

Potassium iodide in dermatology: A 19th century drug for the 21st century—Uses, pharmacology, adverse effects, and contraindications

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Potassium iodide (KI) is a useful drug in the dermatologic armamentarium. It is successfully used for inflammatory dermatoses, most notably erythema nodosum, subacute nodular migratory panniculitis, nodular vasculitis, erythema multiforme, and Sweet's syndrome. KI is also successfully used for cutaneous and lymphocutaneous sporotrichosis. The precise mechanism by which KI acts is unknown. Although many minor side effects are common with this drug, major side effects can occur in pregnant patients and those with a history of kidney or thyroid disease. This article reviews the pharmacology, mechanism of action, indications, contraindications, and adverse effects of KI as a therapeutic agent. (*J Am Acad Dermatol* 2000; 43:691-7.)

Iodine was first discovered in seaweed in the early 1800s and was shortly thereafter used to treat thyroid disease. In subsequent years, clinicians tried iodine to treat almost every disease that failed routine treatment, including syphilis, lupus vulgaris, eczema, and psoriasis.¹

Today, potassium iodide (KI) is most frequently used for inflammatory dermatoses including erythema nodosum, subacute nodular migratory panniculitis, nodular vasculitis, erythema multiforme, and Sweet's syndrome. Clinicians also use KI for cutaneous and lymphocutaneous sporotrichosis and, in the tropics, for entomophthoromycosis (previously called phycosporidiosis), a chronic, slowly progressive subcutaneous disease caused by the fungi *Basidiobolus* and *Conidiobolus*.

This article reviews the pharmacology, mechanism of action, indications, contraindications, and adverse effects of KI as a therapeutic agent. This endeavor will allow the clinician to have a framework for the appropriate use of this versatile drug.

PHARMACOLOGY

KI is a compound made of 76% of the halogen iodine and 23% of the alkali metal potassium by weight.² KI is prepared by reacting iodine with a hot

Abbreviations used:

KI: potassium iodide
SSKI: saturated solution of potassium iodide
TSH: thyroid-stimulating hormone
WCE: Wolff-Chaikoff effect

solution of potassium hydroxide, the product being subsequently reduced to iodide by heating the dry reaction mixture with carbon.³

For convenience, KI is usually administered in the form of a saturated solution (SSKI) at a dose of 47 mg/drop. SSKI is made by adding KI to hot purified water, using sodium thiosulfate as a preservative. To protect against gastrointestinal irritation, this solution can be added to water, fruit juice, or milk before drinking.

The KI dose used to treat dermatoses is much higher than those used to treat thyrotoxicosis (250 mg 3 times daily) or to protect against radiation (a 100-150 mg single dose).⁴ Clinicians typically begin treatment of inflammatory dermatoses with an oral dose of 300 mg (approximately 6 drops of SSKI) 3 times daily, followed by weekly increases as tolerated.¹ The dose used for mycoses is often higher, beginning at 600 mg (approximately 12 drops of SSKI) orally 3 times daily and often increased to 6 g (approximately 127 drops of SSKI) daily if tolerated.⁵

After ingestion, KI is readily absorbed in the intestinal tract and distributes rapidly through the extracellular space. Iodide concentrates in the thyroid gland, salivary glands, gastric mucosa, choroid plexus, mammary glands, and the placenta. Ninety

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Table I. Reported uses for potassium iodide

Infectious
Cutaneous cryptococcosis ³¹
Entomophthoromycosis (caused by <i>Basidiobolus</i> and <i>Conidiobolus</i> fungi) ^{27,28}
Human pythiosis (caused by <i>Pythium insidiosum</i> fungus) ²⁹
Lymphocutaneous <i>Nocardia brasiliensis</i> ³⁰
Sporotrichosis (fixed cutaneous and lymphocutaneous) ^{5,23-26}
Neutrophilic dermatoses
Pyoderma gangrenosum ¹⁸
Sweet's syndrome ^{1,15,17}
Panniculitis
Erythema nodosum ^{1,14,15}
Nodular vasculitis ^{1,14,15}
Subacute nodular migratory panniculitis ¹⁶
Miscellaneous
Behçet's syndrome ^{1,15}
Erythema multiforme ^{1,15}
Wegener's granulomatosis ¹⁹

percent of the orally administered dose is excreted in the urine. Sweat, breast milk, and feces account for the remainder of the excretion.⁶

