Year : 2013

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Buss Guillaume, 2013, Two Cases of Interferon-Alpha-Induced Sarcoidosis Koebnerized along Venous Drainage Lines: New Pathogenic Insights and Review of the Literature of Interferon-Induced Sarcoidosis

Originally published at : Thesis, University of Lausanne

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Document URN : urn:nbn:ch:serval-BIB_E989ECBF89DF8

UNIVERSITE DE LAUSANNE - FACULTE DE BIOLOGIE ET DE MEDECINE

Département de Médecine
Service d'immunologie et allergie

Two Cases of Interferon-Alpha-Induced Sarcoidosis Koebnerized along Venous Drainage Lines: New Pathogenic Insights and Review of the Literature of Interferon-Induced Sarcoidosis

THESE

préparée sous la direction du Professeur M. Gilliet, Médecin-chef

et présentée à la Faculté de biologie et de médecine de
l'Université de Lausanne pour l'obtention du grade de

DOCTEUR EN MEDECINE

par

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Originaire de Kirchberg (BE)

Lausanne

2013

UNIL | Université de Lausanne
Faculté de biologie
et de médecine

Ecole Doctorale
Doctorat en médecine

Imprimatur

Vu le rapport présenté par le jury d'examen, composé de

Directeur de thèse	*Monsieur le Professeur* **Michel Gilliet**
Co-Directeur de thèse	
Expert	
Directrice de l'Ecole doctorale	*Madame le Professeur* **Stephanie Clarke**

la Commission MD de l'Ecole doctorale autorise l'impression de la thèse de

Monsieur Guillaume Buss

intitulée

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Lausanne, le 18 février 2014

pour Le Doyen
de la Faculté de Biologie et de Médecine

Madame le Professeur Stephanie Clarke
Directrice de l'Ecole doctorale

Case and Review

Dermatology

Dermatology 2013;226:289–297
DOI: 10.1159/000346244

Received: August 10, 2012
Accepted after revision: November 26, 2012
Published online: July 23, 2013

Two Cases of Interferon-Alpha-Induced Sarcoidosis Koebnerized along Venous Drainage Lines: New Pathogenic Insights and Review of the Literature of Interferon-Induced Sarcoidosis

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Key Words
Hepatitis C · Interferon-α · Intravenous drug user · Koebner phenomenon · Sarcoidosis · Venous drainage lines

Abstract
Sarcoidosis is a systemic granulomatous disorder of unknown origin commonly affecting the lung, the lymphoid system and the skin. We report here two cases of cutaneous sarcoidosis in two former intravenous drug users following interferon (IFN)-α and ribavirin therapy for chronic hepatitis C. Both patients developed skin sarcoidosis along venous drainage lines of both forearms, coinciding with the areas of prior drug injections. The unique distribution of the skin lesions suggests that tissue damage induced by repeated percutaneous drug injections represents a trigger for the local skin manifestation of sarcoidosis. Interestingly, skin damage was recently found to induce the local expression IFN-α, a well-known trigger of sarcoidosis in predisposed individuals. Here we review the literature on sarcoidosis elicited in the context of IFN-α therapy and propose a new link between the endogenous expression of IFN-α and the induction of disease manifestations in injured skin.

Introduction

Sarcoidosis is a multiple granulomatous disorder of unknown origin affecting the lung (>90% of cases), the lymphoid system (33%), the liver (50–80%), the eyes (11–83%) and the skin (25%) [1]. Interferon (IFN)-α is commonly used for treatment of chronic hepatitis C because of its antiviral and immunomodulatory activities in combination with ribavirin [2]. We report one case of sarcoidosis limited to the skin and one case of systemic sarcoidosis with cutaneous involvement, both of them koebnerized along venous drainage lines in two former intravenous drug users treated with IFN-α and ribavirin for chronic hepatitis C.

