Case Report: Acute Intermittent Porphyria in a 21-Year-Old Active Duty Male

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Abstract

Acute Intermittent Porphyria (AIP) is one of a group of rare metabolic disorders arising from reduced activity of any of the enzymes in the heme biosynthetic pathway. The porphyrias can be very difficult for the practitioner to understand. There are several types of porphyrias, which have been known by various different names and are classified from different perspectives[1] based on where the defective synthesis site is, or what the clinical manifestations are. Since practitioners rarely encounter this disease process, it is commonly not considered in the differential diagnoses. AIP can be confused with other causes of acute abdominal disorders such as appendicitis with peritonitis or nephrolithiasis. Patients with AIP typically give a history of constipation, fatigue, irritability, and insomnia that precede their acute attack. Symptoms occur intermittently in some patients with acute attacks lasting for several days or longer and were usually followed by complete recovery. This case report deals with an initial presentation of AIP in an otherwise healthy 21-year-old active duty male Soldier. Clinical presentation, diagnosis and treatment are discussed as is a brief historical anecdote.

Key Words
Porphyria, emergency department, medication, urine

Introduction

Acute Intermittent Porphyria (AIP) is one of a group of rare metabolic disorders arising from reduced activity of any of the enzymes in the heme biosynthetic pathway. This disorder may be inherited from a defect in the gene encoding these enzymes or acquired from a toxin acting on one of the enzymes. (See table 1 for a list of common toxins). AIP is uncommon; most practitioners see only a handful, if any, cases during their careers. It is rarely diagnosed in the emergency department and Tintinalli's sixth edition of *Emergency Medicine: A Comprehensive Study Guide* devotes only three paragraphs to the disorder.

This case report highlights AIP. This 21-year-old active duty male Soldier will demonstrates how difficult it can be to make a diagnosis. This report will also discuss how to separate this confusing clinical entity from other more acute problems commonly seen in the emergency department.

Case Report

A 21-year-old Hispanic active duty Soldier was slumped over in a wheel chair in the triage area of Womack Army Medical Center's Emergency Department (ED). The triage nurse, one of the most experienced RNs in the emergency department, was worried about nephrolithiasis. The patient was grimacing, with wave-like pain and fit the doorway diagnosis of "sick" versus "not-sick". He was brought immediately to a bed for evaluation and pain control.

In the ED he described his pain as severe, 10/10, and primarily in his left lower quadrant. He stated that the pain started several hours prior while at work. Pepto-Bismol® did not relieve his discomfort. His pain was initially mild, but rapidly progressed to severe and was worse with movement. He denied any history of trauma or history of similar pain. He denied any change in bowel habits, vomiting or diarrhea, but was slightly nauseated. He had not had any lower urinary tract symptoms. He had felt feverish. He denied any recent travel.

His past medical and past surgical history was unremarkable. He had no family history of inflammatory bowel disease. His social history was negative for alcohol, tobacco or drugs. The review of symptoms was positive only for anorexia that coincided with the onset of his pain.

Vital signs in the ED: temperature 98.3, rising to 100.4; pulse of 80, respirations of 26 and blood pressure of 157/102. He was ill appearing. His physical examination was positive for a 2/6 systolic ejection murmur at the lower left sternal border. He had some mild tenderness over the suprapubic area of his otherwise soft, non-tender abdomen. His bowel sounds were normal. There were no rashes or other skin lesions and he did not have any cognitive or other personality changes. A complete blood count revealed a white blood cell (WBC) count of 16,000 and his basic metabolic panel and urinalysis were normal. A computed tomography (CT) scan of the abdomen and pelvis was negative with the exception of a mildly enlarged spleen. He received Dilaudid® (hydromorphone) 1mg with 12.5mg of Phenergan® for his pain. He required several doses of medication throughout his ED stay until his pain was brought under control.

After gathering initial data, general surgery was consulted to further evaluate the patient's fever and abdominal symptoms. After evaluating the patient, reviewing the CT, and laboratory studies, the consultant agreed that the patient was acutely ill and obviously in pain, but did not think that the Soldier required surgery. The surgeon recommended considering AIP in the differential diagnosis and suggested consulting the medicine service.

