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Abdominal Ultrasound Findings Associated with Canine Visceral Leishmaniasis in Endemic Areas

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Abstract

The aim of the study is to describe the ultrasound (US) findings associated to canine leishmaniasis (CVL) in dogs from an endemic area.

Thirty-four dogs naturally infected by Leishmania Infantum were enrolled. Morphologic changes of abdominal parenchymatous organs, including size, change in echogenicity and echo-texture and the presence of focal or diffused lesions were recorded. When possible the return to normal of US changes after treatment were monitored, in order to further confirm the association with the disease.

The most common pathological findings were: spleen from mild to severely enlarged, hyperechogenicity of renal cortex and hepatomegaly. Interesting findings were: the honey-comb splenic parenchymal pattern (2 dogs), the cirrhotic/fibrotic aspect of the liver (2 dogs) and the abdominal lymphadenopathy in absence of peripheral lymphadenopathy (2 dogs). Focal macro nodular lesions in spleen and liver, as described in some human cases, were not registered.

Results of this study suggest the ultrasonography could contribute to define the systemic involvement of dogs with leishmaniasis and to monitor the response to treatment. Canine leishmnaiasis need to be included in the differential diagnosis of the US honey-comb appearance of the spleen.

INTRODUCTION

Canine leishmaniasis (CVL) by *Leishmania infantum* is a vector borne protozoal disease widely distributed in many Mediterranean countries. In endemic areas a high percentage of clinically healthy infected dogs have been registered [1,2] serving as reservoirs for the transmission of *Leishmania* spp. to receptive animals and humans. The diagnosis of CVL in pauci-symptomatic animals may be troublesome because of the vague and nonspecific clinical signs and the complexity of interpretation of specific tests.

In human medicine the abdominal ultrasound (US) findings associated with visceral leishmaniasis has been reported [3-5]. Moreover, single or multiple macronodular focal lesions in liver and spleen have been described in single case reports [6-8]. Differently, few are actually available on abdominal ultrasonographic findings registered in CVL. The aim of this study was to describe and discuss the US abdominal findings associated with CVL and, when possible, to monitor the reverse to normal of US abnormalities after specific treatment. Furthermore, the correlation between the US pathological changes with an assigned clinical score and with selected laboratory tests has been investigated.

MATERIALS AND METHODS

Study design

Dogs of different age, sex and breed with a diagnosis of *L. infantum* infection achieved by amastigotes detection on lymph-nodal smears at microscopy and positivity to IFAT test were selected for the study. All the dogs came from and lived in the same region (Apulia, Italy). A clinical examination was performed on each dog and blood and urine samples were collected for routine laboratory exams at presentation (HCT%, Urea, Creatinine, Albumine, Total Proteins, Albumin/Globulin ratio, urinalysis). Animals with evidence of concomitant diseases were excluded. In particular a negative test for other vector borne diseases (SNAP rapid ELISA Kit multi tests, Idexx, Belgium) was required as inclusion criteria. Animal data (breed, sex, age, and weight), presenting complaint and history were recorded on an individual form. The animals were kept under their usual housing conditions before, during and after the study. The owner gave written consent for his/her animal(s) to participate in the study. Each dog was assigned to a clinical set based on the following criteria: A= absence of clinical signs suggestive of leishmaniasis; B= vague and nonspecific clinical signs, fairly suggestive of leishmaniasis; C= clinical signs highly suggestive of leishmaniasis.

Dogs were treated with a standardised treatment protocol for CVL: meglumine antimoniate (Glucantime, Merial) 50mg/kg/bid subcutaneously for 28 days and allopurinol 10 mg/kg/bid per so for 6 months or allopurinol in monotherapy for 6 months in case of severe disease with evidence of organ impairment [9,10]. Abdominal ultrasonography was performed at the beginning of the study in all dogs (T0= diagnosis). In dogs that showed US

pathological signs, the US abdominal examination was repeated at the end of treatment with meglumine antimoniate (T1) or after allopurinol discontinuation (T2).

Microscopic diagnosis

Popliteal lymphnode tissues were sampled using a non-aspiration technique [11]. Smears were prepared and stained using May-Grunwald-Giemsa and examined under light microscope. The cytological examination was considered positive if *Leishmania* spp. amastigotes (1.5 - 2.0 x 2.5 - 5 µm) were revealed (Figure 1). In some dogs other tissues samples (spleen, liver, abdominal fluid) were also collected using an US guided fine needle aspiration technique and microscopically examined.