Case Reports

Case 1

A 36-year-old man with a history of intravenous drug use and chronic hepatitis C was started on a 48-week course of pegylated IFN and ribavirin. The patient had injected heroin into the veins of both forearms on daily basis for several years. However, he said he had stopped any drug consumption for more than 6 months before starting the treatment. Chest X-ray before treatment was normal and abdominal sonography showed mild hepatomegaly. At week 30 of IFN-α and ribavirin therapy, painless, firm, erythematous skin papules developed in a linear distribution along the cephalic and the median antebrachial veins of both forearms, former areas of drug injections (fig. 1). The patient did not complain of itching, and no other site was involved. He had no history of trauma, infection or vaccination, and there was no family history of similar skin diseases. A skin biopsy revealed dermal inflammatory infiltrates with the presence of numerous noncaseating granulomas (fig. 2). No foreign material was detected by polarization light microscopy. This histologic finding along with the clinical picture was consistent with skin sarcoidosis. Furthermore, we found elevated serum levels of angiotensin-converting enzyme and mediastinal, retroperitoneal and axillary adenopathies, consistent with a systemic involvement of the sarcoidosis. Topical corticosteroids were administered and antiviral therapy was continued to maximize the chance of cure

G. Buss and V. Cattin contributed equally to this work.

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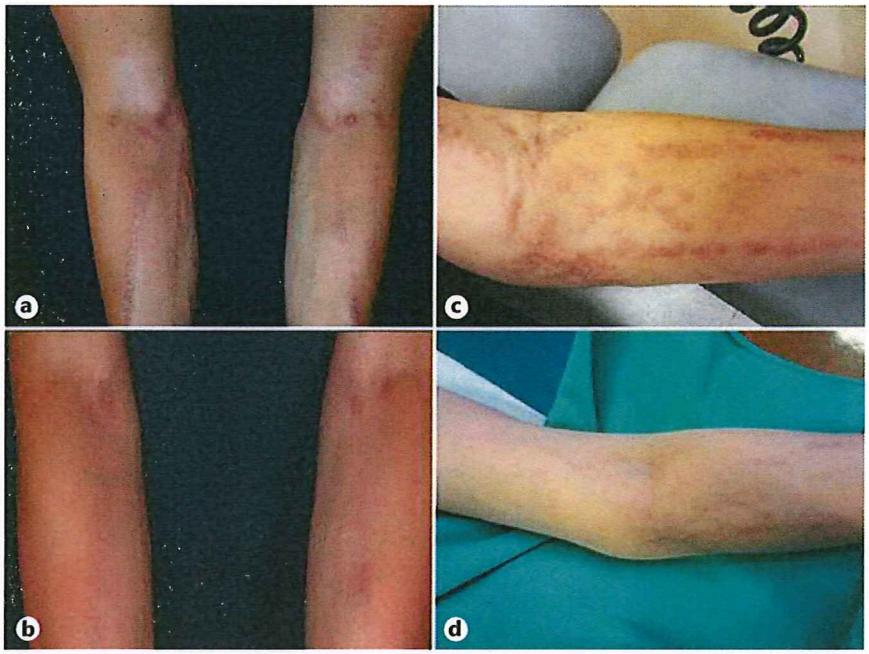

Fig. 1. Clinical aspect of skin sarcoidosis along forearm veins of patient 1 (**a**, **b**) and patient 2 (**c**, **d**). **a**, **c** At presentation. **b**, **d** After recovery.

