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Case Report

Combined Dopamine Agonists to Ameliorate Idiopathic Treatment Refractory Gastroparesis

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Abstract

Gastroparesis is a condition with significant morbidity related to a problem in gastric emptying caused by dysmotility rather than a mechanical obstruction. The only approved drug in the United States for this condition is metoclopramide which is used to block dopamine receptors. Unfortunately, the drug is frequently not effective. Dextroamphetamine sulfate, a dopamine agonist, has been found to effectively treat a wide variety of chronic conditions that were refractory to standard therapy. The hypothetical mechanism of action is that there is a cellular permeability defect leading to failure to prevent unwanted substances from crossing a mucosal barrier leading to inflammation or mitochondrial dysfunction.

Dextroamphetamine has been successful in treating gastric motility disorders, e.g., gastroparesis and also small bowel motility problems, e.g., pseudointestinal obstruction. The case presented here required a high dosage of dextroamphetamine that fully corrected her very severe symptoms of gastroparesis for 20 years while taking 150mg amphetamine salts. When she was forced to reduce the dosage to 40% of her present dosage by her pharmacist, her severe symptoms returned. However, by adding another dopamine agonist, carbidopa-levodopa, she once again had complete remission. Since dextroamphetamine not only releases more dopamine from sympathetic nerve fibers, but also norepinephrine and epinephrine, one could not be sure that the release of dopamine was responsible for the improvement. The addition of a pure dopaminergic drug with no effect on catecholamines helps to support the hypothesis that the mechanism of action is by releasing dopamine. This seems at odds with the only approved drugs being dopamine receptor antagonists. One hypothesis is that when the cause of the gastroparesis is increased cellular permeability, correcting that defect prevents the state of gastric dysmotility so that inhibiting some of the symptoms by using dopamine receptor antagonists is not needed.

Keywords: Gastroparesis, Increased cellular permeability syndrome, Dopamine agonist, Dopamine receptor antagonists

Introduction

Gastroparesis is a pathologic condition related to a gastric motility disorder that produces symptoms of a mechanical blockage, but it is actually a functional blockage related to a physiological problem of dysmotility [1]. The symptoms can be mild or severe, according to the severity of the delay in gastric emptying.

The classic symptoms of gastroparesis include nausea, vomiting, early satiety, post- prandial fullness, and upper abdominal pain [2]. These symptoms are generally chronic but tolerable [2]. However, there may be sporadic but severe exacerbations that could require hospitalization [2].

The physiology of normal gastric mobility has been summarized by *Mekaroonkamol, et al* [1]. These authors explain in detail the complex etiology of gastric dysmotility, including intraduodenal hypomotility, impaired fundic accommodation and pylorospasm [1]. The understanding of these physiological disturbances could

lead to drugs targeting the gastric hormones and enzymes and neurotransmitters that eventually lead to gastroparesis [1].

Peptides, e.g., ghrelin and motilin act as receptors in the stomach and small intestine to stimulate muscular contraction. The result of peptide-receptor interaction contributes to the regulation of interdigestive gastrointestinal motility, which contributes to rhythmic contraction to accelerate gastric emptying [3]. This results in increased appetite [3]. Thus, prokinetics, e.g., relamorelin and erythromycin, have been used to treat gastroparesis [4-6]. These drugs have proven especially beneficial when the etiology of the gastroparesis is associated with constipation and is either related to diabetic mellitus or Parkinson's disease [7-8]. In the study by *Ye, et al* 19.4% of the cases were associated with Type I diabetes mellitus and 14.3% Type 2 [9].

Interestingly, the most commonly used drugs to treat gastroparesis are anti-dopaminergic receptor drugs. Metoclopramide is the most commonly used drug in the world for gastroparesis [1]. It is the only approved medication by the Food and Drug Administration (FDA) of the United States for treating gastroparesis [1]. However, its long-term use seems to be limited by the development of extrapyramidal side effects that seems to occur with long-term use [10].

