

# ERYTHEMATOUS EDEMATOUS ERUPTION

*Nejib Doss*  
*Rym Benmously*  
*Soumaya Youssef*  
*Med. Raouf Dhaoui*

## Erythematous Edematous Eruption

### Report of Case:

A 32-year old man referred to us for an erythematous, edematous and itchy lesions scattered on the left leg evolving over a 2-week period. Seven days after the onset, bullous lesions appeared over the urticarial plaque of the leg and a reddish and vesicular patch appeared over his forearms (Fig.1).

Otherwise the patient was healthy and a laboratory examination revealed the following values: white blood cells:  $14.10^9/1$ ; eosinophils:  $3,5.10^9/1$ ;

platelets:  $450.10^9/1$ .

The results of a serum chemistry profile were within normal limits. Rheumatoid factor and anti-nuclear antibodies were negative.

Radiography of the chest and doppler of the lower extremities revealed no associated pathologies.

Intravenous antibiotics were administered for seven days without a real improvement. Biopsy specimen obtained from the forearm is shown (Fig.2).

What is your diagnosis?



Fig. 1 Bullous lesions of the forearms.

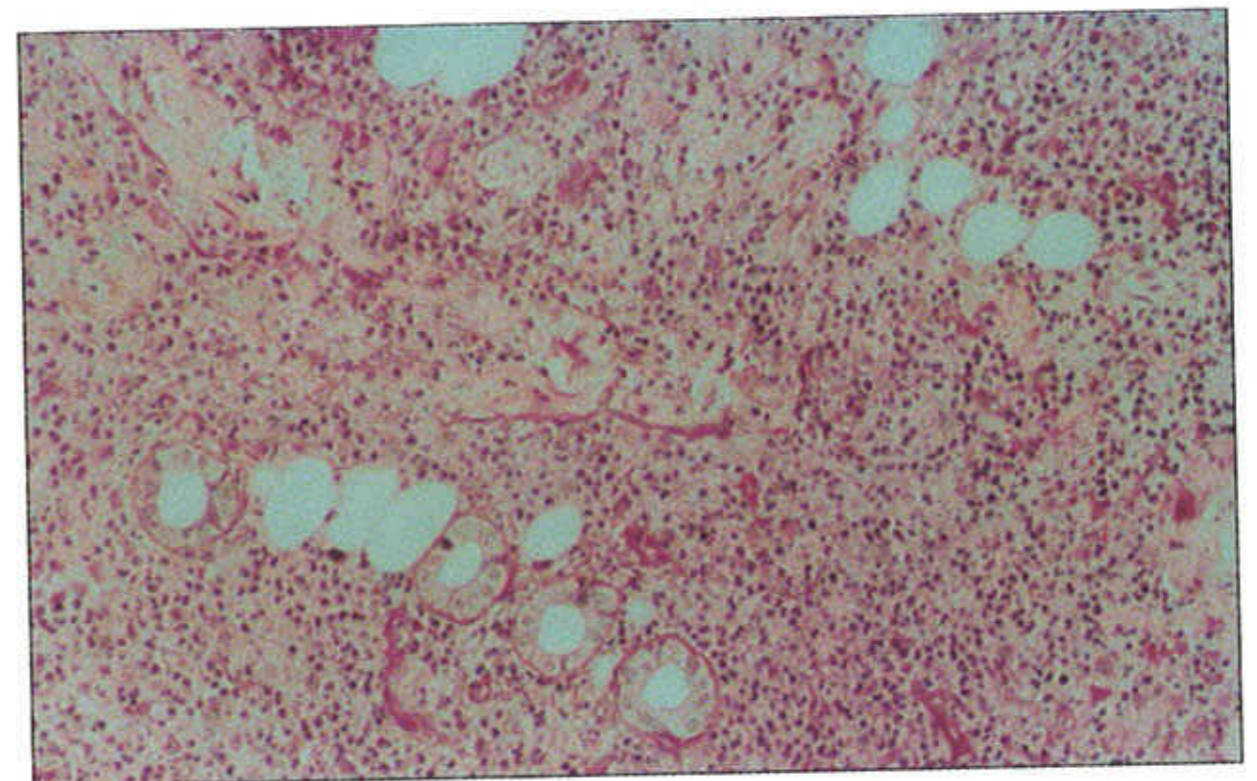


Fig. 2 Stain H& S x 200

Department of Dermatology  
Military Hospital of Tunis  
1089- Tunis.

Address for correspondence:

Nejib Doss, MD

Department of Dermatology  
Military Hospital of Tunis  
1089 Tunis ñ Tunisia.

