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Meglumine antimoniate is more effective than sodium stibogluconate in the treatment of cutaneous leishmaniasis

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Abstract

Sodium stibogluconate (SSG, Pentostam) and meglumine antimoniate (MA, Glucantime) are two antimonials that are widely used to treat cutaneous leishmaniasis (CL), but the relative efficacies of these treatments are not clear. The aim of this study is to compare the efficacy of intralesional SSG with intralesional MA therapy in the treatment of CL. One month after completion of the therapy, 1431 of 1728 patients (82%) who received intralesional MA showed complete clinical cure compared to 1157 of 1728 patients (67%) in the SSG group. Patients who did not respond to the first round of therapy were re-administered the same treatment but with twice weekly injections. Following completion of the second course of therapy, 237 of 297 patients (80%) in the MA group and 407 of 561 patients (72%) in the SSG group healed their lesions by 1-month post-treatment. At both times, the differences in cure rates between MA and SSG groups were statistically significant ($p<0.05$). Cure rates in the MA group were always significantly higher than SSG groups irrespective of other parameters including age, gender, lesion site and type of lesion. Intralesional MA is more effective than intralesional SSG in the treatment of CL.

Keywords

Cutaneous leishmaniasis; meglumine antimoniate; sodium stibogluconate

Introduction

Over 12 million people currently suffer from leishmaniasis, and approximately 2 million new cases occur annually, making it a global health problem and a World Health

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Organization (WHO) classified neglected tropical disease (NTD) (1). Cutaneous leishmaniasis (CL) manifests as a localized solitary or multiple lesions on the face, head, neck and extremities (2,3) that can become chronic, leading to significant tissue destruction and disfigurement. In 98 countries worldwide, 350 million people are at risk of CL disease, the majority of who are residing in developing countries (1,4). There are two forms of CL: Old World and New World (1). Old World CL is seen in Mediterranean countries, including Turkey, the Middle East, the Arabian Peninsula, Africa, Western Asia and the Indian subcontinent (5,6). In Turkey, CL is highly endemic in the Sanliurfa province of southeastern Anatolia and is caused by *Leishmania tropica* or *Leishmania major* (5).

The pentavalent antimonials, i.e. meglumine antimoniate (MA) and sodium stibogluconate (SSG), are the commonly used drug of choice for the treatment of CL in most disease endemic-countries including Turkey (5,7–11). This therapy involves daily intramuscular or intravenous injections of the drug for 3 weeks or 5–8 intralesional injections once or twice weekly (2,7–9,12,13). Soto et al. (14) had previously reported that systemically administered MA and SSG have comparable efficacies against CL. However, the relative efficacies of intralesional MA and SSG therapies in the treatment of CL are not clear.

The goal of the present study was to compare the clinical efficacy of intralesional MA versus SSG in the treatment of CL by analyzing clinical records of 3456 CL patients from the province of Sanliurfa, Turkey, who were treated with intralesional MA or SSG.

Study design and methods

A retrospective evaluation was made of the clinical records of 3456 patients with parasitologically confirmed CL who were treated with intralesional MA ($n = 1728$) or SSG ($n = 1728$) at Harran University Medical Faculty, Dermatology Clinic and at Sanliurfa Public Health Department, Şark Çıbanı Center between January 2009 and December 2012. Approval for this study was obtained from the Ethics Committee at Harran University. The socio-demographic characteristics and other parameters including size, duration, type, location and number of lesions, age, gender, treatment applied and lesion healing were analyzed.

All patients were treated with eight intralesional injections of either MA or SSG twice weekly at a dosage of 50 mg cm^{-2} (0.5 mL) according to the size of their lesion (total amount 0.5–5 mL per lesion per injection). Total re-epithelization was considered to be complete clinical cure, whereas decreased induration and erythema were defined as partial cure. The patients who showed a partial cure after the first round of therapy received a second cycle of the same treatment. The number of patients who were clinically cured of the lesion 1 month after completion of the first and second therapy was determined.

