BRIEF REPORT

LYMPHOSCINTIGRAPHIC EVALUATION IN PATIENTS AFTER ERYSIPELAS

J.M.P. de Godoy, M.F. de Godoy, A. Valente, E.L. Camacho, E.V. Paiva

Departments of Cardiology and Vascular Surgery (JMPdG,MFdG) and Nuclear Medicine (AV, ELC, EVP), São José do Rio Preto University School of Medicine, São Paulo, Brazil

ABSTRACT

Erysipelas (cellulitis/lymphangitis) is a superficial cutaneous infection spread by the lymphatic system which may result in permanent injury to the lymphatic vessels. The study evaluated the lymphatic drainage in the lower limbs of 30 patients with at least two episodes of erisipelas by means of lymphoscintigraphy.

Twenty-two (73%) were female and 8 (27%) were male with ages ranging from 26 to 77 years (mean 52 years). Lymphoscintigraphy was performed by intradermal administration of 500 μ Ci (20 Mbq) of 99m Tc antimony sulfur-colloid in two interdigital spaces of the feet. Whole body scintigraphy was performed 45 minutes after the administration of the radiopharmaceutical using a computerized gamma camera. Significant lymphatic abnormalities were found in 23 (77%) of these patients.

We conclude that most patients with repeated erysipelas have significant and even permanent abnormalities in regional lymphatic drainage. Recurrent erysipelas suggests underlying primary or secondary lymphedema.

Erysipelas (also termed cellulitis/ lymphangitis) is a superficial cutaneous infection spread by the lymphatic system and often caused by β-hemolytic *streptococcus* pyogenes. However, other types of bacteria may also be responsible (1). In patients with lower extremity lymphedema in conjunction with erysipelas, 77% had evidence of primary or secondary abnormalities of the lymphatic system (2). Radioisotope lymphoscintigraphy displays transport of the radiopharmaceutical through the lymphatic system and is a marker of lymph flow (3). Clinical edema develops when the accumulated interstitial fluid is 20% greater than the normal volume (4). Lymphedema, defined as impaired lymphatic flow, is characterized by an increase in the amount of interstitial water and proteins derived from plasma along with an accumulation of lymphocytes, monocytes and migrating dendritic cells. The transport of autologous antigens, of parenchymal byproducts and immune cells is also restricted which may account for the proliferation of fibroblasts (sclerosis) and epithelial cells (acanthosis) in the affected limb (5).

In this study, we evaluated the prevalence of impaired lymph drainage (lymphedema) in patients with one or more episodes of erysipelas.

MATERIAL AND METHODS

The lower limbs of thirty patients with two or more episodes of erysipelas (*Fig. 1*) were examined by lymphoscintigraphy 40 to

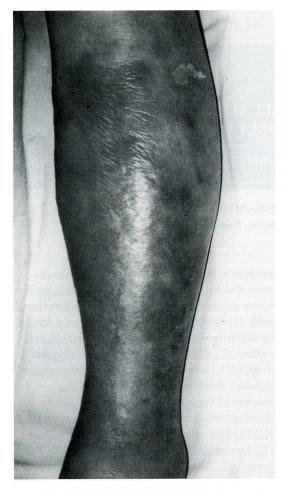


Fig. 1. Typical erysipelas (patient #13).

90 days after treatment. Of these patients 22 (73%) were female and 8 (27%) were male, with ages ranging from 26 to 77 years (mean 52 years). Lymphoscintigraphy was obtained after the intradermal administration of 500 _Ci (20 Mbq) of ^{99m}Tc antimony sulfurcolloid into two interdigital spaces of the feet. Scintigraphy was performed 45 minutes after the administration of the radiopharmaceutical using a computerized gamma camera (Elscint SP 4 Haifa). Two experienced nuclear medicine physicians, blinded to each other's opinions evaluated the images, and in cases of disagreement a third reviewer rendered the final interpretation.

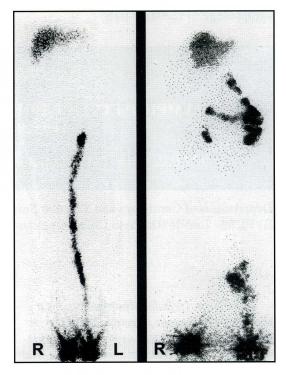


Fig. 2. Typical lymphoscintigrams of 2 different patients demonstrating impaired radiotracer transport in the right leg after repeated episodes of "erysipelas".

