastroparesis.

The most common symptom of gastroparesis is nausea and it can be enormously challenging to control. Often, medications used to reduce nausea and vomiting "antiemetics" are used in combinations and in conjunction with prokinetics. Common antiemetics used are prochlorperazine (Compazine), promethazine (Phenergan), and ondansetron (Zofran).

Abdominal pain is best managed by a pain specialty clinic where a selection of analgesics with the least impact on slowing the GI tract can be made.

Keeping a good bowel routine and avoiding constipation helps in the management of upper digestive symptoms of gastroparesis.

Other clinical management issues to consider are metabolic bone disease (all gastroparetic patients are at risk) and routine monitoring for small bowel bacterial overgrowth (SBBO). SBBO commonly occurs in gastroparesis and may greatly contribute to upper digestive symptoms, malnutrition and vitamin deficiencies. Antibiotics and perhaps probiotics help to keep SBBO in check.

Bezoars (congealed food residue left in the stomach) are a rare occurrence in gastroparesis but are found in more severe cases of gastric stasis. Whenever a patient's usual pattern of nausea and vomiting rises to a new intensity, then suspicion of a bezoar should be investigated via endoscopy.

Individuals with severe gastroparesis, or those who do not respond to medications, may require enteral or parenteral feeding to compensate for nutritional deficiencies and to prevent dehydration. Enteral feeding refers to a feeding tube that transverses from the skin and comes to rest directly in either the stomach (G-tube) or small intestine (J-tube, for jejeunal) or a G-J tube (a tube that enters the stomach and transverses the pylorus to enter the small intestine). Feeding tubes are generally needed for long durations (> 3 months) and selection of a tube should be made with this in mind.

Parenteral feeding refers to the implantation of a small, thin tube (catheter) into a vein so that nutrients and fluids can be delivered directly into the bloodstream.

Enteral Tubes for GP Patients

Enteral tubes are often not successful for feeding (due to slow infusion rates related to stomach and small bowel dysmotilities) but may be helpful as a conduit for free water or medications. In fact, medications are more evenly absorbed when administered via enteral tubes. For diabetics, improved blood glucose can be achieved when nutrients bypass the gastroparetic stomach and are then absorbed in a more predictable way for matching insulin needs.

Additionally, enteral tubes-either gastric or small intestinal may be useful as venting, particularly for patients with localized dilated segments of small bowel or stomach (watermelon stomach) commonly the result of visceral myopathies.

Finally, small bowel bacterial overgrowth may be treated more successfully if antibiotics can be given by tube, especially if the patient suffers from vomiting.

IV Access for Gastroparesis

Intravenous access may be a life-saving delivery of medication and nutrition for some severely affected patients. However, IV access may also be life-threatening due to infection and/or thrombosis. Recent work has revealed that many GP patients are hypercoagulable and in some centers, all GP patients who receive IV access also undergo a hypercoagulable evaluation. Those that are at highest risk for thrombosis are then usually started on anticoagulation. Gastroparesis, itself, may pose special

problems for anticoagulation, as absorption of oral anticoagulants may be erratic. A good short-term plan is to use sub-cutaneous, low molecular-weight heparin, at least until nausea and vomiting can be controlled.

Surgical Interventions for Gastroparesis

Surgical interventions for palliation of symptoms of severe diabetic and idiopathic gastroparesis such as: pyloroplasty, or elimination of the stomach through near total gastrectomy (Billroth I, Antrectomy, Roux-En-Y); show little improvement in symptom management. A review of the literature by Reardon et al, showed that of 12 patients, only 3 had resolution of their symptoms, with the majority having no improvement or only temporary improvements, or a worsening of symptoms to include bilious vomiting.

Experts agree that the entire stomach is affected in severe gastroparesis; therefore, treatment by partial gastrectomy is unsatisfactory as a method to attempt to ameliorate symptoms. Treatment by total gastrectomy is used as a palliative measure; however, it is a major surgical procedure which carries risks. This procedure has significant morbidity, and a mortality rate of 3.5%. Also, once the stomach is removed, the option no longer exists to take advantage of any new pharmacological therapies that may come along; and dependency on enteral / parenteral nutrition is permanent. Also, nausea remains a very troubling symptom even after total gastrectomy.

Investigational Therapies

Researchers have been studying the use of a procedure known as gastric electrical stimulation (GES) for the treatment of individuals with gastroparesis. In 2000, the U.S. Food and Drug Administration (FDA) approved the use of Enterra Therapy System (GES) under the “humanitarian device exemption” for the treatment of diabetic and idiopathic gastroparesis. This special regulatory category was created for devices that would benefit fewer than 3,000 individuals. The FDA restricts its use to medical centers with an Institutional Review Board (IRB). The Enterra Therapy System is manufactured by Medtronic (www.medtronic.com). To date, approximately 7,000 patients have been implanted with GES.