All analyses were conducted using the SPSS statistical program (Version 11.5 for Windows; SPSS, Chicago, IL). The age, gender, number of lesions, lesion localization (head-neck, upper extremity, lower extremity, trunk, mucosal or generalized), lesion type (ulcer, papule, nodule and recidivans), duration of lesions (<6 weeks, 6–12 weeks,>12 weeks) and lesion dimension (<5 cm, >5 cm) for all the patients were evaluated with the Chi-square test. Pre- and post-treatment number of lesions, lesion localization, lesion type, duration of lesions,

lesion dimension for the first and second cycles of intralesional MA treatment and intralesional SSG treatment were evaluated with the paired sample *t* test. A value of $p \leq 0.005$ was considered statistically significant.

Results

Of 3456 CL patients that were included in this study, 1487 (43%) were males and 1969 (57%) were females. 712 (45%) in the MA treatment group were males and 1016 (55%) were females with a mean age of 22.57 ± 17.76 years. The SSG group comprised 775 (41%) males and 953 (59%) females with a mean age of 25.44 ± 16.57 years (Table 1).

Of 3456 patients, 3358 (97%) had single lesions which were located in the head and neck region (48%), the lower extremities (35%), upper extremities (10%), abdomen (3%) and around the oral cavity affecting the mucosa (3%) (Table 1). The most common type of lesion was the ulcerated type (55%) with lesion diameter generally below 5 cm. The duration of disease was more than 6 weeks in the majority (88%) of the patients when they first visited the clinic (Table 1).

Efficacy of intralesional MA and intralesional SSG in the treatment of CL

The results of CL patients after receiving the first course of intralesional MA and intralesional SSG treatment are shown in Tables 2 and 3. One month after completion of therapy, 1431 of 1728 patients (82%) in the MA group showed total healing of their lesions compared to 1157 of 1728 patients (67%) in the SSG group. The difference between these two treatments was statistically significant ($p<0.005$). Intralesional MA treatment was significantly more effective than intralesional SSG regardless of gender, duration of lesion, type of lesion (ulcer, papule, nodule and recidivans) or lesion size ($p<0.005$) (Table 3). When examined in terms of lesion site, MA was found to be more effective than SSG only in the lesions of the upper and lower extremities ($p<0.005$) (Table 3).

Efficacy of second cycle treatment of intralesional MA and intralesional SSG

The results of CL patients who received second intralesional MA or SSG treatments are shown in Table 4. Of 297 patients, 237 (80%) in the MA group compared to only 407 of 561 patients (72%) in the SSG group responded to second round of the treatment. The difference between these two treatments was statistically significant ($p<0.005$). Interestingly, intralesional MA was significantly more effective than SSG in females, as well as in smaller lesions (less than 5 cm) which were papule, nodule and recidivans subtypes and located in the head-neck region or upper extremities ($p<0.05$).

Discussion

CL is a major public health problem and often causes disfiguring scars, and mucosal spread if left untreated. Several forms of treatment have been in use for CL with varying degrees of efficacy, and no well-standardized treatments for localized CL are as yet available. Commonly used therapeutic approaches to CL include oral administration using azoles, azithromycin, pentamidine, miltefosine and zinc sulphate, which are not always effective (5). Topical administration of paromomycin, imiquimod and amphotericin B has also been

used. Cryotherapy (15) and heat therapy (16) in the treatment of CL will require more studies to determine their long-term efficacy. The current recommended treatment for CL involving the systemic administration of antimonials present a number of side effects including hepatotoxicity and nephrotoxicity, leading to poor patient compliance (17). Consequently, intralesional administration of antimonials, which eliminates side effects associated with systemic treatment, has gained wide acceptance and has proven to be effective in CL management (18). Furthermore, WHO has acknowledged the efficacy of intralesional treatment and has recommended its therapy for use (4).

Numerous studies demonstrate the efficacy of intralesional MA and SSG for therapy against CL (17–33). Previous reports using intralesional SSG therapy showed cure rates which ranged between 58.3 and 94.6% (17,19–22). In our study, the efficacy of intralesional SSG was 67.53%, which is well within the range of these other studies. However, as reported by Solomon et al. (20), pain associated with intralesional SSG injection is a common side effect, leading to poor patient compliance. Further, animal studies evaluating MA and SSG treatments of experimental CL demonstrate increased toxicity and localized inflammation in SSG-treated hamsters (7).

