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Toxicities of Herbal Abortifacients

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Abstract

Background: In the post-*Roe* era, barriers to facility-based abortions may lead to an increased incidence of self-managed abortions. While misoprostol-based medication abortions have significant literature supporting its safety profile, there is a knowledge deficit within the medical community regarding the toxicities of commonly used herbal abortifacients.

Methods: This is a narrative review, based on a MEDLINE and HOLLIS database search, of self-managed abortion methods with herbal abortifacients and their associated toxicities.

Results: Common herbal abortifacients with significant morbidity and mortality implications include pennyroyal, blue cohosh, rue, and quinine. Other commonly reported abortifacients considered to be less toxic also are discussed in brief. Special considerations for hepatic, cardiac, renal, and hematologic toxicities are important in patients with significant exposures to these herbal substances.

Conclusion: There is an anticipated increase in the utility of herbal xenobiotics for self-managed abortions with post-*Roe* restrictions to standard mifepristone-misoprostol protocols. Frontline providers should be aware of the associated toxicities and have special considerations when treating a poisoned patient in this population.

Keywords

herbal abortifacient; abortifacient toxicity; self-managed abortion; emergency care

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1. INTRODUCTION

In an era of significant restrictions on abortion care, self-managed abortion, or ending one's own pregnancy without clinical oversight, may play an increasingly important role in the exercise of reproductive autonomy. A scoping review on self-managed abortion demonstrated that some individuals pursue this care for positive reasons, such as comfort, confidentiality and control [1]. While the decision to pursue self-managed abortion is not always one of necessity, many do so because of insurmountable logistical barriers (e.g., lost wages and cost of the procedure, distance to travel, need for child care), fear of mistreatment and stigma, and risk of criminalization or being reported to police [1]. These latter reasons may prompt an increase in self-managed abortions following the fall of *Roe vs Wade*, now with 14 states severely restricting or eliminating abortion [2,3].

Medication abortion is a two-step process, involving the use of mifepristone, an antiprogesterone, and 24–48 hours later, misoprostol, a prostaglandin. These two medications comprise the FDA-approved regimen for both medication abortion and medication management of early pregnancy loss, now standard of care in clinical and hospital settings. Substantive and growing evidence, in international and U.S.-based settings, supports that limited clinical involvement or completely self-managed medication abortion with misoprostol, with or without mifepristone, is effective and safe [4–6]. However, self-managed abortion by other means is concerning to the clinician, particularly to the emergency and urgent care clinician, for two primary reasons: 1) while some methods have a long history in traditional practices and potentially do have some abortifacient properties, the efficacy is unlikely to match that of the FDA-approved regimen for medication abortion, delaying access to time-sensitive care and 2) alternative methods may pose risk of complications unfamiliar to allopathic practitioners, because of limited research and limited clinical exposure in the half century of federal abortion protection [7].

It is not anticipated that the abortion experience in the U.S. will replicate the morbidity and mortality of the pre-*Roe* era given advancements in medication abortion, telehealth and internet-based services, as well as a sophisticated abortion fund network [8–10]. However, familiarity with alternative self-managed abortion practices, particularly herbal remedies which represent the largest non-misoprostol-based class of self-management agents, is a critical part of being an informed clinician in a new era of reproductive care [11–13].

Herbal abortifacients are available in a variety of formulations, including pills, teas, tinctures, and oils. Ingestion of these xenobiotics is the most common method, although topical applications and intravaginal suppositories have also been reported. While the American College of Medical Toxicology and the American Academy of Clinical Toxicology state that the use of herbal supplements as treatment modalities is not recommended, the vast majority of these products likely do not pose significant toxicologic risk and are considered safe for its consumer [14]. However, we highlight a selection of herbal abortifacients that have the potential to cause harm and illustrate key characteristics of each one, summarized also in Table 1. Herbal agents that will be discussed include pennyroyal, blue cohosh, rue, and quinine. We will also mention in brief other abortifacient with a lower toxicity profile. The goal is to broaden the clinician's differential and offer

treatment considerations when assessing an ill patient who is presenting following induced abortion or early pregnancy loss.

