Answers to the Medical Quiz

A1. Angioneurotic edema (angioedema)
Nephritis or nephrosis
Acute ethmoid sinusitis
Dermatomyositis
Cavernous sinus thrombosis
Superior vena cava syndrome
Orbital infarction due to sickle cell disease
Vaso-occlusive crisis

A2. Angioneurotic edema (angioedema)
Because this patient also had urticarial lesions in other parts of her body.

A3. Aetiologic classification of angioedema include:

1. Immunoglobulin-E-mediated which can be further classified into (a) episodic angioedema associated with eosinophilia and characterised by attacks of angioedema, fever, striking leukocytosis and weight gain; (b) allergic reactions to food or drugs; and (c) physically induced angioedema as caused by pressure, vibratory or exercise-induced.

2. Type I hereditory angioedema which occurs on an autosomal dominant basis. In the most frequent form C_1 estrase inhibitor is low but functionally normal. Patients with type II hereditary angioedema have dysfunctional C_1 inhibitor. Recurrent episodes of angioedema, abdominal pain, nausea and vomiting, occur either spontaneously or after local trauma, especially of the upper respiratory tract, vigorous exercise, emotional stress, or because of menstrual periods.

3. Idiosyncratic, due to non-steroidal anti-inflammatory drugs or other drugs.

4. Systemic lupus erythematosus and other collagen vascular diseases may cause angioedema as well as persistent urticaria.

5. Idiopathic.

A4. Treatment will include use of anti-histamines epinephrine and steroids. Wherever the allergen is known advise avoidance.

REFERENCES

1. Gleich GJ, Schroeter AL, Marcous JP, Sacko MI, O'Connell EJ, Kohler PF. Episodic angioedema associates with eosinophilia. N Eng J Med 1984;310:1621-6.

2. Green M. Edema. In: Green M, ed. Paediatric Diagnosis Interpretation of Symptoms and Signs in Infants, Children and Adolescents. 5th ed. Philadelphia: WB Saunders Co, 1992.

3. Behrman RE, Klieman RM, Nelson WE, Vaughan III VC. Nelson Textbook of Pediatrics. 14th ed. Philadelphia: WB Saunders Co, 1992:599-601.

4. Twarog FJ. Urticaria in childhood: Pathogenesis and management. Pediatr Clin North Am 1983;30:887.

Pathogenesis:

The principal non-cytotoxic mechanism of urticaria and angioedema is interaction of antigen with mast-cell or basophil - bound IgE antibodies. The release of histamines from these cells causes vasodilatation and increased vascular permeability and stimulates an axon reflex, which produces a typical wheal and flare reaction. Leukotrienes may contribute to the edema of the IgE mediated reaction. A second mediator pathway to urticaria involves the complement system. A third mediator pathway involves the plasma kinin-forming system of the coagulation scheme. Brady-kinin is at least as potent as histamine in increasing vascular permeability.