Successful treatment rates reported for MA ranged from as high as 97.2% (33) to as low as 41.7% (23). In other studies, cure rates have varied between 50 and 92% (18,24–32). In our current study, successful treatment rate of intralesional MA in CL patients was determined to be 82.8%, which is similar to the study conducted by Vasconcellos et al. (32) (83%). Differences between our results and those with lower cure rates for MA (18,25,30) can be explained by various host factors such as genetic background and nutritional status which can influence response to treatment. Alternatively, different strains of *Leishmania* isolated from different geographical regions may display differential susceptibility to MA. Indeed, in a study conducted in a similar geographical location as in our study, cure rates for intralesional MA treatment of CL was as high as 97.2% (33).

In this study, a comparison of the clinical efficacies of intralesional MA and SSG in CL patients within the same geographical region of Sanliurfa, Turkey, revealed a significant advantage for the use of intralesional MA. Other reports show similar efficacies for systemic administration of MA and SSG in CL patients caused by *Leishmania braziliensis* in an endemic region of Brazil (34). However, to the best of our knowledge, this is the first report demonstrating the improved efficacy of MA over SSG in intralesional CL treatment.

In the evaluation of patients receiving a second cycle of treatment with intralesional pentavalent antimonials when there had been no recovery in the first cycle, MA was also more effective than SSG. This was also true for all clinical forms of CL lesions, as well as in lesions localized in the head-neck, upper extremities and generalized areas of the body. Our study therefore indicates an overall enhanced clinical efficacy of MA over SSG in intralesional CL treatments in this endemic region of Turkey.

In conclusion, our findings demonstrate that in this endemic region, intralesional MA is more effective than SSG in the treatment of CL, generally caused by *L. major* and *L. tropica*. We therefore propose the use of intralesional MA for the treatment of CL.

References

1. Alvar J, Velez ID, Bern C, et al. Leishmaniasis worldwide and global estimates of its incidence. PLoS One. 2012; 7:e35671. [PubMed: 22693548]
2. Munir A, Janjua SA, Hussain I. Clinical efficacy of intramuscular meglumine antimoniate alone and in combination with intralesional meglumine antimoniate in the treatment of old world cutaneous leishmaniasis. Acta Dermatovenerol Croat. 2008; 16:60–4. [PubMed: 18541100]
3. Neves LO, Talhari AC, Gadelha EP, et al. A randomized clinical trial comparing meglumine antimoniate, pentamidine and amphotericin B for the treatment of cutaneous leishmaniasis by *Leishmania guyanensis*. An Bras Dermatol. 2011; 86:1092–101. [PubMed: 22281895]
4. World Health Organization. Control of the leishmaniases. 2010:xii–xiii. 1–186. back cover. World Health Organ Tech Rep Ser
5. Gurei MS, Tatli N, Ozbilge H, et al. Efficacy of cryotherapy and intralesional pentostam in treatment of cutaneous leishmaniasis. J Egypt Soc Parasitol. 2000; 30:169–76. [PubMed: 10786028]
6. Masmoudi A, Hariz W, Marrekchi S, et al. Old World cutaneous leishmaniasis: diagnosis and treatment. J Dermatol Case Rep. 2013; 7:31–41. [PubMed: 23858338]
7. Henao HH, Osorio Y, Saravia NG, et al. Efficacy and toxicity of pentavalent antimonials (Glucantime and Pentostam) in an American cutaneous leishmaniasis animal model: luminometry application. Biomedica. 2004; 24:393–402. [PubMed: 15678803]
8. Rezaei Riabi T, Sharifi I, Miramin Mohammadi A, et al. Evaluation of a possible synergistic effect of meglumine antimoniate with paromomycin, miltefosine or allopurinol on in vitro susceptibility of *Leishmania tropica* resistant isolate. Iran J Parasitol. 2013; 8:396–401. [PubMed: 24454432]
9. Mohammadzadeh M, Behnaz F, Golshan Z. Efficacy of glucantime for treatment of cutaneous leishmaniasis in Central Iran. J Infect Public Health. 2013; 6:120–4. [PubMed: 23537825]
10. Blum JA, Hatz CF. Treatment of cutaneous leishmaniasis in travelers 2009. J Travel Med. 2009; 16:123–31. [PubMed: 19335813]
11. Khatami A, Firooz A, Gorouhi F, et al. Treatment of acute Old World cutaneous leishmaniasis: a systematic review of the randomized controlled trials. J Am Acad Dermatol. 2007; 57:335.e1–29. [PubMed: 17337090]
12. Perez-Ayala A, Norman F, Perez-Molina JA, et al. Imported leishmaniasis: a heterogeneous group of diseases. J Travel Med. 2009; 16:395–401. [PubMed: 19930379]
13. Khatami A, Talaee R, Rahshenas M, et al. Dressings combined with injection of meglumine antimoniate in the treatment of cutaneous leishmaniasis: a randomized controlled clinical trial. PLoS One. 2013; 8:e66123. [PubMed: 23826087]
14. Soto J, Valda-Rodriquez L, Toledo J, et al. Comparison of generic to branded pentavalent antimony for treatment of new world cutaneous leishmaniasis. Am J Trop Med Hyg. 2004; 71:577–81. [PubMed: 15569787]
15. Panagiotopoulos A, Stavropoulos PG, Hasapi V, et al. Treatment of cutaneous leishmaniasis with cryosurgery. Int J Dermatol. 2005; 44:749–52. [PubMed: 16135144]
16. Reithinger R, Mohsen M, Wahid M, et al. Efficacy of thermotherapy to treat cutaneous leishmaniasis caused by *Leishmania tropica* in Kabul, Afghanistan: a randomized, controlled trial. Clin Infect Dis. 2005; 40:1148–55. [PubMed: 15791515]
17. Bumb RA, Mehta RD, Ghiya BC, et al. Efficacy of short-duration (twice weekly) intralesional sodium stibogluconate in treatment of cutaneous Leishmaniasis in India. Br J Dermatol. 2010; 163:854–8. [PubMed: 20500797]
18. Asilian A, Sadeghinia A, Faghihi G, et al. Comparative study of the efficacy of combined cryotherapy and intralesional meglumine antimoniate (Glucantime) vs. cryotherapy and intralesional meglumine antimoniate (Glucantime) alone for the treatment of cutaneous leishmaniasis. Int J Dermatol. 2004; 43:281–3. [PubMed: 15090013]
19. El-Sayed M, Anwar AE. Intralesional sodium stibogluconate alone or its combination with either intramuscular sodium stibogluconate or oral ketoconazole in the treatment of localized cutaneous leishmaniasis: a comparative study. J Eur Acad Dermatol Venereol. 2010; 24:335–40. [PubMed: 19744259]