2. METHODS

Several survey studies document the use of herbal remedies used for self-managed abortions. However, although common herbal xenobiotics are often reported, comparative prevalence of each herb has not been completely described in the literature. This review employed a narrative search strategy from the MEDLINE and the Harvard Online Information System (HOLLIS) database to characterize some of the recurrent herbal abortifacients. Of the reported herbal abortifacients, we chose to elaborate on those that have documented adverse clinical outcomes. Other herbal abortifacients without any or with only minimally reported toxicities are mentioned in brief as well. Due to a dearth of high-quality evidence within the field of toxicology, best practices frequently require justification based on theoretical groundwork and our working subspecialty knowledge of toxicologic mechanisms. Further, the assessment of the toxicities of self-sourced herbal xenobiotics can be confounded by impurities or mixed composition within these supplements, given that they are products of an unregulated industry. Lastly, the herbal abortifacients discussed in this review is not an exhaustive list of all possible herbal abortive agents and only contains the substances that clinicians will most likely encounter in the post-*Roe* era.

3. HERBAL ABORTIFACIENTS

The following is a selection of herbal agents that have been used as abortifacients. The mechanisms for their respective abortifacient properties have not been clearly elucidated although they likely mimic pathways similar to routinely used abortifacients like progesterone blockade leading to a disruption of the gravid endometrium or prostaglandin receptor agonism leading to uterine myometrium contraction. Here we highlight pennyroyal, blue cohosh, rue, and quinine as theses herbal xenobiotics have demonstrated significant toxicity to its user. We also briefly discuss other common herbal abortifacients without clearly reported significant toxicities.

3.1. Pennyroyal *(Mentha pulegium)*

3.1.1. Background—Pennyroyal oil is derived from the plant *Mentha pulegium* and has a mint-like scent, occasionally used for perfume fragrance. While historically used as an abortifacient, there has been a recent resurgence in interest as demonstrated by the increase in Google searches both after the leak of Justice Alito's majority draft opinion on May 2nd, 2022 and following the *Dobbs* decision on June 24th, 2022 [15]. Social media trends since the sentinel judicial changes on platforms such as TikTok and Instagram have also featured pennyroyal as an abortifacient [16,17].

3.1.2. Toxicity—Pennyroyal oil is a mixture of several monoterpene constituents, the largest component being pulegone which is hepatotoxic and causes hepatic centrilobular necrosis [18]. Pulegone metabolites also have similar effects, including the more hepatotoxic product menthofuran [19]. Like acetaminophen toxicity, the initial cytotoxic effect to the liver is temporized by endogenous stores of glutathione. Once depleted, a delayed

hepatotoxic clinical picture develops. Numerous case reports depict patients that ingest pennyroyal oil with progression to fulminant liver failure and death [20,21]. As little as 10 mL of pennyroyal oil ingestion was associated with moderate to severe hepatic toxicity in one case series [22].

3.1.3. Management—Given the parallel pathophysiology of pennyroyal hepatotoxicity to acetaminophen hepatotoxicity, there is theoretical support for the use of N-acetylcysteine. The first reported successful treatment of pennyroyal oil toxicity with N-acetylcysteine was in 2005, suggesting that early N-acetylcysteine administration, as in acetaminophen-induced hepatic injury, has an important therapeutic role in pennyroyal toxicity [23]. Therefore, given the low-risk profile of N-acetylcysteine, we recommend the use of this therapy, in conjunction with poison center guidance or consultation with a medical toxicologist, when facing potential hepatic failure that may require transplantation or lead to death. There are no reports of hemodialysis being beneficial for pennyroyal toxicity.

3.2. Blue Cohosh *(Caulophyllum thalictroides)*

3.2.1. Background—Blue cohosh naturally grows throughout North America and extracts from the roots have been sold as a tincture. It has been traditionally used as an emmenagogue and uterotonic. When ingested as a labor induction agent, blue cohosh has been shown to have transplacental toxicity through case reports of neonatal congestive heart failure and stroke following maternal exposure [24,25]. Additionally, blue cohosh derivatives have been reportedly utilized as an abortifacient.