Another anti-dopaminergic receptor drug, domperidone is restricted in the United States because of the risk of QTC prolongation on the electrocardiogram that could lead to sudden cardiac death [11]. The frequency of this risk is low so that its use is approved in Asia and Europe. One advantage of domperidone over metoclopramide is that it does not increase the risk of extrapyramidal side effects because in contrast to metoclopramide it does not cross the blood brain barrier [12].

Another anti-dopaminergic receptor drug levosulpiride increases lower esophageal sphincter pressure and inhibits dopamine D2 receptors on both stomach and small intestine [13].

The physiology of gastric motility function and potential areas where malfunction can lead to dysmotility of the stomach leading to gastroparesis or a milder dysmotility condition called dyspepsia has been summarized by *Mekaroonkamol, et al* [1]. *Tonini, et al* in their review article on gastroparesis explain the potential sites of action where these dopamine receptor antagonists exert their action to benefit the symptoms of gastroparesis [14]. They attribute the beneficial effect of these drugs on the nausea and vomiting related to both binding enteric dopamine receptors and dopamine receptors in the chemo receptor trigger zone in the area postrema in the central nervous system [14]. This leads to blocking dopamine action, and they claim that gastroparesis may be related to the physiological effect of dopamine in suppressing gastrointestinal motility [14].

Interestingly, in the epidemiologic study by *Ye, et al* they list Parkinson's disease as an etiological factor in their study, but it occurred in only about 1% of their cases [7]. Parkinson's disease is one of dopamine depletion so this would seem in conflict with *Tonini, et al's* concept that dopamine activity contributes to the inhibition of gastric motility. However, it is possible that it was not the Parkinson's disease per se that was responsible for the gastroparesis in the *Ye, et al* [9] study, but possibly treatment with carbidopa-levodopa. *Mekaroonkamol, et al* list a table of various etiological factors associated with gastroparesis, and similar to *Ye, et al* include Parkinson's disease as a cause. However, in that same table, they list medication induced factors leading to gastroparesis, and in addition to opioids, anti-cholinergic agents, tricyclic anti-depressants, calcium channel blockers, octreotide, lithium, glucagon-like peptide-1 analogs, cyclosporine, levodopa is also listed as a possible etiologic factor [1].

Mekaroonkamol, et al besides listing Parkinson's disorder under the etiological factors for gastroparesis and neurological disorders also lists autoimmune gastrointestinal dysmotility [1]. They also list scleroderma and mixed connective tissue disease under musculoskeletal disorders as etiological factors [1]. *Ye, et al* list scleroderma and systemic lupus erythematosus as an etiologic factor in 0.6% of the cases [7].

The sympathomimetic amine dextroamphetamine sulfate has for many years shown very good efficacy for providing significant improvement of a variety of autoimmune disorders that were resistant to conventional therapy [15-32]. *Ye, et al* [9] listed idiopathic gastroparesis as an etiologic factor in 39.5% of cases of gastroparesis, and possibly the majority of these are of autoimmune nature.

The reason for choosing dextroamphetamine for treating autoimmune disorders and other conditions associated with pain from a known source, autoimmune source, or idiopathic source is theoretically to decrease cellular permeability by releasing dopamine from sympathetic nerve fibers thus correcting the defect in the mucosal barrier from allowing infiltration of unwanted substances into tissues.

A case report showing a clinical benefit only demonstrates that a given therapy may work, but it is not clear whether it would only be effective in a rare case or the majority of cases. It is even possible that the condition spontaneously had a remission fortuitously while receiving a potential therapy. Nevertheless, the knowledge of the possible efficacy of a drug that seemed to treat a condition e.g., gastroparesis, could influence a clinician to try this therapy if it was a very convincing case, and if that therapy had less risks or side effects than other potential therapies, whether approved or used off label.

Dextroamphetamine sulfate was used to treat these various medical conditions because of a hypothetical model based on some experimentation that most chronic conditions have as their basis, the infiltration of irritants across the mucosal barrier related to a cellular permeability defect which frequently will cause inflammation and pain, but may also cause by infiltration into

mitochondria leading to skeletal and smooth muscle dysfunction [33-34]. The research that led to the thought of using drugs that could decrease cellular permeability was conducted to study the mechanism of successful embryo implantation and fetal survival hoping that cancer would "borrow" a similar mechanism and could thus lead to novel anticancer medications [35-38].