Concurrent use of KI with other potassium-containing medications, potassium-sparing diuretics, and angiotensin-converting enzyme inhibitors (ACE inhibitors) may result in hyperkalemia and potassium toxicity.⁷

Likewise, the use of KI with iodide-containing drugs (eg, amiodarone) and drugs that inhibit thyroid function (eg, lithium, phenazone, and, possibly, sulfonamides) may cause hypothyroidism.^{7,8}

MECHANISM

The precise mechanism by which KI exerts its therapeutic effect against inflammatory dermatoses is unknown. Many of the disorders for which KI is indicated display neutrophils in early stages of the disease such that neutrophil toxicity or neutrophil chemotaxis could be affected. In 1982, Miyachi and Niwa⁹ found that KI, as well as dapsone, significantly suppresses the neutrophil's ability to generate the toxic oxygen intermediates hydrogen peroxide and hydroxyl radical in vitro. In 1990, Honma et al¹⁰ studied the effects of KI on neutrophil chemotaxis. Chemotaxis is a complex process that depends on the detection of a concentration gradient of a chemoattractant and the movement of the cell toward the attractant. Honma et al¹⁰ administered 15 mg/kg daily of KI for 3 days to 15 healthy patients. The patients' blood was then drawn, and neutrophil chemotaxis studied by means of the modified Boyden chamber, a process whereby neutrophil migration rate is measured after activation with

Escherichia coli endotoxins. In this study, KI significantly inhibited neutrophil chemotaxis in peripheral blood. However, the study could not explain what part of chemotaxis was altered by KI. Although the above mechanisms may help to explain KI's effect on neutrophilic dermatoses, KI's mode of action is still an enigma for the panniculitides in which neutrophils may not be of paramount importance.

The precise mechanism by which KI kills fungi is also unknown. It is unclear whether KI works against fungi by a fungicidal mechanism or by enhancing the body's immunologic and nonimmunologic defense mechanisms. *Sporothrix schenckii* and *Basidiobolus* actually grow when plated in KI.¹¹ Cell degeneration, however, has been shown by electron microscopy to occur in *Sporothrix schenckii* yeast dipped in varying concentrations of iodine.¹² KI does not appear to increase monocyte or neutrophil killing of *Sporothrix schenckii*.¹³

INDICATIONS

Table I summarizes indications for the use of KI. In 1976, Schulz and Whiting¹⁴ repopularized the use of KI after reporting that 24 of 28 patients with erythema nodosum, and 16 of 17 patients with nodular vasculitis (erythema induratum), responded to KI. Relief of symptoms occurred within 2 days, with most lesions clearing within 2 weeks. The doses of KI used by Schulz and Whiting ranged from 360 to 900 mg daily.

Horio et al¹⁵ confirmed the findings of Schulz and Whiting and also found KI effective for Behçet's syndrome, erythema multiforme, and Sweet's syndrome. Using a dose of 300 mg 3 times daily, they observed relief of subjective symptoms such as lesional tenderness, fever, and arthralgias within 24 hours. Complete remission of lesions 10 to 14 days after administration of KI was noted in 11 of 15 patients with erythema nodosum, 7 of 10 patients with nodular vasculitis, 1 of 4 patients with leg lesions of Behçet's syndrome, 14 of 16 cases of erythema multiforme, including those with concomitant herpes simplex, and 7 of 8 cases of Sweet's syndrome. The best response was noted in those patients with erythema nodosum associated with systemic symptoms and a positive C reactive protein, and in those patients to whom the medication was administered shortly after the onset of illness.^{1,15} A rapid response to KI has also been reported in a case of subacute nodular migratory panniculitis (erythema nodosum migrans)¹⁶ and in a case of neutrophil-poor Sweet's syndrome, in which KI was tried after oral prednisolone, dapsone, and minocycline failed.¹⁷

Successful use of KI for other inflammatory dermatoses has also been reported. In 1993, Richardson

and Callen¹⁸ reported using KI to successfully treat recalcitrant pyoderma gangrenosum in the inguinal crease. They found marked improvement after 2 months and complete clearing after 4 months using a dose of 300 mg 3 times daily.