3.2.2. Toxicity—Blue cohosh contains the plant alkaloid N-methylcytisine, which is a nicotinic acetylcholine receptor agonist, with structural similarity to nicotine found in the tobacco plant (*Nicotiana tobacum)*. When used in high doses as an abortifacient, a toxidrome analogous to nicotine poisoning can be anticipated [26]. Symptoms and exam findings include, but are not limited to, nausea and emesis, headache, dizziness, muscle fasciculations, seizure, and respiratory failure. Vital signs typically reveal tachycardia, tachypnea, and hypertension. However, the patient may also display muscarinic symptoms such as salivation, bronchorrhea, and bradycardia. In addition to N-methylcystisine, blue cohosh also contains plant glycosides such as caulosaponin and caulophyllosaponin, which have vasoconstrictive properties and can lead to hypertension and vaso-occlusive syndromes. With severe toxicity and without appropriate management, death can ensue.

3.2.3. Management—There are, unfortunately, no reversal antidotal agents for the nicotinic symptoms that may occur. Standard cardiovascular and respiratory support is the mainstay of management, with fluids and vasopressors in the event of hypotension and antihypertensives for hypertensive crises, although the latter tend to be transient and self-resolving. In the event of a seizure, benzodiazepines remain the first-line anti-epileptic. Atropine can be considered in the patient with muscarinic symptoms, titrated to the clearing of secretions and improvement of respiratory effort. If necessary, intubation and mechanical ventilation may be considered [27]. It is unlikely that extracorporeal removal of N-methylcytisine has a role in therapy as its high volume of distribution render hemodialysis

ineffective [28]. Specialty guidance for management of this toxidrome should be sought, especially if considering the use of antidotes.

3.3. Rue *(Ruta graveolens)*

3.3.1. Background—Rue is typically used in the form of a tea and has been mentioned as an abortifacient in case reports following self-managed abortion [1]. This use of rue has been well documented both internationally and domestically, specifically within the traditional New Mexican communities in the Southwestern United States [29]. Traditional medicine texts have cautioned against the toxic effects of rue teas when used in higher doses as an abortifacient [29].

3.3.2. Toxicity—A characterization of herbal abortifacient toxicities from a South American poison center demonstrated a higher incidence of hepatic, renal, and hematologic derangements among individuals who have taken rue as either a single agent or mixed with other herbals xenobiotics for self-managed abortions [30]. Toxicity with large ingestions have been severe, with a documented case of multiorgan failure causing significant electrolyte derangements as well as hemodynamic instability requiring aggressive resuscitative measures and hemodialysis [31]. End organ injury is suspected to be caused by coumarin subtypes, such as chalepesin, found in the plant. The specific mechanism of this toxicity, however, has not been elucidated. The rue plant also contains psoralen, a furanocoumarin that intercalate DNA of dermal cells leading to the formation of photoadducts when exposed to ultraviolet spectrum light. Clinically, this results in photodermatitis of light-exposed skin [32].

3.3.3. Management—In the setting of liver failure, as in other causes of acute liver failure, it is reasonable to attempt treatment with N-acetylcysteine given that the risk-benefit assessment in a critically ill patient tends to favor the administration of this therapy. The poison center hotline or a medical toxicologist should be contacted to help with this decision-making. Complications from end organ damage will necessitate close monitoring of electrolytes to prevent cardiac arrhythmias such as in the setting of hyperkalemia. Renal replacement therapy may be necessary if renal failure develops although there is no evidence supporting the utility of dialysis for significant rue ingestion in the absence of renal dysfunction. Usually, dermatitis can be managed with topical steroids, but severe manifestations of photodermatitis may need to be managed in consultation with a burn specialist [33].