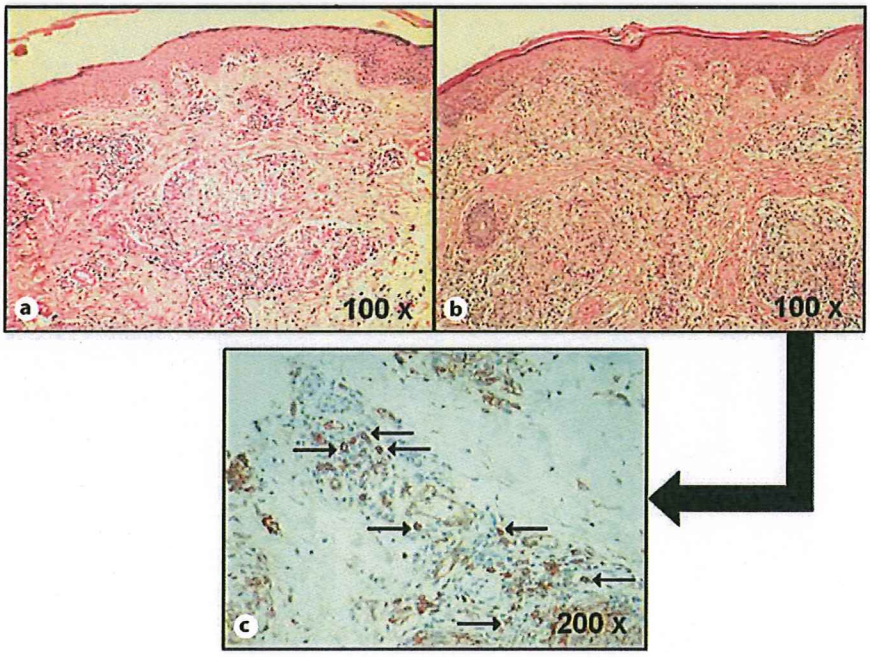

Fig. 2. HE staining of tissue sections showing numerous sarcoid granulomas in the dermis of patients 1 (**a**) and 2 (**b**). Immunostaining with anti-CD123 antibody (**c**) revealed numerous pDCs in the inflammatory dermal infiltrate between the granulomas of patient 2 (some of which are indicated by black arrows).

from hepatitis C. At week 37, the patient developed polyuria and polydipsia in the setting of severe hypercalcemia and mild cholestasis, which has been described in systemic sarcoidosis [3]. Because IFN-α and ribavirin therapy has been associated with the development of systemic sarcoidosis [4, 5], this treatment was discontinued and we introduced a short course of systemic steroids (prednisone 30 mg/day) as recommended in symptomatic hypercalcemia [6]. This led to a rapid regression of skin lesions (fig. 1) and resolution of hypercalcemia and cholestasis. Prednisone was progressively reduced and stopped after 2 months. No relapse of the cutaneous or systemic sarcoidosis was observed. Despite the incomplete IFN and ribavirin treatment of 37 weeks instead of 48 weeks, the patient remained without evidence of hepatitis C virus (HCV) viremia after 6 months.

Case 2

A 42-year-old woman with a history of intravenous drug use and genotype 6 chronic hepatitis C was started on a 48-week course of pegylated IFN and ribavirin. The patient had injected drugs intravenously into both forearms on a daily basis for several years. She told us that she had stopped any drug consumption 5 years before starting the treatment. After 16 weeks of IFN-α and ribavirin therapy, the patient developed painless, nonpruriginous, firm, erythematous skin papules in a linear distribution along the veins of the forearms and of the dorsal aspect of the hands bilaterally, former areas of drug injections (fig. 1). Complete examination revealed no other cutaneous lesions. She had no history of trauma, infection or vaccination and there was no family history of similar skin diseases. A skin biopsy revealed dermal noncaseating granulomatous inflammatory infiltrates, containing multinucleated giant cells (fig. 2). No foreign material was detected by polarization light microscopy. This histologic finding along with the clinical picture was consistent with skin sarcoidosis. Serum levels of angiotensin-converting enzyme, blood chemistry and chest X-ray were normal and excluded a systemic involvement of the sarcoidosis. In the absence of a systemic sarcoidosis, we did not introduce systemic treatment and IFN and ribavirin therapy was continued for a total of 48 weeks. Skin lesions spontaneously disappeared rapidly after completion of IFN-α and ribavirin therapy. The patient remained without evidence of HCV viremia 6 months after the end of the IFN-α and ribavirin therapy.