In 1994, Torinuki¹⁹ reported successfully treating Wegener's granulomatosis using a combination of KI and prednisolone. The patient had gangrenous rhinitis and diffuse skin involvement with necrotic papules and ulcerations. Prednisolone alone only slightly improved the lesions, but when 800 mg of KI daily was added, the lesions rapidly improved and almost completely disappeared in 3 months without recurrence at a 1-year follow-up.

Clinicians have also reported treating granuloma annulare with KI. In 1979, Giessel, Graves, and Kalivas²⁰ reported improvement of disseminated granuloma annulare in 3 of 4 patients taking 900 mg of KI daily for 12 weeks. In 1984, Caserio, Eaglstein, and Allen²¹ reported similar success in one patient. However, in a double-blind, placebo-controlled, cross-over study in 1994, Smith, Hansen, and Zone²² found no difference between placebo and KI in the 7 patients with disseminated granuloma annulare who completed their study. They attributed an earlier report of success to the waxing and waning nature of granuloma annulare.

Oral iodide was first used at the beginning of the 20th century for sporotrichosis and continues to be used in most of the world for fixed cutaneous and lymphocutaneous disease because of its effectiveness and low cost.²³ Where available, however, itraconazole has supplanted KI use.²⁴ Adults initially start KI therapy for sporotrichosis at 500 to 1500 mg daily divided into 3 doses and gradually increase to 4000 to 6000 mg daily and then continue for approximately 6 to 10 weeks. Children typically take one half to one third this dose.⁵ These large drug amounts, spread out over 3 daily doses, frequently result in patient noncompliance because of frequent gastrointestinal irritation.^{5,25}

Because of high patient noncompliance, Cabezas et al²⁵ in 1996 performed a randomized, nonblinded study on 57 patients with culture-confirmed cutaneous or lymphocutaneous sporotrichosis to compare the safety and efficacy of once-daily versus 3-times-daily dosing in the pediatric population. Patients (mean age, 7 years) received KI in the form of SSKI that began at 150 mg/day and increased rapidly over approximately 11 days to a maximum of 160 mg/kg daily in both groups. Mild side effects, most frequently nausea, were common in both groups (61% in the once-daily and 42% in the 3-times-daily group). Three patients had to discontinue therapy because of side effects; one patient in

each group discontinued therapy because of a severe urticarial rash, and one patient in the once-daily group discontinued therapy after experiencing erythema nodosum leprosum. (The authors do not state specifically in their article whether this patient had a diagnosis of leprosy.) Of the 51 patients who entered the study, all were cured by the treatment in a similar time of approximately 32 days. No relapses occurred after a 45-day follow-up. Cabezas et al²⁵ concluded that daily dosing in the pediatric population appeared to be appropriate treatment for sporotrichosis because of the low compliance with 3-times-daily dosing.

Of interest, Rafal and Rasmussen²⁶ in 1991 reported using KI to treat an 84-day-old patient with fixed cutaneous sporotrichosis. They used a dose of 3 drops of SSKI 3 times daily (approximately 450 mg/day) for 3 months with complete response. They claimed that this was the youngest patient reported to have sporotrichosis.

In tropical areas, KI is used as a first-line agent to treat entomophthoromycosis, a type of zygomycosis caused by the fungi *Basidiobolus* and *Conidiobolus*. These fungi are found in soil, decaying vegetation, insects, and the gastrointestinal tracts of amphibians. Basidiobolomycosis occurs mostly in Nigeria and Uganda and presents as a single, painless, hard, circumscribed subcutaneous nodule. Conidiobolomycosis occurs in tropical Africa, India, Puerto Rico, Colombia, and Brazil and presents as a tumor in the nasal skin, nasal obstruction, or sinusitis. Conidiobolomycosis often requires surgical intervention along with KI treatment. KI is not effective for the other group of zygomycoses, the mucormycoses, which are characterized by vascular invasion and tissue necrosis and include the organisms *Mucor*, *Rhizopus*, *Absidia*, and *Rhizomucor*.^{5,27,28}

KI is also used to treat subcutaneous granulomatous ulcers in human pythiosis, a disease occurring in thalassemic patients in Thailand caused by the aquatic fungus *Pythium insidiosum*. Although KI is effective for the subcutaneous type of infection caused by *P insidiosum*, it is not effective against keratitis or systemic vasculitis, also caused by this fungus, which is well known for causing serious economic losses because of its infection of crops, horses, and cattle.²⁹

Further success with KI has been reported in 1975 with lymphocutaneous *Nocardia brasiliensis*³⁰ and in 1957 with cutaneous cryptococcosis.³¹

