Diagnosis and Treatment of
Common Skin Diseases

Diagnosis and Treatment of Common Skin Diseases

Fifth Edition

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Preface to the Fifth Edition

The success of the previous edition was encouraging enough to warrant enlarging of the current contents to meet the increasing requirements of the audience. Accordingly, the contents of Chapter 3 (Drugs that may Cause Specific Side Effects) are updated by incorporating the definitions of Specific Dermatoses, which may accrue as adverse effects following administration of drug(s) bearing generic and/or brand name in order to facilitate to counter them by appropriate measures, including immediate withdrawal of the incriminating drug(s). The volume of Chapter 5 (Drugs Formulary of Dermatological Preparations) has also been increased by the inclusion of a few recent and a new drug formulary. A new Chapter 6 (Biologics: Therapeutic Applications), an upcoming fascinating subject, has been added to enrich the existing drug choices for various dermatoses. Their indications, the generic and the brand names along with their doses and modes of administration are highlighted. And emphasis on their adverse effects has been vividly outlined as an alert.

Emergency Dermatology too has been given attention, and new chapters on Erythroderma and Angioedema have been included emphasizing their diagnostic criteria and management strategies.

Recommended Reading is an important inclusion to enrich the text by selected references designed according to the PubMed pattern and Research Gate.

Virendra N Sehgal

Preface to the First Edition

Understanding of drug therapy in the specialty of Dermatology, Venereology and Leprosy, in particular, is paramount for practitioners, who have been responsible for managing most of these patients. Common skin diseases are not difficult to diagnose. Their treatment, however, may be a handicap. An endeavor in preparation of the present *Dermatologic Drug Directory* has been made to bring together scattered information on their management under this caption, in order to facilitate the family physicians. Commonly used systemic and topical drugs, their availability, dosages, side/untoward effects and contraindications may provide a ready reference and meet the growing needs of the family physicians, medical students, resident doctors and also specialists absorbed in alleviating dermatosis, venereal diseases and leprosy.

Virendra N Sehgal

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CHAPTER 8

Emergency Dermatology: Angioedema

Angioedema/angio-oedema and **Quincke's edema** is the rapid swelling (edema) of the dermis, subcutaneous tissue (Swelling. Dorland's Illustrated Medical Dictionary (31st edn.). Saunders. 2007), mucosa and submucosal tissues. It is very similar to urticaria, but urticaria, commonly known as hives, occurs in the upper dermis, may affect any part of the body where skin and/or mucous membrane is/are lose including the upper respiratory tract. The term angioneurotic edema was used for this condition in the belief that there was nervous system involvement, but this is no longer thought to be the case.

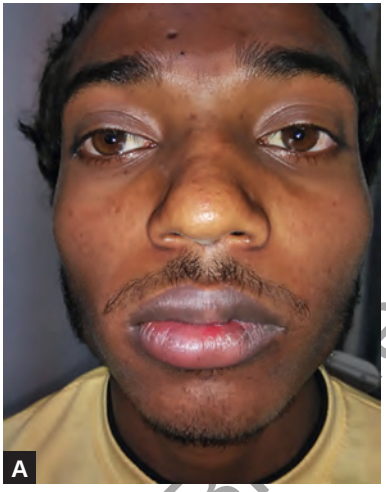
Instant Diagnostic Criteria

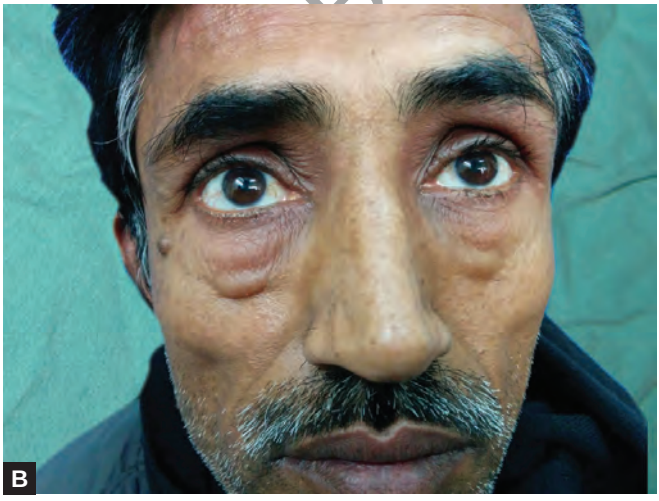
- Non-pitting, skin-colored/erythematous ill-defined edema.
- Attended by burning sensation and/or pain.
- Pruritus conspicuously absent in contrast to urticaria characterized by wheal/hives and pruritus.
- Rigorous attempts to find the offending agent.
- Skin of the face, around the mouth, the mucosa of the mouth, tongue and/or throat **(Figs 8.1A and B)**
- Acquired angioedema: It is immunologic, nonimmunologic, or idiopathic. (Axelrod S, Davis-Lorton, M. Urticaria and angioedema. *The Mount Sinai Journal of Medicine, New York.* 2011;78(5):784–802. doi:10.1002/msj.20288. PMID 21913206.)

