

Renal Disease in Boxer Dogs

C.Brovida ¹, E.A.Galli ¹, L.Aresu ², R.Heiene ³

¹ ANUBI® Companion Animal Hospital, Moncalieri, Italy;

² Department of Comparative Biomedicine and Food Science, University of Padua, Italy;

³ NCVS, Oslo, Norway, and BSD, Gothenburg, Sweden.

Introduction

- Renal disease in younger dogs may be congenital (present at birth), inherited (genetically derived), familial (a trait present in a group of related dogs) or acquired.
- In young animals, renal disease is often considered congenital; however, development of advanced level renal lesions (end-stage kidney) may develop already in 60 days (Finco 1995).
- Clinical signs are typically related to CKD as well as the diagnostic procedures.

Microscopic Features of Canine Renal Dysplasia

C. A. PICUT AND R. M. LEWIS - Vet.Pathol. 24:156-163 (1987)

Department of Veterinary Pathology, New York State College of Veterinary Medicine,
Cornell University, Ithaca, NY

Abstract. Forty-five cases of renal dysplasia in dogs are examined. Microscopic lesions of dysplasia include asynchronous differentiation of nephrons, persistent mesenchyme, persistent metanephric ducts, atypical tubular epithelium, and dysontogenic metaplasia. These may be distinguished from secondary lesions including compensatory hypertrophy and hyperplasia of the nephron and a variety of degenerative and inflammatory lesions. Although morphological features of renal dysplasia in dogs differ somewhat from those in man, microscopic criteria used in the diagnosis of human dysplasia may be useful when applied to the dog.



© Dr. Claudio Brovida

Familiar nephropathies (Lees 2010)

Renal dysplasia

Lhasa Apso
Shih Tzu
Standard Poodle
Soft Coated Wheaten Terrier
Chow Chow
Alaskan Malamute
Miniature Schnauzer
Dutch Kooiker (Dutch Decoy) Dog

Polycystic Kidney Disease

Bull Terrier (autosomal dominant)
Cairn Terrier and West Highland White Terrier (autosomal recessive)

Amyloidosis

Shar-Pej
English Foxhound
Beagle

Immune-mediated Glomerulonephritis

Soft Coated Wheaten Terrier
Bernese Mountain Dog (autosomal recessive, suspected)
Brittany Spaniel (autosomal recessive)

Primary Glomerulopathies

Samoyed kindred and Navasota kindred (X-linked)
English Cocker Spaniel (autosomal recessive)
Bull Terrier (autosomal dominant)
Dalmatian (autosomal dominant)
Doberman Pinscher
Bullmastiff
Newfoundland
Rottweiler
Pembroke Welsh Corgi
Beagle

Miscellaneous

Boxer-reflux nephropathy with segmental hypoplasia

Basenji-Fanconi syndrome
German Shepherd Dog-multifocal cystadenocarcinoma (autosomal dominant)
Pembroke Welsh Corgi-telangiectasia

Introduction

- Few cases of juvenile nephropathy in boxer dogs are reported. Two single cases (Lucke et al. 1980, Peeters et al. 2000) and in two boxer dogs litter mates (Hoppe and Karlstam 2000), a third boxer puppy in this litter showed similar signs and clinico- pathological evidence of renal disease, but histopathology was not available (Hoppe and Karlstam 2000).
- Only two papers has been recently published on this topic; both are retrospective studies.

- **Juvenile nephropathy in 37 boxer dogs**

M. L. Chandler, C. Elwood, K. F. Murphy, I. Gajanayake And H. M. Syme

Journal of Small Animal Practice (2007) 48, 690–694

Objectives: The purpose of this study was to review and characterise the clinical presentation of young boxer dogs with chronic kidney disease referred to the authors' institutions.

Methods: Records were collected retrospectively from 37 boxer dogs, less than five years of age, which had presented with azotaemia, inappropriately low urine concentrating ability, and ultrasound or radiographic evidence of abnormal kidneys.

Results: Clinicopathological findings included azotaemia, hyperphosphataemia, anaemia, isosthenuria and proteinuria. Ultrasonographic findings included hyperechoic renal cortices, loss of corticomedullary junction definition, dilated pelves and irregularly shaped small kidneys. Renal histopathological findings (9 dogs) included pericapsular and interstitial fibrosis, inflammatory cell infiltration, dilated tubules, sclerotic glomeruli and dystrophic calcification. Survival time of the dogs varied from zero to over five years after diagnosis.

Clinical Significance: This paper documents features of the presentation and progression of juvenile nephropathy in boxer dogs. While juvenile nephropathy has been reported in individual cases of boxer dogs previously, this is the first reported case series.

Introduction

- **End-Stage Kidney Disease Probably due to *Reflux Nephropathy with Segmental Hypoplasia (Ask-Upmark Kidney)* in Young Boxer Dogs in Norway.**

A Retrospective Study

Ø. KOLBJØRNSSEN, M. HEGGELUND, AND J. H. JANSEN

Vet Pathol 45:467-474 (2008)