SIDE EFFECTS AND CONTRAINDICATIONS

Patients taking KI frequently suffer minor side effects and occasionally are afflicted by serious ones

Table II. Reported side effects of potassium iodide

Dermatologic
Bullous pemphigoid ⁴⁵
Dermatitis herpetiformis ^{46,47}
Iododerma ^{*48-53}
Endocrine/metabolic
Fetal and neonatal goiter ^{*7,61,62}
Hypothyroidism ^{*8,55}
Hyperthyroidism ^{*8}
Negative ion gap ⁴²
Metabolic acidosis ⁴³
Potassium toxicity ^{*7}
Gastrointestinal
Diarrhea ⁷
Nausea ⁷
Vomiting ⁷
Stomach pain ⁷
Miscellaneous
Cardiac irritability ^{*44}
Iodism (mouth soreness, headache) ⁷
Pulmonary edema, angioedema, myalgias, eosinophilia, lymphadenopathy, and urticaria ^{*32-36}
Periarteritis nodosa ^{*38}
Prolonged fever ^{40,41}
Pustular psoriasis ^{*39}
Vasculitis ^{*37}

*Potential severe reaction.

(Table II). Common acute side effects include diarrhea, nausea, vomiting, and stomach pain; these acute side effects can be ameliorated by avoiding rapid dosage increases.

With prolonged use, patients may experience symptoms of iodism or potassium toxicity. Symptoms of iodism including a burning mouth, increased watering of the mouth, a metallic taste, soreness of the teeth and gums, and severe headache. Signs and symptoms of potassium toxicity include confusion, arrhythmia, hand numbness, or general weakness. Patients with renal function impairment or those taking angiotensin-converting enzyme inhibitors or potassium-sparing diuretics are at increased risk for potassium toxicity.⁷

KI has also been reported to cause pulmonary edema, angioedema, myalgias, eosinophilia, lymphadenopathy, and urticaria.³²⁻³⁶ KI has also been reported to trigger vasculitis,³⁷ periarteritis nodosa,³⁸ pustular psoriasis,³⁹ prolonged fever,^{40,41} a negative anion gap,⁴² metabolic acidosis,⁴³ cardiac irritability,⁴⁴ and bullous pemphigoid.⁴⁵

KI is well known to aggravate dermatitis herpetiformis. Before the widespread use of direct immunofluorescence, KI was used topically by clinicians to induce dermatitis herpetiformis lesions for diagnostic purposes.^{46,47}

The use of iodides can also cause acneiform eruptions, typically consisting of papulopustules or pustules on skin rich in sebaceous glands.⁴⁸ More severe eruptions can occur, however, and are classified as iododerma. These eruptions may appear erythematous, vesicular, bullous, urticarial, petechial, nodular, or vegetating, and occur on the face, shoulders, trunk, and extremities.⁴⁹

Histopathologic changes in acute iododerma reveal a diffuse dermal inflammatory infiltrate composed mostly of neutrophils with rare eosinophils, mast cells, and plasma cells.^{48,50} Chronic lesions demonstrate pseudoepitheliomatous hyperplasia with a mixed inflammatory infiltrate.⁴⁹

Iododerma has been reported in increased frequency in patients with underlying disease including multiple myeloma, polyarteritis nodosa, rheumatoid arthritis, lymphoma, and subacute glomerulonephritis, leading some authors to suggest that systemic disease may predispose patients to iododerma.⁴⁹ The exact mechanism of iododerma, however, is unknown, but may be the result of a hypersensitivity or hyperinflammatory reaction to iodide.^{51,52}

During the widespread use of KI in the 1920s and 1930s, many patients died because of the side effects associated with KI, most notably pulmonary edema and associated heart failure.⁵³ However, most of the above-mentioned side effects regress rapidly with discontinuation of KI. If necessary, corticosteroids can be used to control these side effects.

EFFECT OF KI ON THYROID METABOLISM

Also of concern, and not uncommon, is the myriad of effects that KI can have on the thyroid gland. The thyroid gland normally uses iodine to synthesize thyroid hormones L-thyroxine (T₄) and L-triiodothyronine (T₃). The biosynthetic pathway and control mechanisms of thyroid hormone synthesis have been reviewed by Heymann.^{54,55}

Control mechanisms of the thyroid gland must be in place to maintain euthyroid hormone levels. The thyroid gland possesses two such control mechanisms.