The first case report of a 29-year-old woman with gastroparesis whose symptoms markedly improved following treatment with dextroamphetamine sulfate, despite failing to respond to metoclopramide, ondansetron, and ethromycin was published in 2006 [39]. Interestingly, the dextroamphetamine helped her lose a considerable amount of weight related to idiopathic edema and eradicated hot flashes despite her euestrogenic status [39]. Dextroamphetamine sulfate has been shown to ameliorate vasomotor symptoms in normal estrogenic women and to help some patients lose weight who were not successful with dieting [40-42].

Dextroamphetamine sulfate successfully treated constant upper abdominal pain that was exacerbated by eating and caused such early satiety in a young woman (age 22) that she could hardly eat resulting in a 35-pound weight loss from 110 pounds to 75 pounds [43]. She also had infrequent bowel movements, 1-2 per week. Over several weeks the pain markedly improved as did her early satiety and constipation following treatment with dextroamphetamine. In two months, this woman gained back 25 pounds to her desired weight of 100 pounds [49].

A teenager suffered from severe constipation, usually not having a bowel movement with an interval shorter than 9 days and frequently she went 6 to 8 weeks without one. She obviously had some type of bowel motility problem. She had an appointment with a gastroenterologist to conduct tests to decipher the problem, but she responded within one month of taking lisdexamfetamine so that the consult was canceled [44].

Our group had treated several other cases of gastroparesis in which the large majority have improved with sympathomimetic amine therapy. Our policy is to only publish case reports when it is the first such case to respond to a given therapy. Sometimes we will submit a case report for publication if there is something interesting that happened with that patient that adds to the understanding of the pathophysiology of that condition or offers a new insight on treatment.

For example, we published the case of a 46-year-old male who had gastroparesis manifested by nausea, vomiting, abdominal distention, and upper abdominal pain. He was diagnosed with gastroparesis by a delayed emptying time [37]. In his case, metoclopramide was ineffective and caused a sensation of severe burning throughout his body associated with erythema of his neck and chest and severe headaches [37].

This reaction was found to be related to failure to adequately metabolize the metoclopramide sufficiently leading to very high blood levels on this drug. He was found to be heterozygous for the CPY2D6 41 reduced activity variant. He did, however, have a moderate initial response to domperidone, but overtime the drug lost its efficacy, and it was stopped. He suffered for 7 years, but at age 53 he consulted our group with the knowledge of this abnormal drug metabolic disorder. He was started on a lower dosage of 5 mg extended-release capsule of amphetamine salts, instead of the usual starting dosage of 15 mg amphetamine salts upon arising and at noon (there is 9.5mg of dextroamphetamine sulfate in 15mg amphetamine salts).

The dextroamphetamine provided immediate relief of his gastroparesis symptoms in one day. However, he experienced chest pain (he had a history of coronary artery disease). The dosage was decreased to 2.5 mg, and he was symptom-free for 8 years at the time of the reporting of this case report [37].

The case that is presented here is another example of very severe treatment refractory gastroparesis who responded very well to dextroamphetamine sulfate, but not until the dosage was the highest with which we have ever used to treat a patient with the various manifestations of the increased cellular permeability syndrome [45]. She is reportable again because a change in her treatment regimen helps to demonstrate that it is probably the dopamine agonist effect of dextroamphetamine sulfate that has led to the successful treatment of so many different chronic treatment refractory conditions rather than its effect on increasing catecholamine secretion.

Case Report

The woman at age 33 had a gastric bypass procedure but started to gain weight three years later, and nine years later complained of frequent vomiting, diarrhea, and abdominal pain. Multiple gastroenterologists agreed that she was suffering from gastroparesis confirmed by testing showing delayed gastric emptying in the absence of an obstruction [45]. Interestingly, despite hardly being able to eat without vomiting, she continued to gain weight. Over a 3-year period the pain and swelling were constantly present, but she would have severe exacerbations requiring her to be hospitalized at least 6 times per year. She consulted our practice, but it was not until she reached 150 mg of amphetamine salts that she attained 98% relief of her symptoms.