Abstract. This paper is a retrospective morphologic study of 7 young Boxer dogs , showing end-stage kidney lesions compatible with chronic pyelonephritis with severe segmental cortical atrophy and fibrosis, associated with chronic tubulointerstitial inflammation of varying degree. Azotemia was observed in 6 of the 7 cases. The gross kidney lesions were as follows: bilateral small kidneys with numerous segmental cortical scars causing depression of the renal cortical surface. Histologic examination revealed salient atrophy of nephrons, including paucity of glomeruli, glomerulocystic lesions, colloid-filled tubular microcysts, and a conspicuously increased occurrence of arteries with narrowed lumina caused by intimal thickening. These segmental abnormalities were accompanied by pronounced interstitial fibrosis. All but 1 dog showed salient tubulointerstitial lympho-plasmacytic infiltration, which in 3 cases also included diffuse infiltration of polymorphonuclear neutrophilic leukocyte (PMN)-cells and occurrence of tubular PMNcasts. Morphologic signs of abnormal metanephric differentiation (renal dysplasia) were observed in all cases in the form of atypical tubules or asynchronous nephronic development (immature glomeruli) or both. However, other morphologic primary dysplastic features were absent. **Based on the morphologic features, it is concluded that the end-stage kidney disease in these young Boxer dogs was the result of chronic atrophic non obstructive pyelonephritis, most probably caused by vesico-ureteral reflux, compatible with reflux nephropathy causing segmental hypoplasia (Ask-Upmark kidney) in man. It is proposed that atypical tubular epithelium in the form of adenomatoid proliferation of collecting duct epithelial cells should be considered an acquired compensatory lesion, rather than the result of disorganized metanephric development.**

Introduction

- **End-Stage Kidney Disease Probably due to *Reflux Nephropathy with Segmental Hypoplasia (Ask-Upmark Kidney)* in Young Boxer Dogs in Norway.**

A Retrospective Study

Ø. KOLBJØRNSSEN, M. HEGGELUND, AND J. H. JANSEN
Vet Pathol 45:467-474 (2008)

Upmark kidney.² The Ask-Upmark kidney or so-called *segmental hypoplasia* of human kidneys is characterized by unilateral or bilateral occurrence in the juvenile or adolescent human being of abnormally small kidneys with so-called segmental hypoplasia, and the presence of 1 or more grooves on the capsular surfaces. Microscopically this

The kidney lesions of the Boxers in our data contained immature glomeruli, atypical tubules and “proliferative arterioles” as primary features of renal dysplasia.¹⁴ The prevailing view is,

Vesico-ureteral reflux was not proven clinically in any of our cases, but we find that comparing the renal lesions in these dogs with descriptions of the renal lesions in reflux nephropathy in man makes a pathogenesis of reflux nephropathy most probable.

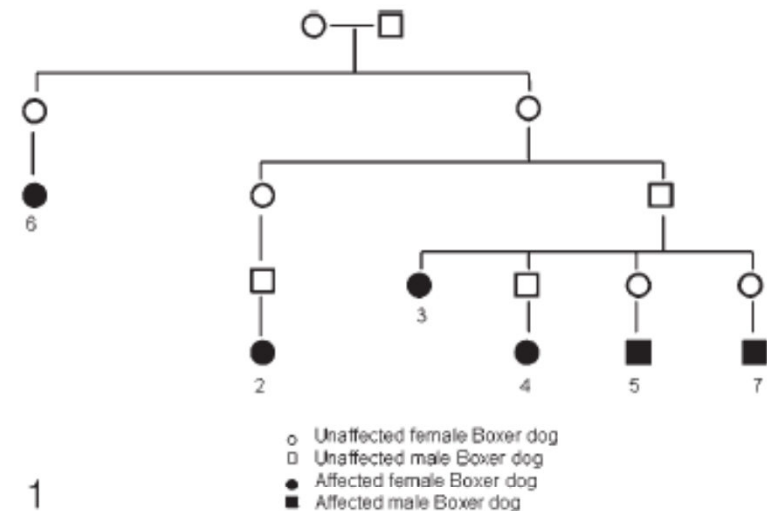
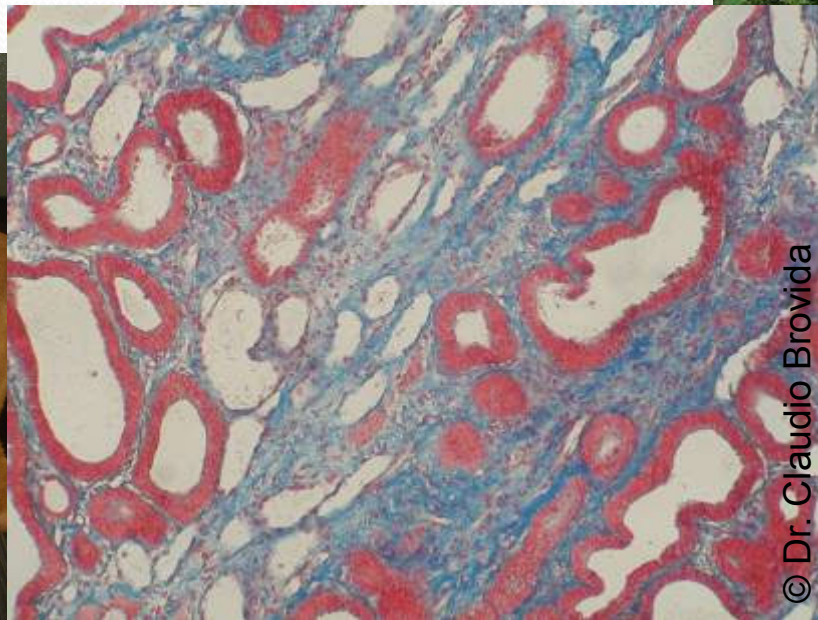


Fig. 1. The genetic relationship between 6 of the 7 Boxer dogs in the study. The 6 related dogs were all ancestors from 1 mating. Numbers refer to the dog's number in the study. Siblings are not included.

Renal Disease in Boxer dogs

We retrospectively evaluated kidney biopsies and clinical data from seventeen boxers enrolled between 2005 and 2012 with CKD stage 1 – 4.

The aims of the study were: 1) to describe the light microscopy lesions in a population of Boxers, 2) to evaluate clinical findings, and 3) to compare them to published data, on Boxers, in particular.