3.4. Quinine *(Cinchona officinalis)*

3.4.1. Background—Quinine, derived from the bark of the cinchona tree, has historically been used as treatment for malaria by indigenous South American groups and later adopted by Europeans. Additionally the use of quinine derivatives for termination of pregnancy has historical precedence and persists in present day as well [34–36]. This product is available online and can be purchased without a prescription.

3.4.2. Toxicity—In cases of mild to moderate toxicity, patients may present with the classically described trio of *cinchonism*: headache, gastrointestinal upset, and tinnitus.

Refractory hypoglycemia has also been reported secondary increased endogenous insulin release from pancreatic beta-islet cells, similar to the mechanism of sulfonylurea medications [37.38]. However, the primary organ system of concern in severe toxicity is cardiac as quinine's sodium channel blockade properties can lead to a prolonged phase I of the cardiomyocyte depolarization cycle with a corresponding widened QRS complex seen on electrocardiogram [39]. As in other cardiotoxic drugs with sodium channel inhibition, fatal wide-complex tachyarrhythmias can ensue. Although less than its derivative quinidine, QT widening with quinine has also been observed via its effects on cardiomyocyte inward-rectifier potassium channels, predisposing patients to polymorphic ventricular tachycardia [39,40]. Additionally, there have been reports of microangiopathic hemolytic anemia suspected to be secondary to quinine-induced thrombotic thrombocytopenic purpura-hemolytic uremic syndrome [41,42].

3.4.3. Management—Patients with suspected quinine ingestion should universally have serial electrocardiograms performed. Identification of a QRS greater than 100 milliseconds warrants consideration for treatment with sodium bicarbonate, as adopted from tricyclic antidepressant overdose management standards [43]. If the QT interval is greater than 500 milliseconds, magnesium can be given for cardiac membrane stabilization to prevent ventricular tachyarrhythmias. With clinical findings of anemia and jaundice, it is worth investigating for acute hemolytic anemia. In the setting of systemic hemolysis and thrombosis leading to renal failure, dialysis should be considered. Additionally close monitoring for hypoglycemia and intervening with IV dextrose, food, glucagon, and octreotide should be considered. These interventions, with guidance from a medical toxicologist or the poison center, can be lifesaving.

3.5. Other Herbal Abortifacients

There are many other commonly reported abortifacients that do not carry significant toxicity, if at all. For example sesame, ginger, pineapple, papaya, turmeric, and chamomile, amongst many others, have been used and generally considered to be safe [13,44]. Other common herbs with comparatively benign toxic profiles include mugwort (*Artemesia sp)*, parsley (*Petroselinum crispum)*, dong quai (*Angelica polymorpha*), and black cohosh (*Cimicifuga 11racemosa*). These are often used concomitantly with other herbal abortifacients.

Mugwort has historically had been used for the treatment of malaria, and has been mentioned in popular culture as an abortifacient. It can have mild hallucinogenic properties in the form of vivid dreams as well as pruritus secondary to its psoralen component. Parsley can also cause photodermatitis but has not been implicated as a sole cause of life-threatening toxicity. Dong quai, a root used in traditional Chinese medicine, contains several coumarin xenobiotics. It is recommended that patients taking warfarin and other anticoagulants avoid using dong quai [45,46]. The theoretical adverse event is a bleeding diathesis, although reports of such complications are absent from the literature.

Black cohosh (*Cimicifuga racemosa*) is a flowering plant found in North America with its roots used to make extractions for the management of post-menopausal symptoms. There had been speculation for hepatotoxicity with ingestion of black cohosh. However,

a systematic review in 2008 that included case studies of hepatotoxic patients who have ingested black cohosh was ultimately unable to demonstrate a direct causative relationship given confounding elements such as coingestions of other herbal xenobiotics [47]. Nonetheless, the available evidence is limited, and the clinician at bedside should consider an evaluation of liver enzymes and liver synthetic function when confronted with a sick patient that has taken black cohosh [48]. Overall, there is a dearth of evidence showing toxicities from these abortifacients, although this may change in the coming years.