Discussion

Sarcoidosis is a multisystem disorder of unknown cause. It commonly affects young and middle-aged adults and frequently presents with bilateral hilar lymphadenopathy, pulmonary infiltration, and ocular and skin lesions. The liver, spleen, lymph nodes, salivary glands, heart, nervous system, muscles, bones, and other organs may also be involved [7]. Although the exact cause of sarcoidosis remains unknown, it is thought to represent an exaggerated immune response to a yet unknown antigenic stimulus leading to the formation of granulomas. These granulomas are characterized by a predominance of CD4+ T helper cells (TH), producing large amounts of TH1-type cytokines IFN-γ and IL-2. Macrophages and dendritic cells are also activated to produce proinflammatory cytokines such as IL-1, TNF-α, and IL-12. By contrast, relatively few TH2 are found in the granulomas, confirming that this is a TH1-mediated disease [8, 9].

Here we report two cases of sarcoidosis triggered by IFN-α and ribavirin therapy in patients with chronic hepatitis C. IFN-α is a potent immune stimulator that is used for the treatment of infectious and neoplastic diseases. IFN-α was shown to induce the activation and maturation of dendritic cells into potent antigen-presenting cells, and to directly stimulate T cell activation and TH1 polarization. Furthermore, IFN-α differentiates immature B cells into antibody-secreting plasma cells and promotes cytotoxicity of NK cells [10]. Ribavirin is a nucleoside analogue and was found to potentiate IFN-α expression potentially via Toll-like receptor (TLR) 7 and TLR8 [11, 12], providing an explanation for why the combination therapy of IFN-α plus ribavirin may be more efficacious in treating hepatitis C [13]. It is not surprising that this Th1-promoting therapy exacerbates sarcoidosis in predisposed individuals.

The first association between IFN-α and sarcoidosis was reported in 1993 [14]. Since then, 136 cases of IFN-induced sarcoidosis were reported (table 1). A systematic review of this literature revealed that there was no major sex or age bias: 59% of patients were female and the patients' age ranged from 23 to 76 years (with a median of 50.4 years). Our review of 138 cases (including our 2 patients) revealed that cutaneous manifestations of sarcoidosis were evident in 49% of the patients treated with IFN-α, whereas only half as many (25%) [1] of the sarcoidosis patients had skin involvement in the absence of IFN-α treatment. This is consistent with a previous report of IFN-induced sarcoidosis observing that in 50% of cases, skin involvement is the first manifestation of sarcoidosis, and that in 20% of these cases sarcoidosis remains confined to the skin [15]. The onset of IFN-α-induced sarcoidosis varies from 15 days to 30 months after the beginning of treatment [16]. Ninety-five percent of the cases with IFN-α-induced sarcoidosis have been reported to improve or remit after discontinuation or a decrease in the dosage of the drug (table 1).

Interestingly, 83% of patients receiving IFN-α therapy for the treatment of hepatitis C had IFN-induced sarcoidosis, while only 16% of patients with proliferative disorders receiving adjuvant IFN-α therapy had the disease. Several reasons may account for these differences: (1) hepatitis C patients may have received higher doses of IFN-α, although, due to a lack of protocol details in most reports, no formal conclusion can be drawn; (2) in most antiviral protocols, IFN-α is given in combination with ribavirin, which may represent an additional trigger of endogenous IFN-α via TLR7 and TLR8 as described above, and (3) the HCV itself may trigger the expression of endogenous IFN-α and sarcoidosis as suggested by some reports describing sarcoidosis in untreated hepatitis C patients [17, 18].

The Koebner phenomenon (KP) represents a typical characteristic of cutaneous sarcoidosis. In our review, we found that in 29% of cases skin sarcoidosis was triggered by a classical KP (table 1). Typical triggers of the KP in sarcoidosis are foreign material present in tattoos [19–21], synthetic fillers used for cosmetic procedures [22], or talc injected in the context of intravenous drug abuse [23, 24]. Scars are also a typical trigger of KP and sarcoidosis [25, 26], although most of the time foreign particles are undetectable in cutaneous biopsies, namely in 51 (78%) of 65 patients with scar sarcoidosis [25]. Similarly in our two cases, we did not find any foreign body material in the tissue samples by polarization light microscopy. It is possible that the repeated venipuncture over several years has resulted in tissue injury, chronic subclinical inflammation and scar formation, which koebnerized the cutaneous sarcoidosis despite the absence of any venous puncture for several months or even years. Interestingly, endogenous IFN-α was recently found to be expressed in wounded skin upon mechanical or chemical injury and chronic skin inflammation in psoriasis [27]. The endogenous IFN-α was found to be produced by a subset of dendritic cells called plasmacytoid dendritic cells (pDCs), which are usually absent from the skin but rapidly infiltrate wounded skin. At this site, pDCs recognize host-derived nucleic acids released by dying cells into the injured tissue via TLR7 and TLR9, exclusively expressed by the pDCs [28]. This process ultimately leads to the production of a large quantity of IFN-α, which initiates wound healing. It is possible that in our two patients, the repeated trauma to the skin may have triggered a sustained skin infiltration by pDCs and their activation to produce