Family medicine was on call and came to the ED and agreed that porphyria was in the differential and admitted the patient. He was placed on the general medicine ward and Dilaudid® and Phenergan® were continued PRN (pro re nata) for pain. The patient was kept NPO (nulla per os) he was evaluated by general surgery again in the morning.

A 24-hour urine specimen was collected and elevations were seen in coproporphyrin I at 65ug (normal 0-24ug/24 hours), coproporphyrin III at 194ug/24 hours (normal 0-74ug/24 hours), hexacaroxlporphyrin at 4ug/24 hours (normal 0-1ug/24 hours), and P-carboxylporphyrin at 4ug/24 hours (normal 0-3ug/24 hours). The patient's uroporphyrin was 22ug/24 hours (normal 0-24ug/24 hours) and heptcarxylporphyrin was 4ug/24 hours (normal 0-4ug/24 hours). Porphobilinogen (PBG) and D- aminolevulinic acid (ALA) 24 hour levels were not tested during the acute phase of his illness. Both were tested a week later and were within normal limits.

The patient did very well after his admission, tolerating a normal diet by the following late afternoon and became pain free. He was discharged later the next day with follow-up to his primary care provider at an outlying clinic.

Discussion

The porphyrias can be very difficult for the practitioner to understand. So difficult in fact that some believe that the "werewolves" of the Middle Ages were in fact suffering from porphyria. Great Britain's King George III, born in 1738, had normal health until 1765 when he developed intermittent depression and agitation that resolved on its own. His health worsened in 1788 when he had another bout of odd behavior to the point that he would run up and down the halls of his castle naked. His urine was described by his physicians as being bluish red. He was bled frequently without relief. Also, to draw the "poison" out of his brain, his physicians tried blistering his forehead with similar lackluster results. Finally, acknowledging that conservative therapy may have a place, he was locked in his room, in a strait jacket with no fire for warmth. He improved shortly thereafter. Unfortunately, his bouts of illness became more frequent and he was removed from the throne and died blind and demented in 1820. It was thought in the 1960s that Queen Victoria, as well as her daughter and granddaughter, may also have had porphyria. Permission was obtained to exhume the bodies of the daughter and granddaughter and testing revealed that they each had an intrinsic mutation in the protoporphyrinogen oxidase gene; this mutation could cause variegate porphyria which, like AIP, would cause neurovisceral symptoms.[2]

Acute porphyrias include, in order of prevalence, acute intermittent porphyria (AIP), variegate porphyria (VP), hereditary coproporphyria (HCP), and the exceedingly rare δ-aminolevulinic acid dehydratase (ALAD)-deficiency porphyria. Cutaneous porphyrias include porphyria cutanea tarda, erythropoietic protoporphyria (EPP), and the extremely rare hepatoerythropoietic porphyria and congenital erythropoietic porphyria. The acute porphyrias variegate porphyria and hereditary coproporphyria also have cutaneous manifestations.[1,3,4] They are classified from different perspectives[1] based on how they present, where the defective synthesis site is located or by clinical manifestations. Furthermore, diagnosis can be made more confusing because the defective enzyme may have more than one name. Finally, there is disagreement among the experts about when to suspect, how to diagnose, and how to interpret the results in acute porphyria.

Of all the porphyrias, AIP is the most common and presents with the most serious consequences. It is an inherited autosomal dominant disorder due to a genetic enzyme defect that remains largely clinically silent in most patients who carry the trait. On average, of 100 patients with the genetic defect, only 10-20 secrete excess porphyrin precursors. Of these 10-20 patients, only one or two have symptoms. In patients with AIP, the function of porphobilinogen-deaminase is 40-60% of normal. With the advent of molecular technique, the genetic defect clearly is more common than symptomatic AIP.