Ultrasonography

An Esaote Mylab 30 Gold ultrasonic diagnostic apparatus (Medmark-Esaote, Italia) equipped with a curvilinear 5-8 MHz and an 8-12 MHz linear transducer was used for abdominal examinations. A conventional B mode US study of parenchymatous organs was performed. Morphologic changes of parenchymatous organs, including changes in organ size and shape, echogenicity and/or echostructure were recorded. US criteria were used to define US pathological changes as reported in Nyland and Mattoon [12]. In particular, splenomegaly was defined on the basis of rounded edges and contact with a normal distended bladder; hepatomegaly was assessed on the basis of the displacement of the caudal edge of the liver, on their rounding and on displacement of the pylorus. The echogenicity was evaluated as baseline echogenicity relative to the surrounding parenchyma and echostructure was described as homogeneous or heterogeneous and evaluated for the presence of widespread parenchymal changes (diffuse disorders). The presence of single or multiple focal macro nodular lesions was investigated in liver and spleen and registered separately. Further more, mesenteric and medial iliac lymphnodes were evaluated and lymphadenopathy was assessed using previously reported parameters (short/long axis ratio >0.5, thickness >8mm, altered echopattern) [13,14]. Other pathological findings revealed at the abdominal US exam were also registered.

Statistical analysis

For statistical analysis a score was assigned to single US pathological findings ranging from -1 to 1 for liver size and liver and spleen echogenicity (-1= reduced, 0= normal, 1= increased); from 0 to 2 for spleen size (0 =normal, 1 = moderate increased volume, 2= severe increased volume), and from 0 to 1 (0= absence, 1= presence) for the following alterations: increased echogenicity of renal cortex, abdominal lymphadenopathy, diffuse altered echostructure in spleen and liver.

Data (clinical set, US score in single organs and laboratory results) were submitted to Pearson's correlation indices analysis. Data showing high correlation indices were analyzed with simple linear regression analysis. A value of P <0.05 was considered significant.

RESULTS AND DISCUSSION

Thirty four dogs were included in the study. They were 15 males and 19 females aged between 2 to 10 years. All of them underwent abdominal US examination at diagnosis. US follow-up post treatment was available in 11 dogs. Table 1 shows the dogs data, presenting complaint, clinical signs and score. The most common US abdominal pathological findings were: spleen from mild to severely enlarged (24 dogs), mostly associated with a hypoechoic or coarse hypoechoic parenchymal pattern; bilateral increased echogenicity of the renal cortex (14 dogs), hepatomegaly (7 dogs) and abdominal lymphadenopathy (Table 2). The spleen showed a honey-comb pattern in two dogs (Figure 2) and a heterogeneous structure with multiple iso/hyperechoic focal lesions in one. A small liver with irregular margins suggestive of a chronic process was revealed in two other dogs. In one of this dog the presence free fluid in abdomen was revealed. Overall, liver echogenicity appeared increased in 5 dogs (including that with cirrhosis) and decreased in 2. Focal macronodular lesions were not detected in spleen or liver of any dogs.

The kidneys showed regular margins except for two dogs in which one of the kidneys was characterized by irregular margins. Fourteen animals showed an increased echogenicity of the renal cortex and 3 dogs showed also a reduction of the cortico-medullary definition.

In five dogs the jejunal and iliac lymphnodes appeared increased in volume and showed inhomogeneous echogenicity.

Signs suggestive of acute pancreatitis were reported in one case only (i.e. pancreas enlarged, hypoechoic and slightly heterogeneous, surrounded by peripancreatic hyperecogenicity) and a thickened and edematous stomach wall in another (Table 2). Alterations in the reproductive system (i.e. prostatic hypertrophy) were detected in 6 dogs.

Results of the statistical correlation showed that among the US pathological findings the only parameter correlated with clinical score (r = -0.34668, P = 0.0446) and HCT (r = 0.37510, P = 0.0344) was the spleen echogenicity (Figure 3). No statistically significant correlations have been observed between renal US alterations and clinical-pathological data on renal function (Urea, Crea, Albumin, and HCT).