20. Solomon M, Baum S, Barzilai A, et al. Treatment of cutaneous leishmaniasis with intralesional sodium stibogluconate. J Eur Acad Dermatol Venereol. 2009; 23:1189–92. [PubMed: 19298486]
21. Sharquie KE, Al-Talib KK, Chu AC. Intralesional therapy of cutaneous leishmaniasis with sodium stibogluconate antimony. Br J Dermatol. 1988; 119:53–7. [PubMed: 2841964]
22. Bumb RA, Prasad N, Khandelwal K, et al. Long-term efficacy of single-dose radiofrequency-induced heat therapy vs. intralesional antimonials for cutaneous leishmaniasis in India. Br J Dermatol. 2013; 168:1114–19. [PubMed: 23298394]
23. Faghihi G, Tavakoli-kia R. Treatment of cutaneous leishmaniasis with either topical paromomycin or intralesional meglumine antimoniate. Clin Exp Dermatol. 2003; 28:13–16.
24. Alkhawajah AM, Larbi E, al-Gindan Y, et al. Treatment of cutaneous leishmaniasis with antimony: intramuscular versus intralesional administration. Ann Trop Med Parasitol. 1997; 91:899–905. [PubMed: 9579209]
25. Asilian A, Sadeghinia A, Faghihi G, et al. The efficacy of treatment with intralesional meglumine antimoniate alone, compared with that of cryotherapy combined with the meglumine antimoniate or intralesional sodium stibogluconate, in the treatment of cutaneous leishmaniasis. Ann Trop Med Parasitol. 2003; 97:493–8. [PubMed: 12930612]
26. Desjeux P, Mollinedo S, Le Pont F, et al. Cutaneous leishmaniasis in Bolivia. A study of 185 human cases from Alto Beni (La Paz Department). Isolation and isoenzyme characterization of 26 strains of *Leishmania braziliensis braziliensis* [corrected]. Trans R Soc Trop Med Hyg. 1987; 81:742–6. [PubMed: 3449990]
27. Firooz A, Khatami A, Khamesipour A, et al. Intralesional injection of 2% zinc sulfate solution in the treatment of acute old world cutaneous leishmaniasis: a randomized, double-blind, controlled clinical trial. J Drugs Dermatol. 2005; 4:73–9. [PubMed: 15696988]
28. Mujtaba G, Khalid M. Weekly vs. fortnightly intralesional meglumine antimoniate in cutaneous leishmaniasis. Int J Dermatol. 1999; 38:607–9. [PubMed: 10487452]
29. Oliveira-Neto MP, Schubach A, Mattos M, et al. Intralesional therapy of American cutaneous leishmaniasis with pentavalent antimony in Rio de Janeiro, Brazil – an area of Leishmania (V.) braziliensis transmission. Int J Dermatol. 1997; 36:463–8. [PubMed: 9248897]
30. Sadeghian G, Nilfroushzadeh MA, Iraji F. Efficacy of local heat therapy by radiofrequency in the treatment of cutaneous leishmaniasis, compared with intralesional injection of meglumine antimoniate. Clin Exp Dermatol. 2007; 32:371–4. [PubMed: 17376205]
31. Salmanpour R, Razmavar MR, Abtahi N. Comparison of intralesional meglumine antimoniate, cryotherapy and their combination in the treatment of cutaneous leishmaniasis. Int J Dermatol. 2006; 45:1115–16. [PubMed: 16961529]
32. Vasconcellos Ede C, Pimentel MI, Schubach Ade O, et al. Intralesional meglumine antimoniate for treatment of cutaneous leishmaniasis patients with contraindication to systemic therapy from Rio de Janeiro (2000 to 2006). Am J Trop Med Hyg. 2012; 87:257–60. [PubMed: 22855754]
33. Uzun S, Durdu M, Culha G, et al. Clinical features, epidemiology, and efficacy and safety of intralesional antimony treatment of cutaneous leishmaniasis: recent experience in Turkey. J Parasitol. 2004; 90:853–9. [PubMed: 15357081]
34. Saldanha AC, Romero GA, Merchan-Hamann E, et al. A comparative study between sodium stibogluconate BP 88R and meglumine antimoniate in the treatment of cutaneous leishmaniasis. I. The efficacy and safety. Rev Soc Bras Med Trop. 1999; 32:383–7. [PubMed: 10495667]