Her marked relief of the symptoms of gastroparesis continued while taking 150 mg of amphetamine salts. We suspected that she over metabolized the drug because her heart rate was only 68 beats per minute. At the 2-year mark, she tried to see if the treatment was still needed and stopped the amphetamine salt. Her symptoms returned, and before they could get too severe, she restarted the medication.

Her improvement lasted 20 years, and she had no side effects. She did not have one hospital admission for gastroparesis during that time. However, a new pharmacist refused to fill the 150 mg

prescription and stated that he/she would only allow 60 mg per day.

Her symptoms progressively became worse, then became very severe on 60 mg amphetamine salts, leading to her first hospitalization for this problem in 20 years. She reconsulted our group. We suggested to keep the amphetamine salts immediate release tablet at 30mg upon arising and 30 mg at noon but adding carbidopa-levodopa 10/100mg twice per day. Though the carbidopa-levodopa gave her mild nausea for 30 minutes after taking it, the patient has markedly improved the symptoms of gastroparesis, which she states that they are 98% better. This improvement has lasted for 6 months so far with no exacerbations.

Discussion

The events that have happened since the previous publication in 2017 supports the contention that at least some cases, and possibly most cases of gastroparesis, may be related to increased cellular permeability leading to the infusion of unwanted elements that disrupt smooth muscle function. Based on physiological studies on gastrointestinal motility, the beneficial effect of dopamine agonists seems counterintuitive.

However, it may explain why dopamine receptor antagonists e.g., metoclopramide and domperidone have not demonstrated great efficacy in the general treated population with this disorder and were not effective in the few cases described here who were successfully treated with dextroamphetamine sulfate.

There are several possible explanations. For one, it could be that in some cases the problem is related to increased cellular permeability where possibly correcting the defect in mitochondrial dysfunction related to infusion of unwanted substances into the mitochondria by the use of a dopamine agonist is the treatment of choice. However, possibly in some cases where there is a neuropathological neuropathological etiology e.g., possibly diabetic mellitus, there may be clinical benefit found from using a dopamine receptor antagonist e.g., metoclopramide or domperidone. Mekaroonkanol shows a figure where other pharmacologic therapies may target to improve gastroparesis [1]. These pharmacologic therapies, besides anti-dopaminergic receptor drugs, show potential targets for serotonin modulators e.g., prucalopride, mosapride, levosulpride, velusetrag and prucalopride, or neurokinin not for 1 inhibitors e.g., aprepitant and tradipitant, or ghrelin not for agonists e.g., relamorelin, for cannabinoid receptor agonists e.g., medical marijuana, and for various antidepressants e.g., serotonergic anti-depressants, and even antipsychotic medication e.g., haloperidol and levosulpiride [1].

Related to the generalized failure of the standard but mostly non-approved medical therapies i.e., anti-dopaminergic receptor drugs, serotonin modulators, neurokinin-1 inhibitors ghrelin agonists, cannabinoid receptor agonist, and anti-depressants, surgical procedures have risen in popularity to treat gastroparesis. The most promising endoscopic therapy is gastric peroral endoscopic pyloromyotomy [46-49].

Another surgical procedure is the insertion of neurostimulators in the gastric wall [50]. Unfortunately, the efficacy of this procedure has not been shown to be superior to sham controls [51].

More extreme surgical procedures e.g., surgical pyloroplasty , total and subtotal gastrectomy, and subtotal gastrectomy with Roux-en-Y gastrojejunostomy reconstruction should only be considered in patients severely suffering and not responding to medical therapy. Actually, consideration for some of these extreme surgical measures was suggested by some of the consulting physicians of the subject of this case report because of the degree to which she was suffering.