The US follow-up post treatment was available in 11 dogs (Table 3). In particular 4 dogs were monitored at T1, the others at T2. Table 4 shows post treatment follow-up results. A resolution of US changes in spleen, liver and lymphnodes was recorded in most of the dogs. In particular, the honeycomb aspect of the spleen quickly disappeared at T1 and enlarged organs reverse to normal size at T2. At T2 other abnormal findings, previously absent, were observed in 4 dogs including kidney and bladder stones, enlargement of the adrenal glands and mild abdominal effusion.

The study was conducted in an endemic area for CVL. Most of the dogs enrolled in this study showed vague and non-specific clinical signs (Table 1) or atypical signs (lameness, diarrhea) far from the most commonly reported signs of leishmaniasis (i.e. exfoliative dermatitis, onychogryphosis, periocular alopecia, peripheral lymphadenomegaly). Other dogs were presented for routine visit or were presented with signs referable to single apparatus with no evidence of systemic involvement at clinical examination. The 64.7% of dogs in this study was included in set 0 and 1. In this context, the US findings could play an important

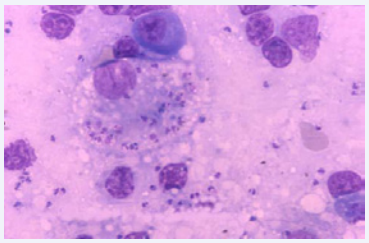

Figure 1 *Leishmania spp.* amastigotes in the lymph nodal smear stained with May-Grunwald-Giemsa, microscopic examination 100X.

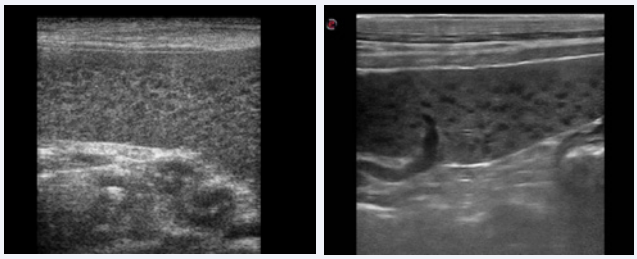

Figure 2 (a, b): The honey-comb appearance of the spleen in two dogs with leishmaniasis.

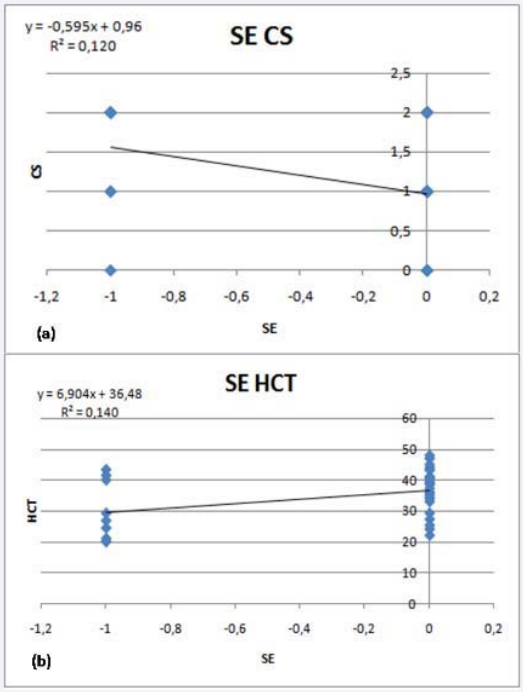

Figure 3 Correlation between Spleen Echogenicity (SE) and Clinical Score (CS) (a) and between SE and HCT% (b) in 34 dogs with leishmaniasis diagnosed by microscopic detection of amastigotes in lymph-node smears.

role. In fact, they can provide new paraclinical data that, if associated to traditional exams, could represent a useful support to address the diagnosis and to define the systemic involvement in dogs with CVL. However, the absence of US signs does not exclude the diagnosis, as shown in 20, 6% of the cases.

Results of this study showed that the most common US findings were: splenomegaly often associated with decreased echogenicity and/or inhomogeneous texture, hepatomegaly and increased echogenicity of the renal cortex. The honeycomb aspect of the spleen, the cirrhotic aspect of the liver and the abdominal lymphadenopathy (in absence of peripheral lymphadenopathy) were less common, but notable findings.