Phone: 00 216 1 494 028

Fax # : 00 216 1 870 163

e-mail:nejib.doss@voila.fr

## Diagnosis : Wells' Syndrome

### Discussion:

Wells' Syndrome, first described by Wells in 1971 as a recurrent granulomatous dermatitis with eosinophilia and was later named eosinophilic cellulitis<sup>(1)</sup>. This syndrome is defined by the following criteria:

sudden onset of annular or circinate erythematous, edematous patches that rapidly evolve to morphea-like blue slate colored plaques.

A histological picture usually characterized by the presence of <<flame figures>>

And blood hypereosinophilia, but this criteria is not a constant feature<sup>(2)</sup>.

Wells' Syndrome may look like an bacterial cellulitis and must not be considered as part of idiopathic hypereosinophilic syndrome, because there is no visceral involvement and eosinophilia is not persistent. Kaufman<sup>(3)</sup> recently reviewed the hypereosinophilic syndrome and concluded that some diseases in this syndrome present with systemic manifestations and others only with cutaneous involvement. Wells' Syndrome, all share similar mechanisms of activation of eosinophils via various lymphokines<sup>(4)</sup>.

### Histologic Findings:

The histopathological picture is characteristic. The dermis is edematous and infiltrated with eosinophils, lymphoid cells and histiocytes between the collagen bundles. There are amorphous or granular

eosinophilic deposits on collagen, constituting flame figures<sup>(4)</sup>.

### Pathogenesis:

The cause of eosinophilic cellulitis is unknown. Etiologic hypotheses focus towards a hypersensitivity reaction. There may be triggering factors, such as myeloproliferative disease, lymphoma<sup>(5)</sup>, toxocariasis<sup>(6)</sup>, and tetanus vaccine<sup>(7)</sup>.

A pathogenic role for circulating CD4+, CD7- T cells expressing interleukin-5mRNA was suggested because the above T cells decreased in the peripheral blood when the disease was treated by recombinant interferon<sup>(8)</sup> and there is a correlation between clinical activity and eosinophil level, eosinophil cation protein and interleukin-5<sup>(4)</sup>.

This mechanism of activation of eosinophils via various lymphokines accompanied all the forms of the hypereosinophilic syndrome.

A case of congenital case of Wells' Syndrome has been reported<sup>(9)</sup>.

Once the diagnosis is confirmed, systemic corticotherapy can be started.

Topical corticosteroid therapy has been used by some authors<sup>(2)</sup>.

Relapses can be seen and are treated with corticosteroids.

Other treatments have been used and these include dapsone, colchicine, griseofulvine<sup>(10)</sup> and PUVA therapy<sup>(11)</sup>.

### References:

1. Wells GC: Recurrent granulomatous dermatitis with eosinophilia. *Trans St. Johns Hosp. Soc* 1971; 57:46-56.
2. Ferrelli C; Pinna Al; Atzori L; et al: Eosinophilic cellulitis (Wellsí Syndrome): a new case description. *Europ J of the EADV* 1999; 13:41-5.
3. Kaufman LD: The expanding clinical spectrum of multisystem disease associated with eosinophilia. *Arch Intern Med* 1997; 133:225-7.
4. Espana A; Sanz ML; Sola J; et al: Wellsí Syndrome (eosinophilic cellulitis): correlation between clinical activity, eosinophil levels, eosinophil cation protein and interleukin-5. *British Journal of Dermatology* 1999; 140: 127-130.
5. Tassava T; Rusonis PA; Whitmore SE: Recurrent vesiculobullous plaques. *Eosinophilic cellulitis. Arch Dermatol* 1997; 133:1580-1, 1583-4.
6. Hurni M.M.; Gerbig AW; Braathen LR; et al: Toxocariasis and wellsí syndrome: a causal relationship? *Dermatology* 1997; 195: 325-8.
7. Moreno M.; Luelmo J.; Monteagudo M.; et al: Casanovas A Wellsí Syndrome related to tetanus vaccine. *Int J Dermatol* 1997; 36:524-5.
8. Yagui H.; Matsushita K.; Hanaoka K.; et al: Wellsí syndrome: a pathogenic role for circulating CD4+ CD7- T cells expressing interleukin-5mRNA. *Br J Dermatol* 1997; 137: 918-23.
9. Garty BZ; Feinmesser M.; David M; et al: Congenital Wellsí syndrome. *Pediatr Dermatol* 1997; 14: 312-5.
9. Porneuf M.; Dandurand M.; Joujoux JM; et al: *Ann Dermatol Venereol* 1995; 122:119-120.
10. Diridl E.; Hyonigsmann H.; Tanew A.: Wellsí syndrome responsive to PUVA therapy. *Br J Dermatol* 1997; 137:479-81.