Renal Disease in Boxer dogs: Material and Methods

- Records were collected from dogs with age less than sixth years, with symptoms of CKD IRIS Stages 1-4, that had laboratory data - considering hematology, biochemistry, urinalysis, urine culture -, ultrasound evaluation of the kidneys, and kidney biopsy
- Hematology, biochemistry, urinalysis and culture were performed in the lab. of ANUBI® hospital
- Kidney biopsies were performed and processed (LM) in ANUBI®
- Renal biopsies were evaluated at the Department of Public Health, Comparative Pathology and Veterinary Hygiene – Anatomical Pathology Section of Padua University, by the same Pathologist (LA)
- In two dogs, EM was performed also



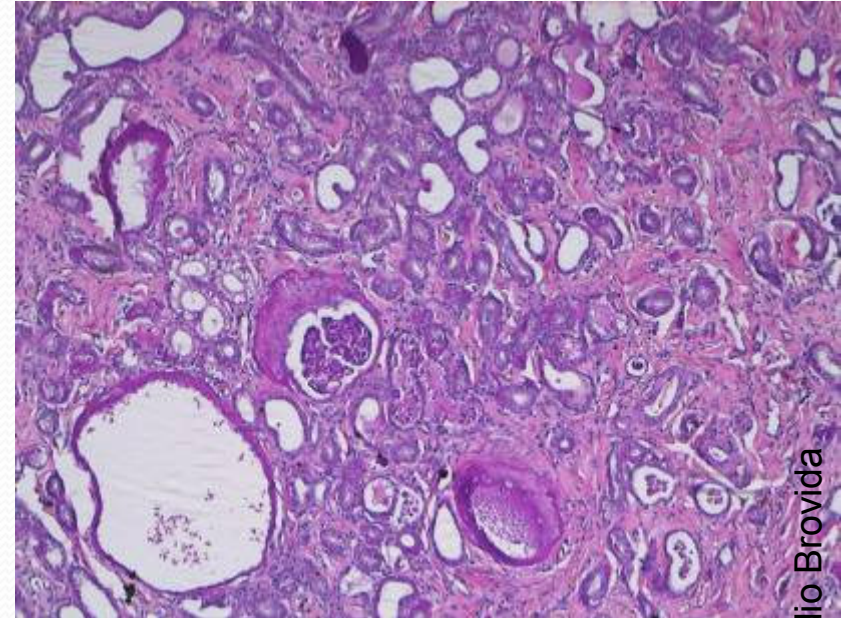
Renal Disease in Boxer dogs: Material and Methods

- The biopsies were ultrasound assisted, and performed using an 18G Tru-cut disposable biopsy needle driven by a gun, with a spring trigger system
- Two biopsies were taken from the caudal pole of the left kidney



Renal Disease in Boxer dogs: Results

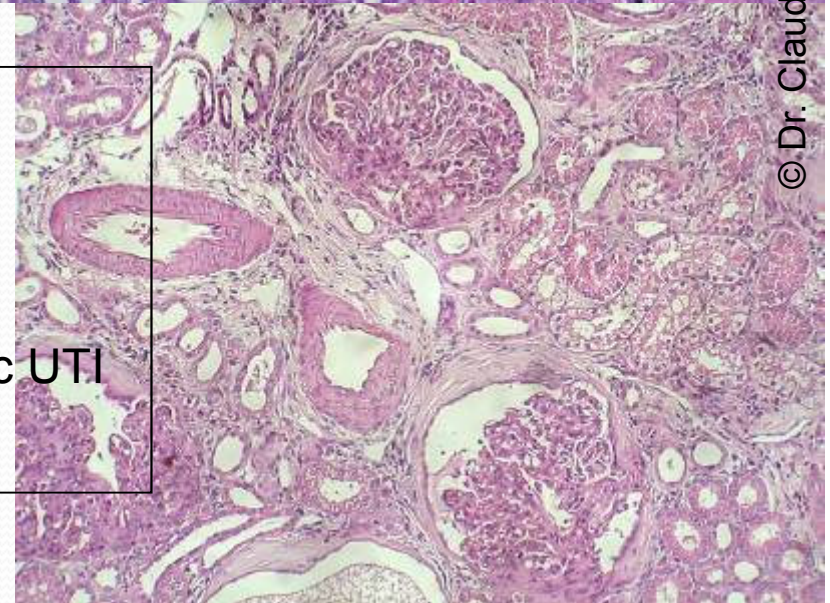
Eight Boxer Dogs: Juvenile Nephropathies



© Dr. Claudio Brovida

Nine Boxer Dogs: other diseases

- Three Leishmaniasis
- One Renal Lymphoma
- One Glomerulonephritis associated with chronic UTI
- Four non classified ESKD





...the most striking features were high number of cystic glomerular atrophy and the alteration in the morphology of the glomeruli characterized by severe podocyte hypertrophy and splitting of the Bowman's capsule with multiple synechiae...