Table 1

Demographic and clinical characteristics of patients with cutaneous leishmaniasis and their responses to intralesional pentavalent antimonials (SSG and MA).

	IL-MA (*n* = 1728)	IL-SS (*n* =1728)
Age (years)	22.57 ± 17.76	25.44 ± 16.57
Sex		
Male	712 (44.8%)	775 (41.2%)
Female	1016 (55.2%)	953 (58.8%)
Duration (weeks)		
<6	172 (10.0%)	227 (13.3%)
6–12	934 (54.1%)	762 (44.5%)
>12	618 (35.8%)	722 (42.1%)
Size (cm)		
<5	1682 (97.5%)	1694 (98.8%)
>5	43 (2.5%)	20 (1.2%)
Type		
Ulcer	811 (47.1%)	1080 (63.2%)
Papule	134 (7.8%)	106 (6.2%)
Nodule	758 (44.0%)	475 (27.8%)
Recidivans	19 (1.1%)	47 (2.8%)
Location		
Head-neck	871 (50.4%)	794 (46.3%)
Upper extremity	158 (9.1%)	189 (11.0%)
Lower extremity	625 (36.2%)	600 (35.0%)
Trunk	13 (4%)	5 (3%)
Mucosal	45 (2.6%)	49 (2.9%)
Generalized	23 (1.3%)	75 (4.4%)

IL-MA: intralesional meglumine antimoniate; IL-SS: intralesional sodium stibogluconate.

Table 2

Comparison of first and second cycles of treatment of intralesional MA and SSG.

Drug	First treatment		Second treatment Responders to treatment
	Responders to treatment	Non-responders to treatment	
IL-MA, $n = 1728$	1431/1728 (82%) *	297/1728 (18%)	233/297 (78%) *
IL-SSG, $n = 1728$	1157/1728 (68%)	561/1728 (32%)	417/561 (73%)

IL-MA: intralesional meglumine antimoniate; IL-SS: intralesional sodium stibogluconate.

* Denotes statistically significant difference ($p<0.05$) between MA and SSG groups.