The classic inducers of porphyria are chemicals or situations that boost heme synthesis. This includes fasting and many medications. Although very large lists of "safe" and "unsafe" drugs exist, many of these are based on anecdotes or laboratory evidence and do not meet strict criteria. In general, drugs that lead to increased activity of the hepatic P450 system, such as phenobarbital, sulfonamides, estrogens, and alcohol, are associated with porphyria.

AIP clinically affects women more often than men. Symptoms usually begin when patients are in their late teens or early twenties.[5] The disorder is caused by a deficiency of porphobilinogen aminase activity, leading to increased excretion of aminolevulinic acid and porphobilinogen in the urine.

The prevalence of AIP geographically varies with the highest incidence reported in England, Ireland and Scandinavia. The estimated gene prevalence in the United States is 1 in 10-20,000; however, most persons do not exhibit clinical features of the disease. In Sweden, the gene prevalence is 7.7 per 10,000 persons with a clinical presentation of AIP in 1.5 persons in 100,000. A higher incidence of AIP seems to appear in persons with psychiatric illness – approximately 2.1 per 100,000 in the United States.[4]

Clinical Presentation

Patients with AIP typically give a history of constipation, fatigue, irritability, and insomnia that precede their acute attack. Symptoms occur intermittently in some patients with acute attacks lasting for several days or longer, usually followed by complete recovery. Attacks are rare before puberty. There is often no family history of porphyria.

Abdominal pain and vomiting are very common early presenting symptoms (as seen in this case study). The pain may be excruciating and is disproportionate to abdominal tenderness. The pain may also be diffuse and poorly localized and colicky in nature. Abdominal manifestations may result from effects on visceral nerves or from local vaso-constrictive ischemia. Because there is no inflammation, the abdomen is non-tender, without peritoneal signs. Tenderness, fever, and leukocytosis are generally absent or mild since abdominal symptoms are neurologic rather than inflammatory. Although most patients have constipation, increased bowel sounds and diarrhea may also be present. Dysuria and bladder dysfunction rarely occur. Recurrent attacks tend to be similar in a given patient. Bowel distention may develop as a result of paralytic ileus. The urine is red or reddish brown and positive for porphobilinogen during an attack. Other presenting symptoms may include pain in the extremities, head, neck, or chest; muscle weakness; and sensory loss.[6,7]

Common conditions that can mimic AIP include: abdominal abscess, abdominal hernias, acute mesenteric ischemia, adrenal carcinoma, adrenal crisis, aortic dissection, appendicitis, chronic pelvic pain, colonic obstruction, constipation, diverticulitis, cholecystitis, pyelonephritis, empyema, endometriosis, esophagitis, factitious disorder, fibromyalgia, gastric outlet obstruction, gastritis, acute hypertension, ileus, intestinal motility disorders, irritable bowel syndrome, nephrolithiasis, nerve entrapment syndromes,

ovarian cysts, pancreatitis, pelvic inflammatory disease, and manic depressive illness.

Diagnosis

The single, most important lesson to be drawn from this case report is that it is easy to misdiagnose acute intermittent porphyria. Because it is not a commonly encountered disease, it is easy to not consider AIP in the differential diagnosis. AIP can be confused with other causes of acute abdominal pain such as appendicitis with peritonitis or, as in this patient, nephrolithiasis. Because patients can also experience lower extremity weakness with back pain, spinal cord compression may also be considered. In older patients, an aortic dissection must be considered in any patient presenting with abdominal pain and lower extremity weakness. An expanding aneurysm causing abdominal pain and through pressure on spinal arteries can cause lower extremity weakness mimicking AIP. Patients may also be misdiagnosed as having either a neurologic or psychiatric disorder. Patients who present frequently to the emergency department may be labeled as "drug seekers" and dismissed as such.[8]

One of the cardinal signs of AIP during a full-blown attack is the presence of red or reddish brown urine. A urine specimen should be examined in patients with abdominal pain of unknown cause, especially if severe constipation, vomiting, tachycardia, muscle weakness, bulbar involvement, or mental symptoms occur.