RESULTS

Lymphoscintigraphy showed altered lymphatic drainage in 23 patients (77%) (Fig. 2) with a clinical history of erysipelas as outlined in *Table 1*. The two reviewing physicians agreed in 27 patients and a third physician established the final diagnosis in the other 3 patients.

COMMENT

The prevalence of deranged lymphatic drainage as demonstrated by lymphoscintigraphy in these 30 patients with two or more episodes of erysipelas was 77%. This result was similar to that previously described (2), suggesting that these patients had underlying primary or secondary lymphedema.

TABLE 1

Demographics of 30 Patients With Erysipelas (Episodes) Studied by Lymphoscintigraphy (Y=lymphatic abnormalities; N=no abnormality)

Patient	Age	Sex	Leg(s)	# Episodes	Edema Manifestation	Lymphoscintigraphy 1
01	29	F	L	2	+	Y
02	56	F	L	2	-	N
03	60	F	R	3	-	Y
04	56	F	L	2	-	N
05	48	M	L	2	-	Y
06	67	M	R	2	+	Y
07	49	F	R	2	-	N
08	45	F	R	2	-	N
09	54	F	L+R	6	+	Y
10	43	F	R	4	+	Y
11	65	M	R	2	-	Y
12	76	F	R	2	-	Y
13	50	F	L	2	-	N
14	47	F	R	5	+	Y
15	47	F	R	2	+	Y
16	40	M	L	. 2	-	Y
17	58	F	L	2		Y
18	66	F	L	2	-	Y
19	71	F	L+R	4	+	Y
20	62	F	R	2	-	N
21	43	M	R	3	+	Y
22	52	M	R	2	-	Y
23	54	\mathbf{F}	L	2	-	N
24	63	F	R+L	8	+	Y
25	64	F	L	2	=	Y
26	26	F	R	2	=	Y
27	41	F	R	2	-	Y
28	30	M	L	4	+	Y
29	32	F	L	2	-	Y
30	29	M	R	2	-	Y

F - female; M - male; L - left; R - right

Previous investigations have shown by immunohistochemical analysis of the skin in secondary lymphedema, that hyperkeratosis, acanthosis, high density of Langerhan cells and epidermal protrusion into the dermis are common (1).

Normally, macrophage migration and regional lymphocytes promptly recognize when particular matter or microorganisms penetrate the skin. In lymphedema, obstructed lymph transport renders this defense mechanism much less effective. The

result allows local bacterial colonization of the dermis and development of regional infection (6).

Chronic prophylaxis with antibiotics may retard progression of lymphedema including reducing the sequelae of hyperkeratosis and fibrosis of the dermis as well as further obliteration of the lymphatic pathways including regional lymph nodes by recurrent infection (6).

Our findings suggest that repeated erysipelas is not only injurious to lymphatics but may be a harbinger of underlying or developing primary or secondary lymphedema. Lymphoscintigraphy is indispensable for determining whether lymphatic drainage is permanently impaired after erysipelas and whether latent primary or secondary lymphedema coexists.

REFERENCES

 Medeiros, A: Linfangites e erisipelas: fatos e perspectivas. In: Pinto RA, Garrido M.

- Linfangites e Erisipelas. Editora Byk Procienx, São Paulo, 1985.
- Volgelfang, D: Fisiopatologia do edema linfático. In: *Linfologia Básica*. Volgelfang D. (Ed.) Icome, São Paulo, 1995.
- Volgelfang, D: Linfografia Radioisotópica. In: Linfologia Básica. Volgelfang D. (Ed.) Icome, São Paulo, 1995.
- Giménez, CJA:. Microcirculácion y sistema linfático. VII Congresso Pan-americano de Flebologia y Linfologia, Madrid, Espanha, 1997.
- Olzewski, WL: El sistema linfático. Patologia Vascular 3 (1995), 59-66.
- Olszewski, WL: Recurrent bacterial dermatolymphangiolymphadenitis (DLA) responsible for progress of lymphedema. Lymphology, 29(Suppl) (1996), 331-334.

José Maria Pereira de Godoy,MD Rua Floriano Peixoto, 2950 São José do Rio Preto, SP – Brazil CEP: 15010-020 E-mail: godoyjmp@riopreto.com.br