Firstly, the thyroid gland participates in a classic negative feedback mechanism via the pituitary secretion of thyroid-stimulating hormone (TSH) in response to thyrotropin-releasing hormone from the hypothalamus. The second regulatory mechanism resides in the thyroid gland itself and is called autoregulation.

Autoregulation serves to maintain a storage pool of organic iodine within the thyroid gland and serves as an initial defense against the fluctuations of the iodine supply. When there is a deficiency of iodine,

autoregulation maintains thyroid hormone synthesis. When iodide deficiency is prolonged and autoregulation is no longer able to continue to defend a normal rate of thyroid hormone synthesis, then activation of the hypothalamic-pituitary axis will ensue. Conversely, when excess quantities of iodine exist, the thyroid will stop producing thyroid hormone; this is called the Wolff-Chaikoff effect (WCE). Autoregulation ensures that the thyroid gland will produce enough hormone for the patient to remain euthyroid even in times of excess iodine. Hence, autoregulation allows escape from the WCE.⁸ It has now been determined (in rat thyroid tissue) that this autoregulation and escape from the WCE are made possible because of changes in the transcription of the mRNA that codes for a sodium/iodide symporter that allows iodide to enter the thyroid.⁵⁶

In some patients, the thyroid gland is dysfunctional and autoregulation does not occur in the face of excess iodine. In these patients, two different pathologic consequences of excess iodine can occur depending on whether autoregulation is either defective or completely absent.

If autoregulation is just defective, escape from the WCE cannot occur, TSH increases, and hypothyroidism and goiter ensue.⁸ Failure to escape with resultant hypothyroidism has been observed in patients treated with KI with Hashimoto's thyroiditis, euthyroid patients previously treated by thyroid surgery or radioactive iodide for Graves' disease, patients taking certain drugs (ie, sulfonamide derivatives, lithium, phenazone), patients with cystic fibrosis,⁸ patients previously treated with interferon- α for chronic viral hepatitis,⁵⁷ and patients with a history of amiodarone-induced thyrotoxicosis,⁵⁸ subacute thyroiditis,⁵⁹ or Graves' disease.⁶⁰ Defective autoregulation can occur years after the initial thyroid insult.

Failure to escape can also occur in the fetal and neonatal thyroid, resulting in possible goiter, hypothyroidism, and fetal death. This process can occur when a mother takes excess quantities of iodine. KI concentrates in, and crosses, the placenta. KI is also distributed in breast milk. KI is currently a category D drug, meaning that it should not be used in pregnant or breast-feeding women unless the benefits of taking KI outweigh the risks.^{7,61,62}

Jod-Basedow disease occurs when there is excess iodine and autoregulation is completely absent instead of just defective. Absent autoregulation occurs in some patients whose thyroids have autonomous foci, which occur in long-standing multinodular goiters, typically only seen in areas of iodine deficiency. In Jod-Basedow disease, the thyroid synthesizes excess thyroid hormone during times of excess iodine, leading to thyrotoxicosis.^{7,8}

For dermatologists who administer KI, knowledge of the WCE and the possibility of hypothyroidism or hyperthyroidism is imperative. Before KI is prescribed, it would be prudent for the physician to inquire about any previous history of thyroid disease, autoimmune or otherwise. It is also essential to determine whether a patient is taking other medications that could affect thyroid function. Unless there is suspicion of underlying thyroid disease, baseline thyroid function studies (ie, TSH, T₄, antithyroglobulin, and antimicrosomal antibodies) are not indicated. Fortunately, for those dermatoses for which KI is currently indicated, therapeutic effect usually occurs within a few weeks. This is within the time frame in which the thyroid autoregulatory processes will ordinarily allow escape from the WCE. If therapy with KI continues for more than 1 month, however, a screening TSH is prudent to ensure that iodide-induced hypothyroidism does not ensue. If iodide-induced hypothyroidism is detected, discontinuing KI will usually result in normal T₄, T₃, and TSH within 1 month.⁶³

CONCLUSION

Despite its side effects, clinicians in many parts of the world are likely to continue to use KI as a first-line drug because of its effectiveness and low cost. For diseases in which superior agents are available, clinicians are likely to continue to use KI as a second-line drug when these agents fail, are contraindicated, or cause intolerable side effects.

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