Referto:

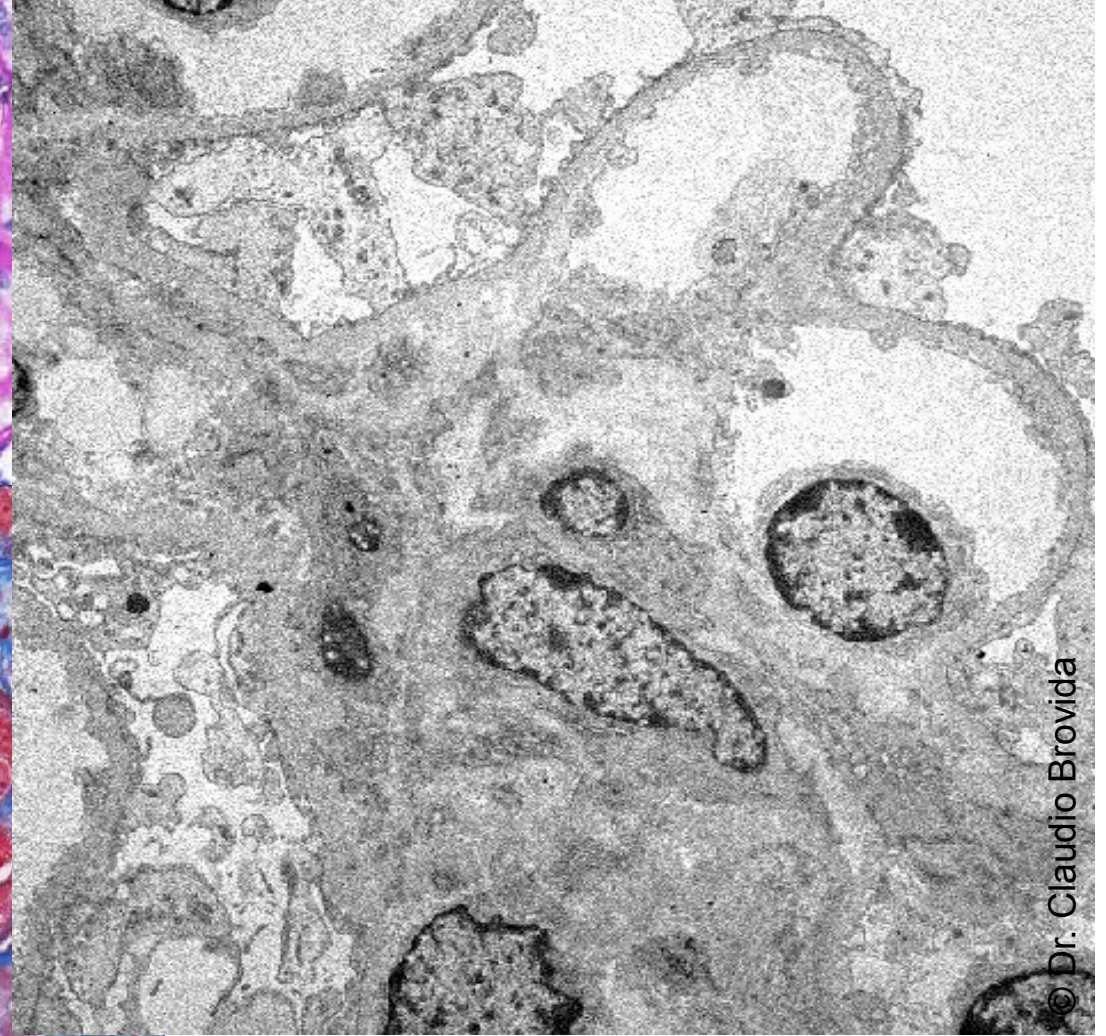
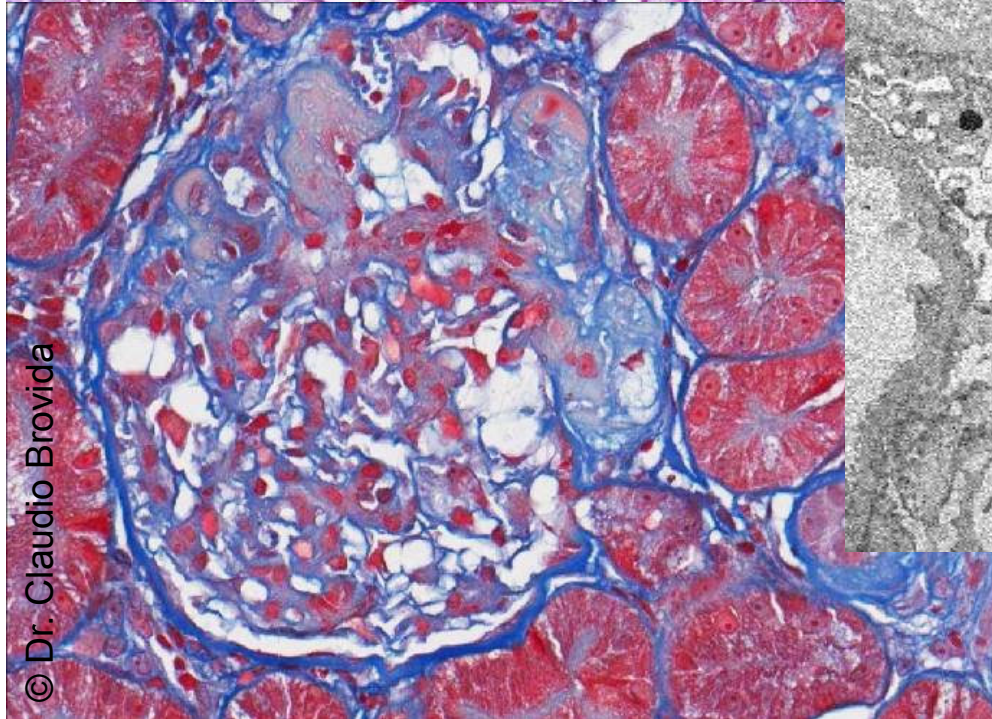
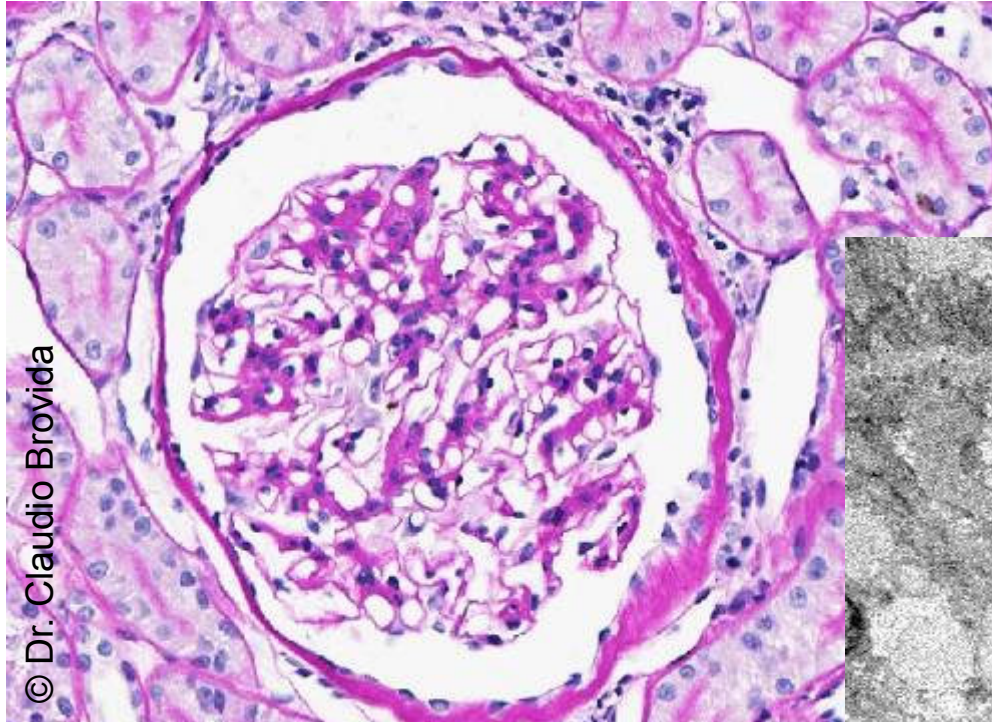
Descrizione:

Two cylindrical core biopsies of renal cortex are examined. There are approximately 16 entire glomeruli, 6 of which are obsolescent. The remaining glomeruli are characterized by varying degrees of similar lesions. Specifically, there is severe mesangial expansion which often is associated with increase in the number of mesangial cells. In general, capillary lumens are empty and endocapillary hypercellularity is not a feature. Most glomeruli contain segments in which the capillary lumens are compressed or effaced by the presence of extracellular matrix (sclerosis). These regions are often adhered to Bowman's capsule (synechiae) or have hyalinosis. Many podocytes and parietal epithelial cells are hypertrophied, and some contain cytoplasmic vacuoles and hyaline protein droplets, indicative of cellular degeneration. There is moderate periglomerular fibrosis. Afferent /efferent arterioles are occasionally characterized by thickened walls and hyalinosis (arteriolosclerosis). The interstitium is widely expanded by a proliferation of mononuclear cells. This population is a mixture of monocytes / macrophages, plasma cells, lymphocytes, megakaryocytes, myeloid progenitor cells and mature neutrophils and eosinophils.

Ultrastructural evaluation of this section of renal tissue, including two glomeruli, reveals moderate to severe glomerular, tubular and interstitial changes. Glomeruli show diffuse areas with visceral epithelial cell foot process fusion. Filtration membrane varies from unremarkable to focally extensive thickening. These areas with thickening are characterized by mesangial cell interpositioning (Figure 3, 4 and 5). Mesangium has focal areas with increased matrix and cells. Scattered visceral epithelial, mesangial and tubular epithelial cells have an increase in cytoplasmic vacuoles and residual bodies (lipofuscin granules). Scattered tubules show expanded lumen with some containing homogenous material, erythrocytes and/or cellular debris. Interstitium has multifocal areas with increased collagen. Within this substrate are scattered lymphocytes and macrophages. Furthermore, glomerular sections evaluated show no electron-dense deposits in the filtration membrane or mesangium.

Diagnosi:

Moderate to severe chronic segmental glomerulosclerosis with arteriolosclerosis, severe interstitial fibrosis and associated tubular degeneration