Hereditary Angioedema

It exists in 3 forms:

- Caused by a genetic mutation inherited in an autosomal dominant form, distinguished by the underlying genetic abnormality.
- Three forms of hereditary angioedema (HAE):
  - Types I and II caused by mutations in the *SERPING1* gene, which result in either diminished levels of the C1-inhibitor protein (type I HAE) or dysfunctional forms of the same protein (type II HAE).





Figs 8.1A and B: (A) Acquired angioedema; (B) Angioedema face

Table 8.1: Acquired angioedema: drug therapy

Generic name	*Brand name*	*Fluid preparations*	*Mode of administration*
Hydrocortisone sodium Succinate injection IP 100 mg	Hycorlin	Reconstitute 100 mg of hydrocortisone sodium succinate in 5 mL of distilled water	The preparation is mixed in 100 mL of normal saline. The reconstituted fluid is injected by slow IV infusion, 10 drops per/min for requisite time
Alternative			
Dexamethasone injection IP 4 mg/2 ml Vial	Dexadran	Constitute 8 to 16 mg of Dexamethasone in 500 mL of normal saline (NS)	Slow IV infusion-10 drop per/min
Promethazine hydrochloride injection IP 25 mg vial OR	Phenergan H_1 antagonist the histamine antagonist	Fill it in the separate syringe	Slow IV infusion
Pheniramine maleate injection IP 22.75 mg vial	Avil H_1 antagonist the histamine antagonist	Fill it in the separate syringe	Slow IV infusion
Ranitidine Hydrochloride Injection IP 50 mg vial	Rantac H_2 Receptor antagonist (H2RA)	Fill it in the separate syringe	Slow IV infusion

Table 8.2: Hereditary angioedema : C1-esterase inhibitor in acute and prophylactic therapy

Brand name	*Indications*	*Dosing forms, strengths and administration*	*IV preparation precautions*	*Adverse effects*
Berinert C1 esterase inhibitor (C1-INH) Concentrate derived from human plasma, following pasteurization and nanofiltration Vials are reconstituted with 10 mL dilutent supplied	Acute abdominal, facial, or laryngeal attacks	20 units/kg IV infused slowly, not to exceed 4 mL /min	Do not use if turbid or discolored (should be colorless to slight blue) Bring to room temp, cleanse stoppers with germicidal solution, allow to dry. Use double-ended transfer needle; invert diluent vial containing 5 mL SWI over upright and slightly angled vial of drug; then rapidly insert free end of needle through the drug's vial stopper at its center; vial vacuum will draw in diluent	>4%: nausea, vomiting, diarrhea, dysgeusia (Dysgeusia is a distortion of the sense of taste), headache, abdominal pain, muscle spasms, and pain. Rarely: laryngeal edema, swelling (shoulder and chest), exacerbation of hereditary angioedema, and laryngospasm
Cinryze C1 esterase inhibitor (C1-INH) Concentrate derived from human plasma, following pasteurization and nanofiltration Each reconstituted vial contains 5 mL of 100 U/mL solution Reconstitute 2 vials for one dose	Routine prophylaxis against angio-edema attacks in adolescent and adult	1,000 units IV q3-4 days; infuse over 10 minutes (i.e. 1 mL/min)		>5%: URI, sinusitis, rash, and headache. Rarely: death to non-catheter related foreign body embolus, pre-eclampsia, stroke, exacerbation of HAE attacks.

  - Type III linked with mutations in the *F12* gene, which encodes the coagulation protein factor XII.
- All forms of HAE lead to abnormal activation of the complement system, and all forms can cause swelling elsewhere in the body, such as the digestive tract. If HAE involves the larynx, it can cause life-threatening asphyxiation. (Zuraw BL. "Clinical practice. Hereditary angioedema". N Engl J Med. 2008;359(10):1027–36.doi:10.1056/NEJMcp0803977. PMID 18768946.)
- *Pathogenesis*: This disorder is suspected to be related to unopposed activation of the contact pathway by the initial generation of kallikrein and/or clotting factor XII by damaged endothelial cells. The end product of this cascade, bradykinin, is produced in large amounts and is believed to be the predominant mediator leading to increased vascular permeability and vasodilation that induces typical angioedema 'attacks'. (Loew, Burr. "A 68-year-old woman with recurrent abdominal pain, nausea, and vomiting". MedScape. Retrieved, 19 October 2012.)

MANAGEMENT

See **Tables 8.1 and 8.2**.

RECOMMENDED READING

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