The use of GES in diabetics has a high rate of success for reducing symptoms of nausea and vomiting. However, success in the idiopathic patients is less predictable. Therefore, following the clinical practice for other neurostimulating devices (examples: spinal cord stimulation, sacral nerve stimulation, deep brain stimulation) whereby a short-term trial of stimulation is used to help identify “responders” to therapy prior to permanent surgical placement; it is possible to conduct a short-term trial of GES to help identify who will benefit from this procedure.

Temporary GES is carried out via the use of endoscopy. Under direct visualization, one wire lead in secured into the stomach, mucosal wall. The wire exits from one side of the nose (nostril) and is connected to the pulse generator (box) which is secured on the outside of the body. A minimum 3 day

trail helps determine if the individual patient will benefit from GES.

In permanent GES a tiny pulse generator and two wire leads with small electrode ends are implanted in the stomach. Similar to a cardiac pacemaker, the generator delivers a series of electrical pulses. Initial studies have shown that GES causes a reduction in nausea and vomiting in individuals with gastroparesis. Additional positive effects of GES include improvement in GI symptoms, gastric emptying, and pancreatic function. Improved autonomic and enteric nervous system function and quality of life, as well as reduction in hospitalizations and medical care costs, and improvements in survival have also been shown. GES has been used for individuals with diabetic and idiopathic gastroparesis as well as for individuals who have not responded to other therapies.

The nutritional effects of GES are related primarily to decreased GI symptoms and are accompanied by increased weight and body mass index, increased albumin, an improvement in pancreatic function, and a decreased need for parenteral nutrition and enteral tubes. Decreased costs and improved quality of life, discussed above, are undoubtedly related to the improvements in GI and nutritional status. Finally, GES is a reversible procedure and carries no mortality risk.

Botulinum toxin is a protein that is toxic to nerves. It is best known as a cosmetic therapy that temporarily eliminates the wrinkles of aging. Some researchers have used botulinum toxin to treat individuals with gastroparesis. Through the use of a thin, flexible tube (endoscope), botulinum toxin is injected directly into the pyloric muscle, which controls how much food passes from the stomach into the small intestines. Botulinum toxin relaxes the pyloric muscle allowing a greater amount food to pass into the small intestines. The effects of botulinum toxin are temporary (generally three to nine months). Research studies have shown mixed results for providing improvement in symptoms for gastroparetic patients. More research is necessary to determine the long-term safety and effectiveness of this potential therapy for individuals with gastroparesis.

A new class of drugs, called ghrenlin receptor agonist, are currently proceeding through clinical trials in the treatment of diabetics with gastroparesis. These orally administered, promotility drugs are showing promise.

Information on current clinical trials is posted on the Internet at www.clinicaltrials.gov. All studies receiving U.S. Government funding, and some supported by private industry, are posted on this government web site.

For information about clinical trials being conducted at the NIH Clinical Center in Bethesda, MD, contact the NIH Patient Recruitment Office:

Tollfree: (800) 411-1222

TTY: (866) 411-1010

Email: prpl@cc.nih.gov

For information about clinical trials sponsored by private sources, contact:

www.centerwatch.com

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Programs & Resources

RareCare® Assistance Programs

NORD strives to open new assistance programs as funding allows. If we don't have a program for you now, please continue to check back with us.

Additional Assistance Programs

MedicAlert Assistance Program

NORD and MedicAlert Foundation have teamed up on a new program to provide protection to rare disease patients in emergency situations.
https://rarediseases.org/patient-assistance-programs/medicalert-assistance-program/

Rare Disease Educational Support Program

Ensuring that patients and caregivers are armed with the tools they need to live their best lives while managing their rare condition is a vital part of NORD's mission.
https://rarediseases.org/patient-assistance-programs/rare-disease-educational-support/

Rare Caregiver Respite Program

This first-of-its-kind assistance program is designed for caregivers of a child or adult diagnosed with a rare disorder.
https://rarediseases.org/patient-assistance-programs/caregiver-respite/

Patient Organizations

Association of Gastrointestinal Motility Disorders, Inc. (AGMD)

NORD Member

Email: info@agmdhope.org

https://rarediseases.org/organizations/association-of-gastrointestinal-motility-disorders-inc-agmd/

International Foundation for Gastrointestinal Disorders (IFFGD)

NORD Member

Email: iffgd@iffgd.org

https://rarediseases.org/organizations/international-foundation-for-gastrointestinal-disorders-iffgd/

IAMRARE® Patient Registry

Powered by NORD, the IAMRARE Registry Platform® is driving transformative change in the study of rare disease. With input from doctors, researchers, and the US Food & Drug Administration, NORD has created IAMRARE to facilitate patient-powered natural history studies to shape rare disease research

and treatments. The ultimate goal of IAMRARE is to unite patients and research communities in the improvement of care and drug development.