If porphyria is suspected, the urine is analyzed for PBG using a rapid qualitative or semi-quantitative determination. A positive result or high clinical suspicion necessitates quantitative ALA and PBG measurements preferentially obtained from the same specimen. Levels of PBG and ALA greater than five times normal indicate an acute porphyric attack unless the patient is a gene carrier with increased porphyrin precursor excretion even during the latent phase of the disorder.

If urinary PBG and ALA are normal, an alternative diagnosis must be considered. Elevated ALA with normal or slightly increased PBG suggests lead poisoning or aminolevulinic-deficiency porphyria. Analysis of a 24-hour old urine specimen is not useful. Instead, a random urine specimen is used in which PBG and ALA concentrations are corrected for dilution by relating to the creatinine concentration of the sample. Electrolytes and magnesium and phosphorous should be measured. Hyponatremia may be present from excessive vomiting or diarrhea with hypotonic fluid replacement or from the syndrome of inappropriate antidiuretic hormone secretion (SIADH).[6]

Management

The management of AIP is two-fold, involving treatment of the acute attack and prevention of future attacks. Treatment of the acute attack is multi-factorial. Patients should be hospitalized for control of acute symptoms and all unsafe and potentially unsafe agents should be stopped immediately. Updated lists are available on the internet at (www.porphyriafoundation.com) and (www.uq.edu.au/porphyria). Alcohol and tobacco should be stopped as well. The work-up includes a thorough investigation for concurrent infections as well as other diseases and if found, aggressively treated. Preventive treatment includes avoidance of drugs known or suspected to exacerbate porphyria and adequate nutritional intake. Anderson et al. suggest nasal or subcutaneous administration of long-acting agonistic analogues of luteinizing hormone-releasing hormone (LHRH) inhibit ovulation and greatly reduce the incidence of perimenstrual attacks of AIP in some women with cyclic exacerbations of the disease.[9] Anderson also reports that a regimen of intravenous heme therapy for prevention of attacks has not been established, but 3mg/kg, given once or twice weekly, has been effective in some patients.[10]

Hypertension and electrolyte disturbances especially hyponatremia due to SIADH should be promptly treated. Serum electrolytes, creatinine, and magnesium levels should be checked at least daily.

Pain should be managed aggressively with narcotic analgesic drugs usually required. This patient did very well with hydromorphone 1mg IV as needed every two hours. Additionally, small to moderate doses of a phenothiazine are indicated for nausea, vomiting, anxiety, and restlessness. Adrenergic blockers for hypertension and symptomatic tachycardia can be used carefully. Bedside spirometry should be available to detect early bulbar paralysis. A falling vital capacity should prompt consideration of mechanical support of respiration and admission to the ICU. Severe cases should be treated with intravenous administration of carbohydrate, initiated with dextrose to provide a minimum of 300-400g of carbohydrate/day. Intravenous glucose alone (10%, at least 300g daily) may resolve mild attacks (mild pain, no paresis, or hyponatremia).

The use of intravenous heme preparations, to repress ALAS-N activity, is most effective in reducing ALA and PBG excretion and in curtailing acute neurovisceral attacks in the hepatic porphyrias. It is recommended if there has been unsatisfactory improvement of the acute attack following the administration of carbohydrate for at least two days. Cimetidine treatment may be offered if standard treatment modalities have failed, or when hemin treatment is not available. Such treatment may be a more cost-effective and more easily administered alternative to hemin treatment.

AIP and Military Service

Army Regulation 40-501, Standards of Medical Fitness, Chapter 2, Physical Standards for Enlistment, Appointment and Induction, paragraph 8(j) states that porphyria, along with other endocrine or metabolic disorders such as cystic fibrosis and amyloidosis, prevent satisfactory performance of duty and require frequent or prolonged treatment are disqualifying.[11]

Chapter 3-41 lists porphyria as cause to start the Medical Evaluation Board (MEB) process. Primarily this is because porphyria's recurrence obviously interferes with successful performance, may require geographic assignment limitations, or requires medication for control that requires frequent monitoring by a physician due to debilitating or serious side-effects. Porphyria is also listed as a cause for medical unfitness for flying duties in Chapter 4.[12]