Focal macro nodular lesions in spleen and liver, as described in humans, were not registered in this study, but they could not be ruled out and a larger population needs to be examined. These findings aroused interest in human medicine because their appearance could easily resemble neoplastic lesions [6-8]. Canalias and collaborators [6] observed the presence of solid lesions with peripheral hyperechoic halo (3 and 1.5 cm of diameter) in the right lobe of the liver in an HIV positive patient. Similar macronodular lesions were observed in the liver of another patient [7], the bigger measuring 5 cm and having a necrotic center. Similarly Raeymaeckers and collaborators [8] observed an enlarged spleen with multiple hyperechoic nodules in another patient with leishmaniasis.

Statistical analysis comparing US findings with clinical set and laboratory data show that significant correlations were found only with splenic echogenicity. In particular, spleen echogenicity was negatively correlated with the clinical score and positively correlated with HCT (%), thus indicating that a reduction in the spleen echogenicity could early reflects the systemic involvement in course of CVL. The difficulty in categorizing the clinical status of *Leishmania* infected dogs is one of the major hurdles that practitioners encounter when deciding how to manage and treat affected dogs [15], thus abdominal US could contribute to better define the complex clinical picture of dogs with leishmaniasis. This is particularly important in endemic areas for the high presence of pauci-symptomatic infections. Results from US abdominal examination after treatment show that all dogs, except for one (dog 25) had the resolution of US abnormalities (Table 3), thus supporting their association with the disease but also suggesting that US monitoring could contribute to monitor the response to treatment.

Worth of note is the ultrasonographic honeycomb appearance of the spleen, characterized by multiple small hypoechoic nodules, that is suggestive of lymphoma or lymph sarcoma [16] or pyogranulomatous splenitis in dogs [16]. It was never been previously associated specifically with CVL. In this study the microscopic examination of multiple spleen fine needle aspirates revealed the presence of amastigotes of *Leishmania* spp. and the resolution of US changes after specific treatment confirmed the association with the infection (or reaction to the infection). In human medicine, Saxena and colleagues [17] observed a similar sonographic appearance of the spleen in a 7-year-old girl with leishmaniasis.

Kidney disease is often the only clinical abnormality in dogs with leishmaniasis and can evolve into asymptomatic proteinuria, nephrotic syndrome or chronic renal failure with glomerulonephritis, tubulo-interstitial nephritis and amyloidosis [18]. In our study, we found a high frequency of cortical increased echogenicity (41.2%).

Table 1: Signalament and assigned clinical score of the dogs included in the study.