There are many reasons why we chose to write a follow-up in this case of gastroparesis

a) To demonstrate how effective sympathomimetic amines e.g., dextroamphetamine sulfate can be for treating a very severe case of treatment refractory gastroparesis by showing long lasting (20 years) marked improvement to the degree that it no longer existed.

b) To fulfill the requirements of Koch's postulates by demonstrating the return of symptoms when treatment is stopped, or as in this case, when the dosage of the drug is markedly reduced. The case satisfied the final requirement of Koch's postulates in that the symptoms disappeared once treatment was started again, or, as in this case, adding another dopamine agonist to the reduced dosage of dextroamphetamine. As previously mentioned, when a given therapy seems to be effective, but is a single case report, one cannot be sure if there was a spontaneous remission or by some chance, she was no longer exposed to some causative agent. The follow-up of this case established that there are some cases of gastroparesis that will have great long lasting amelioration following treatment with dopamine agonists.

c) The fact that carbidopa-levodopa, though a dopamine agonist, is not a sympathomimetic amine, and thus does not also increase the secretion of catecholamines, supports the original concept for choosing dextroamphetamine to treat the various manifestations of the increased cellular permeability syndrome i.e., to decrease cellular permeability by dopamine agonists and thus correct pathological conditions related to infiltration into tissues of unwanted substances.

The importance of showing that it is the release of dopamine that is needed to correct problems related to transversing mucosal barriers by unwanted elements is not just important for academic issues, but has practical value in that it opens a new field for medications other than amphetamines to treat this widely present, but relatively unknown condition of increased cellular permeability. The authors' have extensive experience in the use of dextroamphetamine sulfate in treating thousands of patients for at least 45 years. In fact, our first case report of using

dextroamphetamine sulfate for a medical disorder was in 2 women who had severe treatment refractory urticaria in 1984 [52]. In fact, one of these 2 women was covered on most of her body by urticaria almost every day for 7 years, and she had complete remission which has continued for 40 years. The one exception was after about 10 years of never missing one day of her dextroamphetamine, the mail order did not arrive, but because there was evidence of being shipped, it could not be rewritten for that month. There were no withdrawal symptoms from sudden cessation of the drug (which is the rule rather than the exception). However, within 3 days, she was covered once again in hives. This persisted the entire month but dissipated within one week when the dextroamphetamine was started again. Thus, this woman also fulfilled Koch's postulate.

Despite extensive use of amphetamines for 45 years, not one person has ever become addicted in the pharmacological dosages that have been used, nor has anyone ever been hospitalized at any time for adverse side effects. There are some who have had side effects; e.g., persistent insomnia (transient insomnia is common when started the drug for the first time) rare tachycardia, and not so infrequent, impatience and anger issues. Many of the side effects are from the catecholamine release and thus could be obviated by more pure dopamine agonists, e.g., cabergoline or carbidopa-levodopa. However, dopamine agonists may have side effects of nausea or lightheadedness, but it is always good to have a potential drug alternative.

Nevertheless, for reasons at least unknown to the authors there seems to be a "black cloud" about the use of amphetamines for medical usage. In the state of New Jersey, where one of the 2 offices are located and the location of Cooper Medical School of Rowan University, there is an unusual law that states that one cannot use a drug with a class II narcotic restriction (which is shockingly the situation for amphetamines) off-label. Thus, patients without concomitant attention deficit disorder would have to be evaluated and treated in the Pennsylvania office where there is no such restriction. For 30 years there was no problem until around 2021, the Attorney General interpreted the law to preclude any patient going to another state to receive amphetamines because when they would be coming back to New Jersey, they are technically breaking the law. The interpretation has been considered unconstitutional by many others in the judicial field, but nevertheless the former Attorney General 's interpretation still stands. Thus, if the case reported here lived in New Jersey, despite the availability of a safe effective medical therapy with dextroamphetamine she would have been forced to undergo extreme surgical procedures that not only carry a mortality risk but would still leave her great morbidity, just hopefully less than what she was presently experiencing. What is interesting is that not only does the state of New Jersey allow the use of medical marijuana and psychodelic mushrooms for treating medical conditions, but patients are allowed to acquire them through "registered clinics" without even having a consult with a physician first. Thus, this law influenced our group to find non-amphetamine drugs that increases dopamine effect for our New Jersey patients suffering from a variety of medical conditions.