In response to the concerns voiced by Vietnam veterans and their families, Congress called upon the National Academy of Sciences (NAS) to review the scientific evidence on the possible health effects, including the development of porphyria cutanea tarda (PCT), secondary to exposure to Agent Orange, a herbicide and defoliant used by the United States military in Vietnam from 1962 until 1971, and other herbicides (Public Law 102-4, enacted on 6 February 1991). The creation of the first NAS Institute of

Medicine committee in 1992 underscored the critical importance of approaching these questions from a nonpartisan scientific standpoint.[13]

Medical research indeed implicated Agent Orange in the development of PCT. Veterans Affairs Secretary Jesse Brown issued a finding in the 1994 *Federal Register* that "the credible evidence for an association (between agent orange and PCT) outweighs the credible evidence against an association". Veterans who have developed PCT within one year of service in Vietnam are considered to have a service connected disability of at least 10%.[14]

Conclusion

Acute attacks of AIP are often precipitated by drugs like barbiturates, sulfonmides, sulfones, chloroquin, griseofulvin, diphenyl hydantoin (and many other drugs), acute infections, and over-indulgence in alcohol. In this patient none of those factors were identified. AIP remains a very difficult diagnosis to formulate. It should be considered in any patient who presents with acute abdominal pain that does not fit another diagnostic picture. Any patient with sudden onset of pain, reddish colored urine or rapidly progressing paralysis should be evaluated for porphyria by quantitative ALA and PBG measurement. Patients suspected of having AIP should be hospitalized during the work-up. Any evidence of a falling vital capacity on bedside spirometry should prompt consideration of mechanical support of respiration and admission to the ICU.