Dog	Breed, sex, age, weight	Owner complaint	Most striking clinical signs	Clinical Set (0-1-2)
1.	Mongrel F10y, 26 kg	Sneezing + nasal discharge	Sero-haemorragicnasal discharge, dermatitis,	1
2.	Mongrel F5y 16 kg	Abdomen enlargement	Splenomegaly, hyperkeratosis of nose	1
3.	Pitt-bull M8y 31.5 kg	Preputial haemorragic discharge	Pawsulceration, lymphadenomegaly	1
4.	Border collie F6y 14.3 kg	Routine visit	Pallor of mucous membranae	0
5.	Boxer FS7y 29.6 kg	Annual monitoring visit for CVL recurrence*	n.n.	0
6.	Pitt-bull FS4y 22.7 kg	Annual monitoring visit for CVL recurrence *	Pallor of mucous membranae and mild lymphadenomegaly	1
7.	Mongrel M adult 20 kg	Preputial haemorragic discharge	Pallor of mucous membrane	1
8.	Great Dane F2.5y 39 kg	Anorexia	Mild lymphadenomegaly	1
9.	Boxer MC8y 32.8 kg	Routine visit	BCS 4, lymphadenomegaly	1
10.	Italian levriere FS 4y 6 kg	Anorexia, vomiting, abdominal pain	Abdominalpain	0
11.	Breton F Adult 16 kg	Epistaxis	Pallor of mucous membranae and lymphadenomegaly	2
12.	Mongrel M adult 14.7 kg	Exfoliative dermatitis	Elbow ulcerations, ear pinnae dermatitis and crusts, lymphadenomegaly	2
13.	Mongrel M adult 27.4 kg	Weight loss and exfoliative dermatitis	Pallor of mucous membranae, ulcers and depigmentation of nose, conjunctivitis	2
14.	Mongrel M adult9.1 kg	Generalized lameness and muscle hypotrophy	Depression, exfoliative dermatitis, deforming arthritis, lymphadenomegaly	2
15.	Mongrel M adult 15 kg	Alopecia and weight loss	Periocular alopecia, BCS 4	2
16.	Mongrel M12y 33.4 kg	Preputial hemorrhagic discharge	Elbow ulcerations, abnormal finding at palpation (spleen)	1
17.	Mongrel M 5y 36.9kg	Depression	Depression, lymphadenomegaly	1
18.	Cavalier king FAdult 8.25kg	Pruritus	Pallor of mucousmembranae	1
19.	Irish setter F 6y 15 kg	dysuria	n.n.	0
20.	Pointer FS young 19.8 kg	Lameness	Onychogryphosis, arthritis, lymphadenomegaly	2
21.	Mongrel F 2y, 27 kg	Routine visit	n.n.	0
22.	Mongrel M adult 27 kg	Abdominal enlargement	Ascites, weight loss	0
23.	Mongrel M 4y 12 kg	Lameness	Lymphadenomegaly, splenomegaly	2
24.	Beagle F 10y 12.1 kg	Anorexia	n.n.	0
25.	German shepherd F 9y 33.8 kg	Pre-surgery visit	Mammary nodules, head muscular atrophy	0
26.	Dobermann M 10y 21 kg	Chronic diarrhoea	BCS 3, Lymphadenomegaly, periocular alopecia	2
27.	Mongrel M 6y 22.7 kg	Mild depression, dermatitis	Eczematous dermatitis, lymphadenomegaly,	2
28.	Rottweiler Fs 9.5y 35.3 kg	Diarrhea, haematochezia, reduced food intake, lameness	Abdominal pain, lameness, pallor of mucous membranae	1
29.	MongrelFs 8y 23.3kg	Epistaxis, ricorr enturinary infection	Nasal ulcerated granuloma.	1
30.	Mongrel F adult 15 kg	Weight loss, dermatitis	BCS 4, periocular alopecia, lymphadenomegaly, skin ulcers	2
31	English setter Fs 10y, 18 kg	Mild depression, weakness	Depression, muscular hypotrophy	1
32	Mongrel M 3y 16 kg	Lameness, dermatitis	Polyarthritis, ear pinnae hypothricosis	1
33	Mongrel F 6y, 12 kg	Blindness	Exfoliative dermatitis, uveitis,	2
34	Breton, m 7y, 16 kg	Mild depression, lameness	Junction swelling, lymphadenomegaly, splenomegaly	2

* = Monitoring of dogs with a history of leishmaniasis which stopped specific treatment at least from 6 months.
Abbreviations: n.n. = none; BCS = Body Condition Score; F = Female; M = Male; y = Year; m = Months

Table 2: Frequency and percentage of dogs according to US findings at time of CVL diagnosis.

	US findings	Frequency	Percentage
	None US payhologicalfindings	7/34	20.6%
Liver	Liver enlargement (mild to moderate)	7/34	20.6%
	Increased echogenicity	3/34*	8.8%
	Decreased echogenicity	2/34	5.8%
	Fibrotic/cirrhoticaspect	2/34	5.8%
	Multiple mineralizations	1/34	2.9%
	Focallesions	0/34	0%
Spleen	Severe splenomegaly Moderate splenomegaly	8/34 16/34	23.5% 47%
	Diffuse hypoechogenicity	9/34	26.5%
	Honey-combaspect	2/34	5.8%
	Inhomogeneous aspect	7/34**	20.6%
	Focallesions	0/34	0%
Lymphnodes	Lymphadenopathy	5/34	14.7%
Kidneys	Increased echogenicity of renal cortex	14/34	41.17%
	Reduced cortex-medullary differentiation	3/34	8.8%
Other Findings	Signs of pancreatitis	1/34	2.9%
	Gastric wall thickness	1/34	2.9%
	Abdominaleffusion	1/34	2.9%

* Not including cirrhotic
** include: coarse pattern (6 dogs), presence of areas at different echogenicity (1 dog); not including spleen honey-comb appearance.

Table 3: Ultrasound monitoring after 1,5 or 6 months from the beginning of therapy in 14 dogs. The ultrasound findings at diagnosis and in follow up are reported.