This misguided perceived notion of risks of amphetamines is not necessarily all related to governmental policy, but to treating physicians themselves. Another case that has been previously reported will substantiate this aforementioned observation of physician bias also, but will serve to also support a "take-home" message from this case report, and that is to also consider that some drugs are over metabolized. Thus, one should not give up on a drug that is the best choice if one needs to go above the normal highest recommended dosage as long as there are no noted side effects. One extremely cachectic man who sought our opinion who was terminal and was advised that death from his "autoimmune" idiopathic pancreatitis would probably be within 3 months." He was treated with a combination of oxycodone, OxyContin, and fentanyl that was barely giving him any relief from his severe constant abdominal pain. He was treated with dextroamphetamine which was gradually raised to 90 mg per day of amphetamine tablets [17]. After 8 months of amphetamine therapy, he was pain-free and had gained 50 pounds and had good energy. Even more importantly, he had completely weaned off all of his opiates 2 months before [17]. He lived in Pennsylvania, but 1.5 hours from our Pennsylvania office. We suggested that he could ask his pain management physician who prescribed the 3 opiate combination to take over his management because his office was only 15 minutes away. The pain management physician refused to take over his case stating that he would not touch "Adderall" with a 10-foot pole.

The patient who is the subject of this case report of gastroparesis lives in Maryland. However, despite observing how ill she was before, and noting how well she had responded to dextroamphetamine, not only did her family physician refuse to take over her case, but for years had encouraged her to not merely reduce the dosage (as insisted by the pharmacist) but to stop the drug altogether. This underscores the need to explore the efficacy of other dopamine agonists because there is no evidence to date that without the support of pharmaceutical companies or state agencies the multitude of publications of a plethora of medical conditions failing conventional therapy, but showing marked amelioration with dextroamphetamine or similar drugs e.g., lisdexamfetamine, prejudice against amphetamine use seems to be waxing rather than waning.

There have been case reports showing the benefit of the dopamine agonist cabergoline in ameliorating dysmenorrhea, constant vaginal burning, headaches, pain from severe carpal tunnel syndrome, and fibromyalgia [53-56]. The dosage of cabergoline that was typically used to treat the clinical manifestations of the increased cellular permeability syndrome is 0.5 mg 3 times a week. We chose for this case carbidopa-levodopa over cabergoline because of the degree of suffering of this patient because we have evidence that cabergoline may be somewhat less effective than either dextroamphetamine or carbidopa-levodopa [57]. Thus, we wanted to try to relieve the symptoms as quickly as possible.

d) This case illustrates one more clinical pearl in treating gastroparesis or other manifestations of the increased cellular

permeability syndrome and that is that one can add another dopamine agonist to dextroamphetamine to achieve clinical improvement if, as in this case, the new pharmacist refused to fill the prescription for dextroamphetamine at the higher dosage, or if there was benefit at a certain dosage but some side effects. Sometimes the side effects from sympathomimetic amines are not purely from increased dopaminergic effect, but its concomitant release of catecholamines also from sympathetic nerve fibers. One woman had great improvement of her severe stomatodynia (burning mouth syndrome) but had very mild, almost unnoticeable, tardive dyskinesia. She was willing to stay on the same dosage since the facial tics were hardly noticeable. We suggested that she decrease the dosage of dextroamphetamine and add cabergoline. The tardive dyskinesia completely dissipated with continued amelioration of the burning mouth syndrome with this drug combination [58].

The hypothesis that unwanted substances infiltrating mitochondria and disrupting physiological motility function is speculative to explain the beneficial action of dopamine agonists in improving gastroparesis. However, support for this hypothesis was provided by another case who had the Mitochondrial Encephalopathy Lactic Acid Stroke-Like Syndrome (MELAS) was wheelchair ridden for 25 years, and was able to walk without any mechanical aids after one month of treatment with dextroamphetamine [59].

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Conflict of Interest

None.

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