3.6. Other Toxicological Considerations for Abortifacients

In addition to herbal abortifacients, other substances for self-managed abortion have been reported, including the use of known toxic non-herbal substances, over-the-counter medications, and prescription medications [13]. Many of these, such as toxic alcohols, acetaminophen, aspirin, caffeine, and antidepressants, have well documented toxic profiles. Descriptions of these are beyond the scope of this article. In these cases, evaluation and management of such poisonings follows the steps outlined below, and expeditious communication with the poison center is key.

4. EMERGENCY DEPARTMENT APPROACH

The general approach to the poisoned patient is similar to the standard approach to the sick, undifferentiated patient in the acute care setting. Special considerations for poisonings such as antidotes and prompt communication with the poison center hotline or medical toxicologist should not be performed in lieu of initial airway, breathing, and circulation stabilization. As in any altered patient, expeditious assessment of the blood glucose level is critical. Any delay in the work up for non-toxicologic causes of clinical symptoms and signs should be avoided. Here we outline a few additional considerations for the poisoned patient: 1) Early electrocardiogram to assess for cardiac toxicity and the potential need for sodium bicarbonate or magnesium therapy and electrolyte management. 2) In addition to standard lab work to assess electrolytes, acid-base status, cell count, and liver/renal function, an expanded laboratory evaluation for coingestants such as serum acetaminophen and aspirin concentrations should be obtained. 3) Consider antidotal interventions such as N-acetylcysteine or atropine, as well as gastric decontamination measures, in conjunction with a poison specialist or medical toxicologist. The national poison center hotline, accessible by calling 1-800-222-1222, is a valued source of medical guidance for clinicians in all practice settings and should be used liberally. Consultations are cost-free and staffed 24/7 by certified specialists in poison information and board-certified medical toxicologists. The outlined steps above often take place in a simultaneous fashion especially when caring for an unstable, critically ill patient.

5. CONCLUSION

Self-managed abortion methods with most herbal remedies are unlikely to cause major toxicity events. Some, such as pennyroyal, blue cohosh, rue, and quinine, however, have notable toxicities that should, now more than ever, be part of the emergency clinician’s fund of knowledge. In the rare occasion of complications related to these herbal remedies, the

presentation and management as detailed above must be framed with adept medical care as these clinical presentations may become more prevalent in the near future.

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Table 1.

Characteristics of and treatment considerations for common reported herbal abortifacients

Herbal Abortifacient	Primary Toxicity	Toxin and Mechanism	Clinical Characteristics	Treatment Considerations
Pennyroyal (*Mentha pulegium*)	Hepatic	Pulegone: hepatotoxic and glutathione depletion	GI upset, hepatomegaly, encephalopathy, seizures, renal failure, DIC	N-acetylcysteine Transplant evaluation for liver failure
Blue cohosh (*Caulophyllum thalictroides*)	Systemic	N-methylcytisine: nicotine receptor agonist Saponin: vasoconstriction	GI upset, headache, dizziness, diaphoresis, fasciculations, seizures, tachycardia, hypertension, hyperthermia, respiratory failure	Atropine for muscarinic symptoms Benzodiazepines for seizures Antihypertensives for hypertensive crises
Rue (*Ruta graveolens*)	Systemic	Chalepesin: hepatotoxic and nephrotoxic Psoralen: photodermatitis	GI upset, multiorgan failure, DIC, photodermatitis	FFP or PCC for DIC Hemodialysis for renal failure Standard treatment for hyperkalemia
Quinine (*Cinchona officinalis*)	Cardiac	Quinine: cardiac sodium and potassium channel blockade, CNS and renal toxicity	Cinchonism trio: GI upset, headaches, and tinnitus. Ventricular arrhythmias, hypoglycemia, renal failure, respiratory failure, jaundice, DIC	Sodium bicarbonate for QRS prolongation Magnesium for QT prolongation Vasopressors for hemodynamic support Dextrose, glucagon, and/or octreotide for hypoglycemia

Definitions: GI= gastrointestinal, FFP= fresh frozen plasma, PCC= prothrombin complex concentrate, DIC=disseminated intravascular coagulopathy, CNS= central nervous system