ID	Time	Liver	Spleen	Kidneys	Otherfindings
Dog n 16	**T0**	n	Splenomegaly (++)	Increased echogenicity of renal cortex	n
	T 2	n	**r.n.**	p.f.	n
Dog n. 18	**T0**	Multiple mineralization	Splenomegaly (++), multiple iso-hyperecogenic lesions	n	n
	T 2	p.f	**r.n.**	n.	Surrenal glands hypo and enlarged
Dog n. 23	**T0**	n.	Splenomegaly (++) with honey-comb aspect	n.	n
	T1	n.	**r.n.**	n.	n
Dog n. 24	**T0**	n	Splenomegaly (+)	n	Lymphadenopathy
	T 2	n	**r.n**	n	**r.n**
Dog n. 25	**T0**	Fibrotic vs cirrhotic aspect+ mucocele	n	Mineralization	n
	T 2	p.f.	n	p.f.	Mild ascites
Dog n. 28	**T0**	Increased volume, inhomogeneous	Splenomegaly (++), honey-comb aspect	Increased echogenicity of cortex	n
	T 1	**r.n.**	**r.n.**	p.f.	n
Dog n. 29	**T0**	Increased volume	Splenomegaly (++), hypoechogenicity	Increased echogenicity, reduced cortico-medullary definition, mineralization	n
	T 2	**r.n.**	**r.n.**	p.f.	Bladderurolithiasis
Dog n. 30	**T0**	Colecistitis.	Splenomegaly (+), hypoechogenicity	n	n
	T 2	**r.n.**	**r.n.**	Renalmineralizations	Bladdersediment
Dog n. 31	**T0**	Increased volume	Splenomegaly (++)	Increased echogenicity of cortex	Lymphadenopathy
	T 1	**r.n.**	**r.n.**	p.f.	**r.n.**
Dog n. 32	**T0**	n	Splenomegaly (+). Inhomogeneustexiture	n	n
	T 2	n	**r.n**	n	n
Dog n. 34	**T0**		Splenomegaly(++)	n	Lymphadenopathy
	T 1		**r..n**	N	**r.n**

n= normal: no pathological ultrasound findings are revealed; r.n. = reverse to normal; p.f .= persisting US finding; in bracket is reported the severity of the pathological finding; T0= diagnosis; T 1= follow up at the end of treatment with melamine; T 2= follow up at discontinuation of allopurinol treatment (about 6 months from diagnosis).

Although dogs with CVL usually do not show clinical evidence of liver disease, the liver involvement has been already demonstrated as well as histopathological changes [19-22]. Inflammation and fibrosis of the liver are induced by CVL [20-23]; liver US changes probably reflect these histopathological changes. The cirrhotic-like evolution detected in two animals of this study could be the result of long-lasting infection as suggested also in humans [4]. In the dog with ascites *Leishmania* spp amastigotes were microscopically found both in the abdominal effusion and in the liver.

It is known that the 80% of symptomatic dogs with CVL has increased volume of peripheral lymphnodes [18]. Differently, poor information are reported in literature about the frequency of abdominal lymphadenopathy in dogs with CVL [24] and it is not known if the increase in volume of the peripheral lymphnodes is associated to abdominal lymphadenopathy. In two out of the five dogs showing abdominal lymphadenopathy, the peripheral lymphnodes resulted not enlarged. It is north worthy that in one of them, the microscopic diagnosis of *Leishmania* spp. infection was possible by sampling the enlarged abdominal lymph node under echo-guidance (dog 31).

Other US pathological findings were registered during this study in the urinary and reproductive system but it is possible that they were expression of concomitant diseases. However, systemic CVL can affect any organ including those of the reproductive system [25]. US signs of acute pancreatitis have been reported in humans in association with leishmaniasis [4].

In follow-up post treatment spleen, liver and lymph nodal US pathological signs resolved but some other findings (not revealed before therapy) were registered. Uroliths and renal mineralization may be due to the formation of xanthine uroliths resulting from xanthinuria, a condition observed in dogs undergoing prolonged treatment with allopurinol [26,27].

CONCLUSION

Results of this study suggest the ultrasonography could contribute to define the systemic involvement of dogs with leishmaniasis and to monitor the response to treatment. Furthermore data suggest that CVL need to be included in the differential diagnosis of the US honey-comb appearance of the spleen, while further investigation are need to rule out the possibility of macro nodular focal lesions associated to CVL.

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