## Plasmocytoma-Induced Intertriginous Amyloid Purpura

Stephan Schreml, Josef Schroeder<sup>1</sup>, Heiko Siegmund<sup>1</sup>, Fabian Eder<sup>1</sup>, Philipp Babilas, Michael Landthaler, Sigrid Karrer

Department of Dermatology, <sup>1</sup>Institute of Pathology, University Medical Center Regensburg, Regensburg, Germany

#### Dear Editor:

A 58-year-old woman presented with inframammary and inguinal purpuric, non-blanching lesions (Fig. 1A, B). Eight months before, diagnosis of immunoglobulin A-light-chain plasmocytoma (type lambda) had been made. However, the etiology of skin lesions was still unclear. She did

not take any anticoagulants. Prothrombin-time as well as partial-thromboplastin time were normal, and blood count showed only slight thrombocytopenia (110/nl).

An inframammary skin biopsy showed subepidermal amorphous eosinophilic material (Fig. 1C, D) and erythrocyte extravasation. In-situ-hybridization revealed lambda

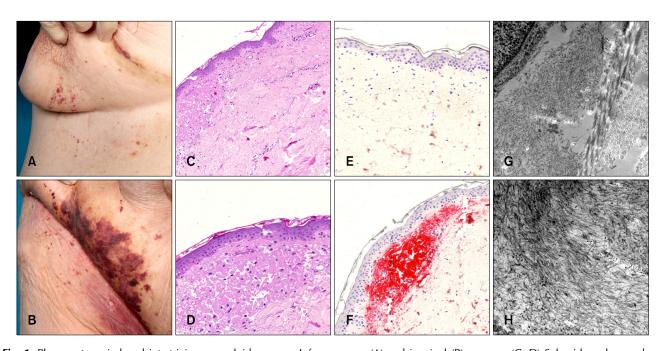


Fig. 1. Plasmocytoma-induced intertriginous amyloid purpura. Inframammary (A) and inguinal (B) purpura. (C, D) Subepidermal amorphous eosinophilic material (H&E,  $\times$ 40). In-situ hybridization for kappa (E) and lamba (F) light chains ( $\times$ 40). Only lambda light-chains were found. (G) Electron microscopy revealed amyloid (left part of the image) next to collagen fibers (right part of the image) (in situ hybridization for immunoglobulin light chains,  $\times$ 10,000). (H) Amyloid fibrils (in situ hybridization for immunoglobulin light chains,  $\times$ 20,000).

Received September 13, 2012, Revised October 9, 2012, Accepted for publication November 12, 2012

Corresponding author: Stephan Schreml, Department of Dermatology, University Medical Center Regensburg, Franz-Josef-Strauss-Allee 11, 93053 Regensburg, Germany. Tel: 49-0-941-944-9601, Fax: 49-0-941-944-9611, E-mail: stephan.schreml@ukr.de

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

light-chains (Fig. 1F, no kappa light-chains were found: see Fig. 1E); hence, systemic immunoglobulin light-chain amyloidosis was suspected. In electron microscopy, amyloid fibrils were seen (Fig. 1G, H). She received bortezomib/dexamethasone, and it was planned to induce remission for autologous stem cell therapy with lenalidomide/dexamethasone. She died from amyloid-induced heart failure prior to the planned treatment.

Typically, amyloid purpura occurs above the nipple-line, mostly on the head and neck, and particularly on the eyelids<sup>1,2</sup>. Among the suspected reasons for dermatorrhagia are that factor X is decreased by binding to amyloid fibrils, and that amyloid deposits in blood vessel walls increase vessel fragility.

As purpura may be among the first signs of systemic amyloidoses, it is of utmost importance for dermatologists to keep this sign in mind. A suspected diagnosis of amyloidosis may be the starting point for an interdiscipilinary treatment regimen as different organs may be involved<sup>3</sup>. Furthermore, it is crucial to treat the underlying cause (e.g., multiple myeloma, plasmocytoma, renal insufficiency with hemodialysis). However, there are also a

couple of hereditary systemic amyloidoses with cutaneous involvement, e.g., Meretoja's syndrome (i.e. gelsolin amyloidosis)<sup>2</sup>. Future treatments with siRNAs or antiamyloid antibodies are in the pipeline<sup>4,5</sup>, and we will see which ones make their way from bench to bedside.

### **REFERENCES**

- Eder L, Bitterman H. Image in clinical medicine. Amyloid purpura. N Engl J Med 2007;356:2406.
- Schreml S, Szeimies RM, Vogt T, Landthaler M, Schroeder J, Babilas P. Cutaneous amyloidoses and systemic amyloidoses with cutaneous involvement. Eur J Dermatol 2010;20:152-160.
- Falk RH, Comenzo RL, Skinner M. The systemic amyloidoses. N Engl J Med 1997;337:898-909.
- Clos AL, Lasagna-Reeves CA, Wagner R, Kelly B, Jackson GR, Kayed R. Therapeutic removal of amyloid deposits in cutaneous amyloidosis by localised intra-lesional injections of anti-amyloid antibodies. Exp Dermatol 2010;19:904-911.
- Hovey BM, Ward JE, Soo Hoo P, O'Hara CJ, Connors LH, Seldin DC. Preclinical development of siRNA therapeutics for AL amyloidosis. Gene Ther 2011;18:1150-1156.

http://dx.doi.org/10.5021/ad.2013.25.3.392

# Cutaneous Septic Embolism Presenting as Erythematous Plaques

Javier Galve, Priscila Giavedoni, Llúcia Alós<sup>1</sup>, Mercè Alsina-Gibert

Departments of Dermatology and <sup>1</sup>Pathology, Hospital Clínic, Universitat de Barcelona, Barcelona, Spain

#### Dear Editor:

Intravascular devices such as prosthetic vascular grafts are therapeutic tools of fundamental importance for patients with several vascular diseases. Due to the expanding usages of these devices, incidences of infectious complications are also increasing. The skin can be one of the first sites where signs of sepsis can appear. It is important to be aware of the different clinical features of septic emboli in

Received June 12, 2012, Revised November 8, 2012, Accepted for publication November 22, 2012

Corresponding author: Javier Galve, Department of Dermatology, Hospital Clínic, C/ Villarroel 170, 08036 Barcelona, Spain. Tel: 34-93-2275400 (Ext 2358), Fax: 34-93-2275438, E-mail: jgalveclinic@gmail.com

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.