Renal Disease in 17 Boxer dogs: Results

Parameter	Mean	Std Dev	Std Error	C.I. of Mean	Max	Min	Median	25%	75%	Missing
Age	4,162	1,926	0,467	0,990	6,0	1,0	5,0	2,625	6,0	0
BSC	3,588	0,712	0,173	0,366	5,0	2,0	4,0	3,0	4,0	0
IRIS Stage	3,0	1,369	0,332	0,704	4,0	0,0	4,0	2,0	4,0	0
WBC (K/μL)	14,297	5,681	1,378	2,921	26,9	5,88	12,8	10,075	17,35	0
RBC (M/μL)	5,729	2,335	0,566	1,201	12,6	1,82	5,83	4,423	6,805	0
PLT (K/μL)	259,294	84,363	20,461	43,375	428,0	91,0	249,0	222,5	296,75	0
Hct (%)	34,506	14,200	3,444	7,301	76,5	11,4	33,0	26,9	41,9	0
Creatinine (mg/dl)	5,721	3,470	0,842	1,784	10,98	1,15	4,75	2,308	9,178	0
Urea (mg/dl)	235,471	155,961	37,826	80,188	480,0	31,0	230,0	83,25	397,75	0
Albumine (g/dl)	2,613	0,569	0,147	0,315	3,6	1,6	2,6	2,325	3,1	2
Tot. Prot. (g/dl)	5,697	0,679	0,170	0,362	6,7	4,77	5,625	5,085	6,38	1
ALKP (U/l)	91,214	83,175	22,229	48,024	318,0	11,0	62,0	36,0	117,0	3
Glucose (mg/dl)	106,688	15,615	3,904	8,321	151,0	81,0	104,0	96,5	115,0	1
Calcium (mmol/l)	6,280	3,966	1,024	2,196	10,4	0,63	8,1	1,232	9,575	2
Phosphorus (mg/dl)	15,311	11,882	2,971	6,332	43,71	3,82	13,735	4,865	23,305	1
Sodium (mEq/L)	148,319	3,833	0,958	2,043	156,0	144,0	146,5	145,55	151,0	1
Potassium (mEq/L)	4,552	0,835	0,209	0,445	6,52	3,19	4,480	4,2	4,81	1
Urine S.G.	1012,882	4,372	1,060	2,248	1025	1007	1014	1009,75	1015	0
pH	6,441	0,705	0,171	0,362	8,0	5,0	6,5	6,0	7,0	0
Blood stx	1,412	1,176	0,285	0,605	3,0	0,0	1,0	0,0	2,25	0
Ptot. Stx	1,882	0,993	0,241	0,510	3,0	0,0	2,0	1,0	3,0	0
Urin. Prot.	177,156	192,235	48,059	102,435	628,7	47,2	76,6	56,4	234,85	0
Urin.Creat.	80,011	70,307	17,577	37,464	318,05	5,65	71,525	41,45	96,225	0
UPC	2,925	2,736	0,664	1,407	8,85	0,14	1,58	0,957	4,553	0

Renal Disease in Boxer dogs: Results

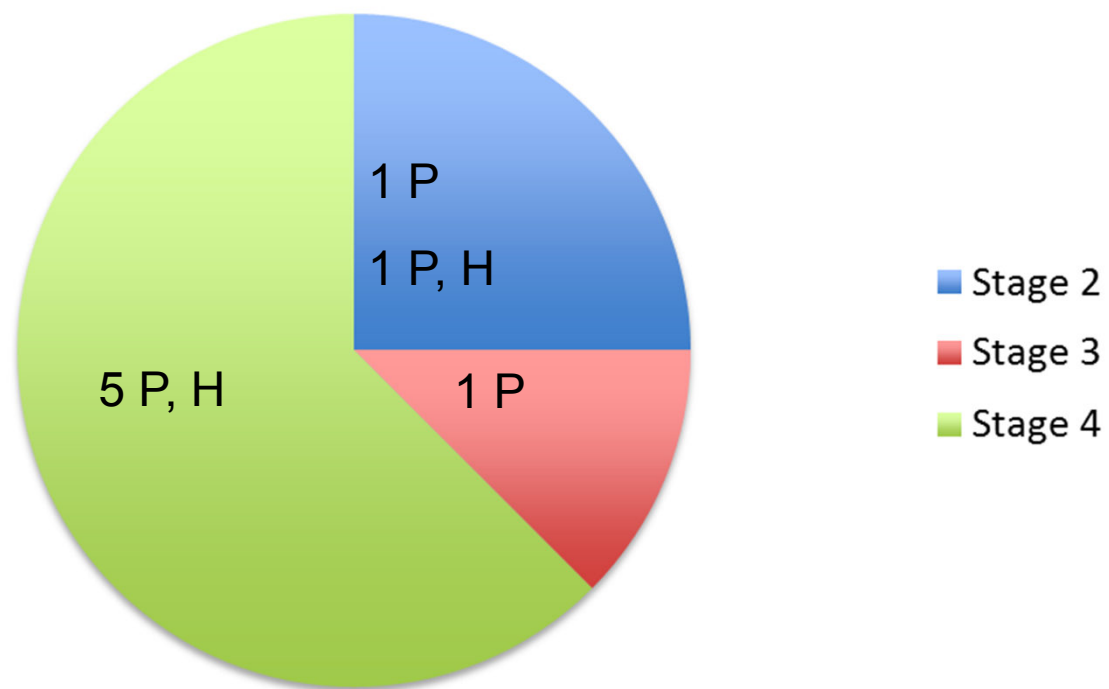
Juvenile Nephropathy Other Cause

	Mean	S.D.	Mean	S.D.
Age (1-6 yrs)	3,58±2,01		4,67±1,80	
BCS	3,37± 0,74			
IRIS Stage (1-4)	3,37± 0,91		2,66± 1,65	