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Table 1

		Maleate	
	Cyclophosphamide	Maprotiline HCl	
	Cycloserine	Mebeverine HCl	
	Cyclosporin	*Mecillinam	
	Danazol	*Medroxyprogesterone	
	*Dapsone	[Mefenamic acid]	
	Dexfenfluramine	Megestrol acetate	*Pivampicillin
	Dextropropoxyphene	*Mephenytoin	*Pivmecillinam
	Diazepam	Mepivacaine	Prazepam
Alcuronium	*Dichloralphenazone	*Meprobamate	Prenylamine
*Alphaxalone	*Diclofenac Na	Mercaptopurine	*Prilocaine
Alphadolone	Dienoestrol	Mercury compounds	*Primidone
Alprazolam	Diethylpropion	Mestranol	[Probenecid]
Aluminium	Dihydralazine	[Metapramine HCl]	*Progesterone
Preparations	*Dihydroergotamine	Methamphetamine	Progabide
Amidopyrine	Diltiazem	Methohexitone	Promethazine
Aminoglutethimide	*Dimenhydrinate	Methotrexate	[Propanidid]
Aminophylline	*Diphenhydramine	Methoxyflurane	*Pyrazinamide
Amiodarone	[Dothiepin HCl]	Methsuximide	Pyrrocaine
*Amitriptyline	Doxycycline	*Methyldopa	Quinalbarbitone
[Amphetamines]	*Dydrogesterone	*Methylsulphonal	Rifampicin
*Amylobarbitone	*Econazole NO_3	*Methyprylone	Simvastatin
Antipyrine	*Enalapril	Methysergide	Sodium aurothiomalate
*Auranofin	Enflurane	*Metoclopramide	Sodium oxybate
*Aurothiomalate	*Ergot compounds	Metyrapone	[Sodium valproate]
Azapropazone	Ergometrine maleate	Mianserin HCl	*Spironolactone
Baclofen	Ergotamine tartrate	Miconazole	Stanozolol
*Barbiturates	*Erythromycin	[Mifepristone]	Succinimides
*Bemegride	*Estramustine	Minoxidil	*Sulfacetamide
Bendrofluazide	Ethamsylate	*Nandrolone	*Sulfadiazine
Benoxaprofen	*Ethanol	*Nalidixic acid	*Sulfadimidine
Benzbromarone	Ethionamide	Natamycin	*Sulfadoxine
[Benzylthiouracil]	*Ethosuximide	*Nandrolone	*Sulfamethoxazole
[Bepridil]	*Ethotoin	[Nicergoline]	*Sulfasalazine
Bromocriptine	Etidocaine	*Nifedipine	*Sulfonylureas
Busulphan	Etomidate	*Nikethamide	Sulfinpyrazone
*Butylscopolamine	Fenfluramine	Nitrazepam	Sulpiride
Captopril	*Flucloxacillin	*Nitrofurantoin	Sulthiame
*Carbamazepine	*Flufenamic acid	Nordazepam	Sultopride
*Carbromal	Flunitrazepam	Norethynodrel	*Tamoxifen
*Carisoprodol	Flupenthixol	*Norethisterone	*Terfenadine
[Cefuroxime]	Flurazepam	[Nortriptyline]	Tetrazepam
[Cephalexin]	*Frusemide	Novobiocin	*Theophylline
[Cephalosporins]	*Glibenclamide	*Oral contraceptives	*Thiopentone Na
[Cephradine]	*Glutethimide	*Orphenadrine	Thioridazine
[Chlorambucil]	*Glipizide	Oxanamide	Tilidate
*Chloramphenicol	Gramicidin	[Oxazepam]	Tinidazole
*Chlordiazepoxide	*Griseofulvin	Oxybutynin HCl	*Tolazamide
*Chlormezanone	[Haloperidol]	Oxycodone	*Tolbutamide
Chloroform	*Halothane	*Oxymetazoline	Tranylcypromine
*Chlorpropamide	*Hydantoins	*Oxyphenbutazone	Trazodone HCl
Cinnarizine	*Hydralazine	Oxytetracycline	Trimethoprim
Clemastine	*Hydrochlorothiazide	Paramethadione	[Trimipramine]
[Clobazam]	*Hydroxyzine	Pargyline	Troxidone
[Clomipramine HCl]	Hyoscine	*Pentazocine	Valproate
[Clonazepam]	*Imipramine	Perhexiline	Valpromide
Clonidine HCl	Iproniazid	Phenacetin	Veralipride
*Clorazepate	Isometheptene mucate	Phenelzine	*Verapamil
Cocaine	[Isoniazid]	*Phenobarbitone	*Vibramycin
[Colistin]	Kebuzone	Phenoxybenzamine	Viloxazine HCl
Co-trimoxazole	Ketoconazole	*Phensuximide	[Vinblastine]
	*Levonorgestrel	*Phenylbutazone	[Vincristine]
	Lignocaine	Phenylhydrazine	Zuclopenthixol
	*Lisinopril	*Phenytoin	
	Loprazolam	Pipebuzone	
	Loxapine	Pipemidic	
	*Lynestrenol	Acid	
	Lysuride	Piritramide	
		*Piroxicam	

Table 2

Abdominal Abscess
Abdominal Angina
Abdominal Hernias
Acute Mesenteric Ischemia
Adrenal Carcinoma
Adrenal Crisis
Amebic Hepatic Abscesses
Aortic Dissection
Appendicitis

Chronic Pelvic Pain
Colonic Obstruction
Constipation
Diverticulitis
Emphysema
Emphysematous Cholecystitis
Emphysematous Pyelonephritis
Empyema, Gallbladder
Endometriosis
Esophagitis
Factitious Disorder
Fibromyalgia
Gastric Outlet Obstruction
Gastritis, Acute
Hypertension
Ileus
Intestinal Motility Disorders
Intestinal Pseudo-obstruction: Surgical Perspective
Irritable Bowel Syndrome
Lead Nephropathy
Mediterranean Fever, Familial
Nephrolithiasis
Nerve Entrapment Syndromes
Ovarian Cysts
Pancreatitis, Acute
Pancreatitis, Chronic
Pelvic Inflammatory Disease
Porphyria, Chester
Porphyria, Hereditary Coproporphyria
Portal Vein Obstruction
Pyelonephritis, Acute
Pyelonephritis, Chronic
Toxicity, Lead
Diverticulosis and Manic Depressive Illness

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