Table 1. IFN-induced sarcoidosis reported [5, 13, 14, for a review 15, 16, 17, 20, 32–88]

Authors	Age/sex	Indication for IFN	Organ involvement	Treatment	Situation	Outcome
Lee et al. [24]	39/M	HCV	lymphadenopathy linear forearm papules	IFN-α + R	discontinuation of topical steroid	resolution (2 weeks)
Bonnet et al. [89]	–/–	HCV	lung	IFN-α	suspension	improvement
Godoy et al. [90]	–/–	HCV	lung, skin	IFN-α + R		resolution
Godoy et al. [90]	45/M	HCV	lung, skin	IFN-α + R		resolution
Alazemi and Campos [91]	42/M	HCV	lymphadenopathy lung subcutaneous nodules	IFN-α + R	discontinuation	resolution (12 months)
Iwashita et al. [92]	72/F	HCV	skin lymphadenopathy uveitis, heart	IFN-α + R	discontinuation of oral steroid	improvement
Atluri et al. [19]	45/M	HCV	tattoo papules	IFN-α + R	oral steroid	resolution
Bitetto et al. [93]	64/F	HCV	nasolabial folds	IFN-α + R	oral steroid	resolution (few weeks)
Meller et al. [94]	55/F	HCV	nodules/papules	IFN-α + R	discontinuation of topical steroid	resolution
Honsová et al. [95]	–/–	HCV	skin	IFN-α + R	discontinuation	resolution
Sanchez-Ruano et al. [96]	43/M	HCV	lung, skin	IFN-α + R		
Perera and Calonje [21]	54/M	HCV	lung lymphadenopathy ulcerated tattoo site nodules	IFN-α + R	topical steroid	improvement
Perez-Gala et al. [26]	59/F	HCV	skin	IFN-α + R	topical steroid	resolution (5 months)
Song and Das [97]	–/F	HCV	nodules	IFN-α + R		
Adla et al. [98]	44/M	HCV	liver	IFN-α + R		
Doycheva et al. [99]	49/F	HCV	ocular lesions renal failure	IFN-α + R	decreased oral steroid topical steroid	resolution (6 months)
Doycheva et al. [99]	37/M	HCV	ocular lesions	IFN-α + R	decreased topical steroid	resolution
Doycheva et al. [99]	65/F	HCV	ocular lesions	IFN-α + R	decreased topical steroid	resolution
Yan et al. [100]	23/F	HCV	ocular lesions lymphadenopathy	IFN-α + R	topical steroid	resolution
Lopez et al. [101]	55/M	HCV	lung lymphadenopathy nodules	IFN-α + R	none	resolution (6 months)
Petousi and Thomas [102]	30/F	multiple sclerosis	lung lymphadenopathy	IFN-α + β	discontinuation of oral steroid	resolution (1 month)
Chakravarty et al. [103]	39/F	multiple sclerosis	lung papules/nodules	IFN-β	discontinuation of oral steroid hydroxychloro-quine methotrexate	resolution (6 months)
Carbonelli et al. [104]	35/M	multiple sclerosis	lung, liver lymphadenopathy	IFN-β		
O'Reilly et al. [105]	52/F	multiple sclerosis	lung lymphadenopathy	IFN-β	discontinuation	resolution (6 months)
Novoa et al. [106]	54/F	HCV	lung, liver papules/nodules	IFN-α + R	discontinuation	resolution
Albaker [107]	48/M	HCV	hypercalcemia lung lymphadenopathy	IFN-α + R	discontinuation	resolution