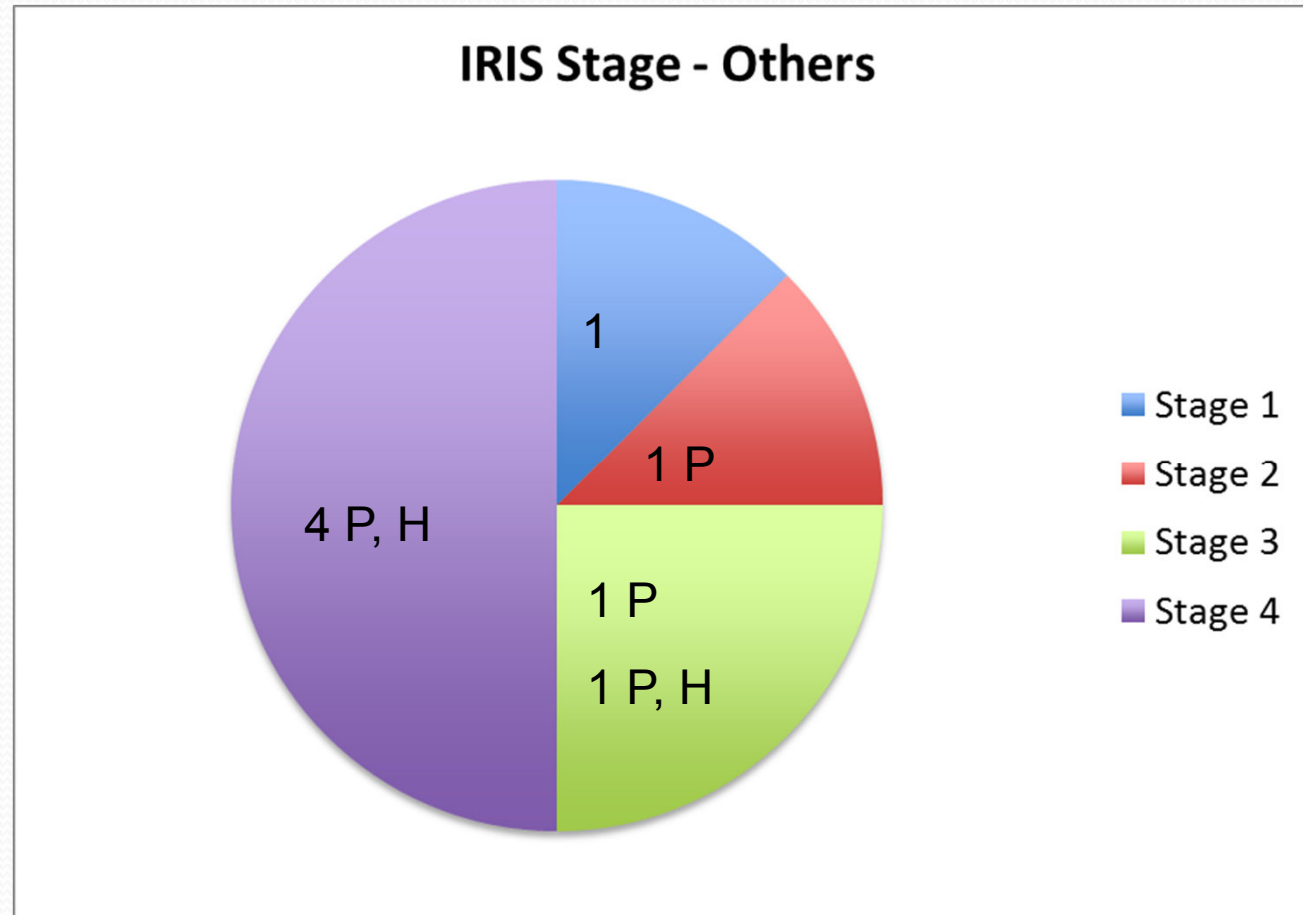
Renal Disease in Boxer dogs: Results

IRIS Stage - Juvenile Nephropathy



P= Proteinuric; H= Hypertensive (> 150mm Hg)

Renal Disease in Boxer dogs: Results

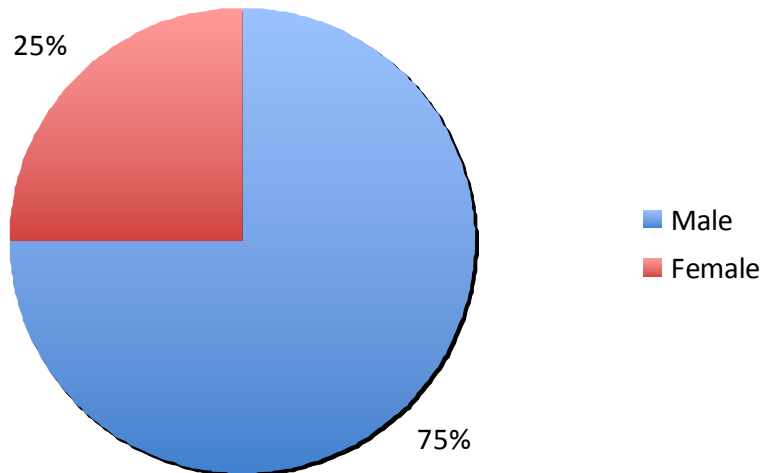


P= Proteinuric; H= Hypertensive (> 150mm Hg)

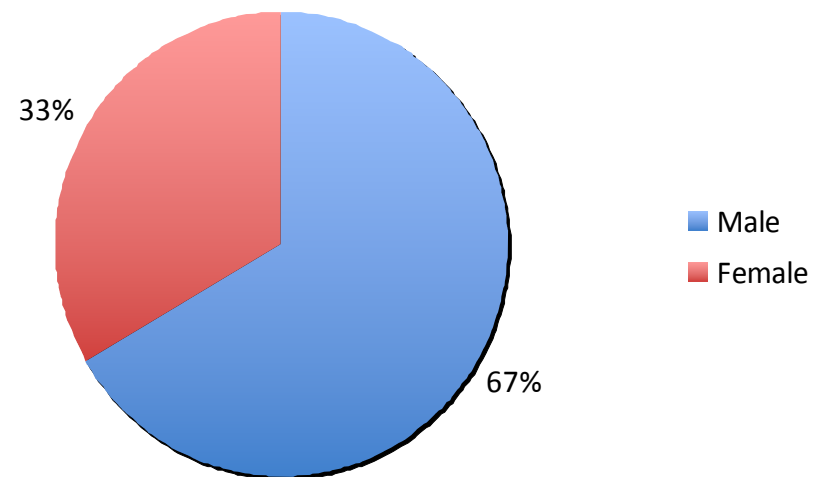
One dog did not have CKD

Renal Disease in Boxer dogs: Results

Gender - Juvenile Nephropathy



Gender - Others



Juvenile nephropathy in 37 boxer dogs

M. L. Chandler et al. *Journal of Small Animal Practice* (2007)

.....Both sexes were represented, *although there were more females than males.*

....Over half of the dogs had urinary incontinence, with the highest incidence in the neutered female dogs. This is likely to be a combination of urethral sphincter mechanism incompetence, which is the most common cause of urinary incontinence in neutered female dogs (Holt 1990), and the increased volume of urine produced secondary to the kidney disease. Boxer dogs have been noted previously to be overrepresented among dogs presenting with sphincter mechanism incompetence...