Table 3

Comparison of the effects of first cycle intralesional MA and SSG on their responses to treatments according to patient's sex, duration, size and type of the lesions.

	IL-MA		IL-SS	
	Responders	**Non-responders**	**Responders**	**Non-responders**
Sex				
Male	587/712 (82%)	125/712 (18%)	516/775 (66%)	259/775 (34%)
Female	844/1016 (83%)	172/1016 (17%)	651/953 (68%)	302/953 (32%)
Duration (weeks)				
<6	120/172 (70%)	52/172 (30%)	117/227 (51%)	110/227 (49%)
6–12	767/934 (82%)	167/934 (18%)	512/762 (67%)	250/762 (33%)
>12	540/618 (87%)	78/618 (13%)	526/722 (73%)	196/722 (27%)
Size (cm)				
<5	1388/1682 (82%)	294/1682 (18%)	1145/1694 (67%)	549/1694 (33%)
>5	40/43 (93%)	3/43 (7%)	13/20 (65%)	7/20 (35%)
Type				
Ulcer	715/811 (88%)	96/811 (12%)	792/1080 (73%)	288/1080 (27%)
Papule	97/134 (72%)	37/134 (28%)	47/106 (44%)	59/106 (56%)
Nodule	599/758 (79%)	159/758 (21%)	285/475 (60%)	190/475 (40%)
Recidivans	15/19 (79%)	4/19 (21%)	28/47 (59%)	19/47 (41%)
Location				
Head-neck	118/205 (58%)	87 (42%)	794 (%)	319 (%)
Upper extremity	142/158 (90%)	16 (10%)	189 (%)	58 (%)
Lower extremity	560/625 (90%)	65 (10%)	600 (%)	149 (%)
Trunk	1/2 (50%)	1/2 (50%)	0/5 (0%)	5 (100%)
Mucosal	37/45 (82%)	8/45 (18%)	40/49 (82%)	9/49 (18%)
Generalized	7/23 (30%)	16/23 (70%)	59/75 (79%)	16/75 (21%)

IL-MA: intralesional meglumine antimoniate; IL-SS: intralesional sodium stibogluconate.

Table 4

Comparison of the effects of second cycle intralesional MA and SSG on their responses to treatments according to patient's sex, duration, size and type of the lesions.

	IL-MA		IL-SS	
	Responders	**Non-responders**	**Responders**	**Non-responders**
Sex				
Male	96/125 (77%)	29/125 (23%)	195/259 (75%)	64/259 (25%)
Female	141/172 (82%)	31/172 (18%)	212/302 (70%)	90/302 (30%)
Duration (weeks)				
<6	42/52 (81%)	10/52 (19%)	64/110 (58%)	46/110 (42%)
6–12	130/167 (78%)	37/167 (22%)	184/250 (74%)	66/250 (26%)
>12	65/78 (83%)	13/78 (17%)	155/196 (79%)	41/196 (21%)
Size (cm)				
<5	234/294 (80%)	60/294 (20%)	399/549 (73%)	150/549 (27%)
>5	3/3 (100%)	0/3 (0%)	4/7 (57%)	3/7 (43%)
Type				
Ulcer	83/96 (86%)	13/96 (14%)	242/288 (84%)	46/288 (16%)
Papule	25/37 (68%)	12/37 (32%)	24/59 (41%)	35/59 (59%)
Nodule	124/159 (78%)	35/159 (22%)	126/190 (66%)	64/190 (34%)
Recidivans	4/4 (100%)	0/4 (0%)	12/19 (63%)	7/19 (37%)
Location				
Head-neck	158/205 (77%)	47/205 (23%)	211/319 (66%)	108/319 (34%)
Upper extremity	16/16 (100%)	0/16 (0%)	51/58 (88%)	7/58 (12%)
Lower extremity	55/65 (85%)	10/55 (15%)	117/149 (79%)	32/149 (21%)
Trunk	2/2 (100%)	0/2 (0%)	5/5 (100%)	0/5 (0%)
Mucosal	5/8 (63%)	3/8 (37%)	6/9 (66%)	3/9 (34%)
Generalized	16/16 (100%)	0/16 (0%)	13/16 (81%)	3/16 (19%)

IL-MA: intralesional meglumine antimoniate; IL-SS: intralesional sodium stibogluconate.