Table 1 (continued)

Authors	Age/sex	Indication for IFN	Organ involvement	Treatment	Situation	Outcome
Oudghiri et al. [30]	47/F	HCV	lung, CNS papules	IFN-α + R	discontinuation of oral steroid	death
North and Mully [108]	52/M	melanoma	lymphadenopathy papules/nodules	IFN-α	discontinuation	resolution (6 months)
Morley et al. [109]	67/F	HCV	ocular lesions	IFN-α + R	oral steroid	resolution (12 months)
Sionidou et al. [110]	36/M	polycythemia vera	lung lymphadenopathy	IFN-α	discontinuation of oral steroid	
Cardoso et al. [111]	43/M	HCV	lung lymphadenopathy papules/nodules	IFN-α + R	discontinuation	improvement
Benzagmout et al. [31]	47/F	HCV	CNS, lung subcutaneous nodules	IFN-α + R	discontinuation of systemic steroid	death
Gayet et al. [112]	57/F	HCV	lung lymphadenopathy subcutaneous nodules	IFN-α + R	none	resolution (6 months)
Heinzerling et al. [113]	50/M	melanoma	lymphadenopathy lung, scar sarcoid	IFN-α	discontinuation	resolution
Heinzerling et al. [113]	47/M	melanoma	lymphadenopathy lung	IFN-α	discontinuation	resolution
Heinzerling et al. [113]	47/M	melanoma	lymphadenopathy lung	IFN-α	discontinuation	resolution (2 months)
Pelletier et al. [18]	60/M	melanoma	lung subcutaneous nodules	IFN-α	discontinuation	resolution
Rodriguez-Lojo et al. [114]	60/F	HCV	subcutaneous nodules	IFN-α + R	discontinuation	resolution (2 months)
Faurie et al. [115]	59/M	HCV	lymphadenopathy lung	IFN-α + R	none	resolution
Faurie et al. [115]	65/F	HCV	lymphadenopathy lung	IFN-α + R	none	resolution
Faurie et al. [115]	49/F	HCV	lymphadenopathy lung subcutaneous nodules	IFN-α	discontinuation	resolution
Faurie et al. [115]	76/F	HCV	uveitis subcutaneous nodules	IFN-α + R	oral steroid	improvement
Faurie et al. [115]	56/F	HCV	lymphadenopathy lung	IFN-α + R	oral steroid	resolution
Le Bras et al. [116]	54/F	HCV	lymphadenopathy lung subcutaneous nodules	IFN-α + R	oral steroid	resolution (7 months)
Shuja et al. [23]	59/M	HCV	linear forearm papules	IFN-α + R	none	
Zampino et al. [117]	59/F	HCV	nodule	IFN-α + R	surgery	resolution
Descamps et al. [22]	48/F	HCV	lung papules/nodules filler site reaction	IFN-α + R	oral steroid	resolution
Descamps et al. [22]	64/F	HCV	lymphadenopathy lung subcutaneous nodules	IFN-α + R	discontinuation of oral steroid	resolution (3 months)
Current report	36/M	HCV	lymphadenopathy hypercalcemia linear forearm papules	IFN-α + R	discontinuation of oral steroid	resolution (2 months)
Current report	42/F	HCV	linear forearm papules	IFN-α + R	topical steroid	resolution (6 months)

R = Ribavirin.

IFN-α, triggering the local manifestation of sarcoidosis. The skin IFN-α levels may be further enhanced by the systemic IFN-α levels and, particularly, by ribavirin, which may further trigger activation of skin pDCs via TLR7. Our hypothesis is supported by the finding of numerous pDCs in the dermal infiltrate surrounding the sarcoid granulomas of patient 2 (fig. 2).

Finally, regarding management of IFN-induced sarcoidosis in HCV infection, we provide a decision algorithm (fig. 3): IFN discontinuation should be based on the extent of sarcoidosis. For instance, antiviral therapy could be continued in skin-limited or asymptomatic stage I pulmonary sarcoidosis, especially if the antiviral therapy has been introduced for more than 6 months. Maintaining treatment to maximize hepatitis C remission has to be evaluated jointly with hepatologists. Systemic steroids should be considered with caution in symptomatic IFN-induced sarcoidosis, because it may increase the viral load [29]. Moreover, most cases resolve spontaneously after drug discontinuation. Nevertheless, we recommend aggressive treatment of neurologic, ocular, cardiac and renal sarcoidosis or hypercalcemia, because death [30, 31], severe loss of vision, fatal arrhythmias, or insidious renal damage may ensue without therapy.

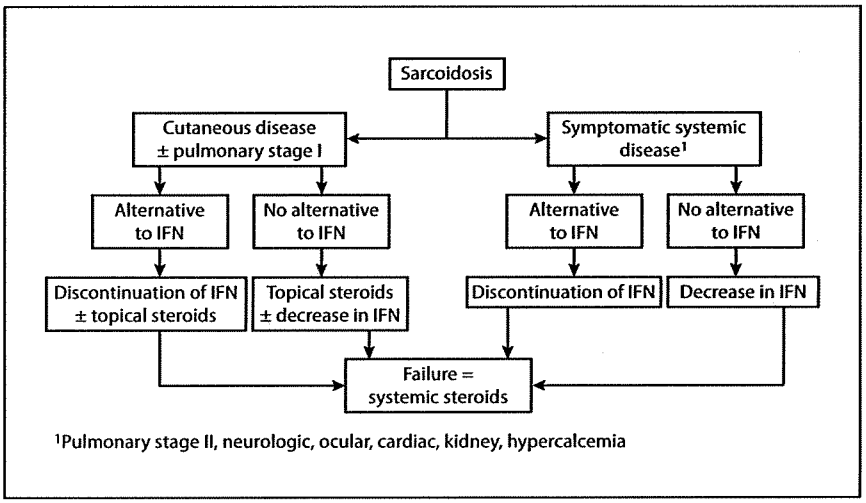

Fig. 3. Decision algorithm for IFN-induced sarcoidosis: IFN discontinuation should be based on the extent of sarcoidosis. For instance, antiviral therapy could be continued in skin-limited or asymptomatic stage I pulmonary sarcoidosis, especially if the antiviral therapy has been introduced for more than 6 months. Maintaining treatment to maximize hepatitis C remission has to be evaluated jointly with hepatologists. Systemic steroids should be considered with caution in symptomatic interferon-induced sarcoidosis, because it may increase the viral load [29]. Moreover, most cases resolve spontaneously after drug discontinuation. Nevertheless, we recommend aggressive treatment of neurologic, ocular, cardiac and renal sarcoidosis or hypercalcemia, because death [30, 31], severe loss of vision, fatal arrhythmias, or insidious renal damage may ensue without therapy.

Conclusion

We report two cases of cutaneous sarcoidosis manifested by linear erythematous skin papules along forearm veins in two former intravenous drug users treated by IFN-α and ribavirin for chronic hepatitis C. In the literature we found two other cases with a similar distribution [23, 24]. Because skin injury was recently found to induce local IFN-α expression by infiltrating pDCs and IFN-α is a well-known trigger of sarcoidosis in predisposed individuals, we propose that our patients' specific skin manifestations along venous drainage lines may result from the additional IFN-α expression in previously injured skin. Moreover, it was shown that ribavirin therapy may further enhance IFN-α expression in previously wounded skin. Identification of pDCs surrounding granulomatous inflammation in our tissue samples seems to be an argument in that direction, supported by the absence of foreign body material detected by polarization light microscopy. This condition may represent a key point in the understanding of the physiopathological mechanisms leading to scar sarcoidosis. Further investigations need to be performed to confirm this hypothesis.

Disclosure Statement

The authors have no conflicts of interest to declare.

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