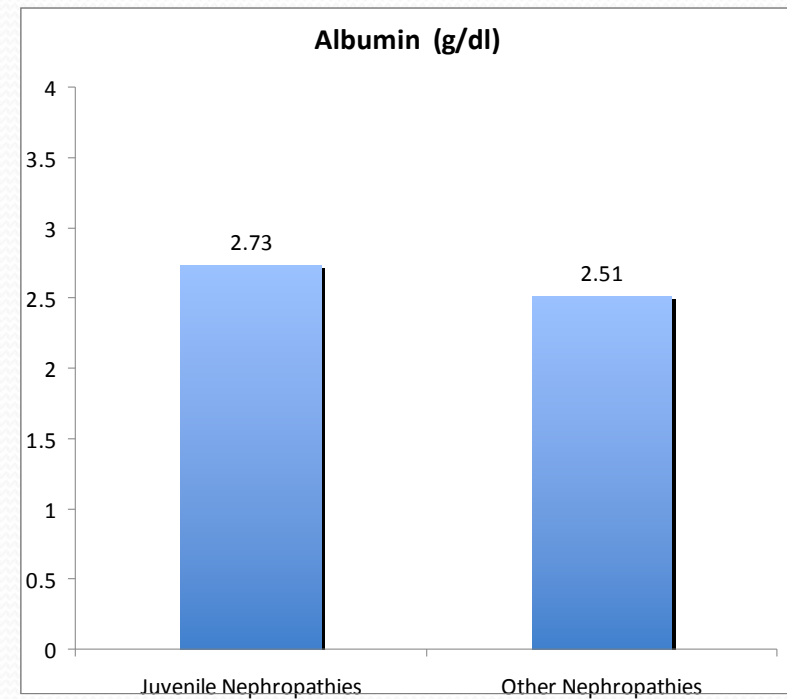
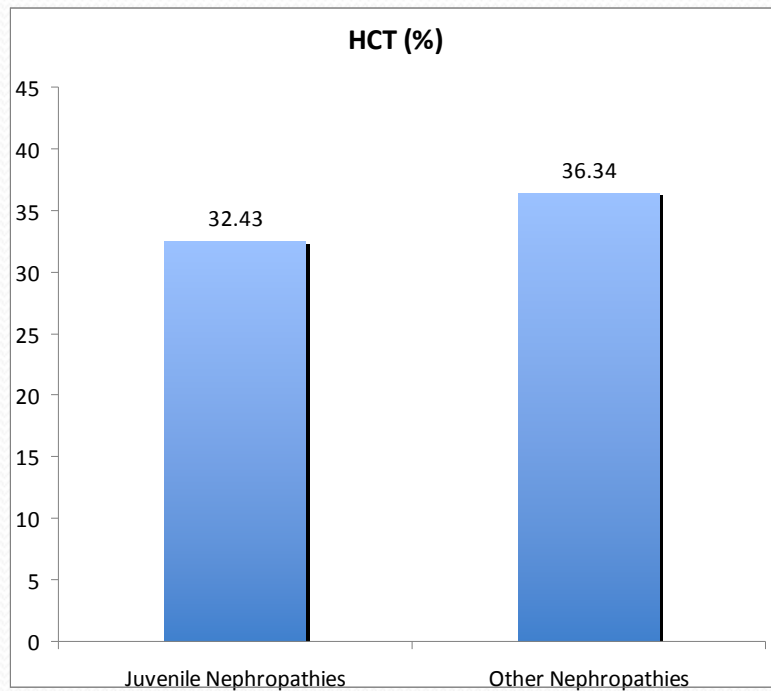
Renal Disease in Boxer dogs: Results

Parameter (time of biopsy)	Juvenile Nephropathy	Others
Hematology		
WBC (K/ μ L)	14.94 \pm 5.62	13.73 \pm 6.01
RBC (M/ μ L)	5.39 \pm 1.15	6.02 \pm 3.08
Hct (%)	32.43 \pm 7.88	36.34 \pm 18.46
Biochemistry		
Creatinine (mg/dl)	6.51 \pm 3.56	5.02 \pm 3.43
(μ mol/L)	575.49 \pm 314.7	443.77 \pm 303.21
Urea (mg/dl)	286.00 \pm 171.11	190.55 \pm 134.92
(mmol/L)	47.61 \pm 28.48	31.72 \pm 22.47
Albumin (g/dL)	2.73 \pm 0.31	2.51 \pm 0.73
(g/L)	27.3 \pm 3.1	25.1 \pm 7.3
Total Protein (g/dl)	5.52 \pm 0.75	5.83 \pm 0.62
(g/L)	55.2 \pm 7.5	58.3 \pm 6.2
Calcium (mg/dl)	8.38 \pm 1.53	8.79 \pm 1.38
(mmol/L)	2.09 \pm 0.38	2.19 \pm 0.34
Phosphorus (mg/dl)	16.64 \pm 11.84	14.27 \pm 12.53
(mmol/L)	5.37 \pm 3.8	4.6 \pm 4.04
Sodium (mmol/L)	147.91 \pm 4.41	148.63 \pm 3.56
Potassium (mmol/L)	4.85 \pm 0.77	4.32 \pm 0.85

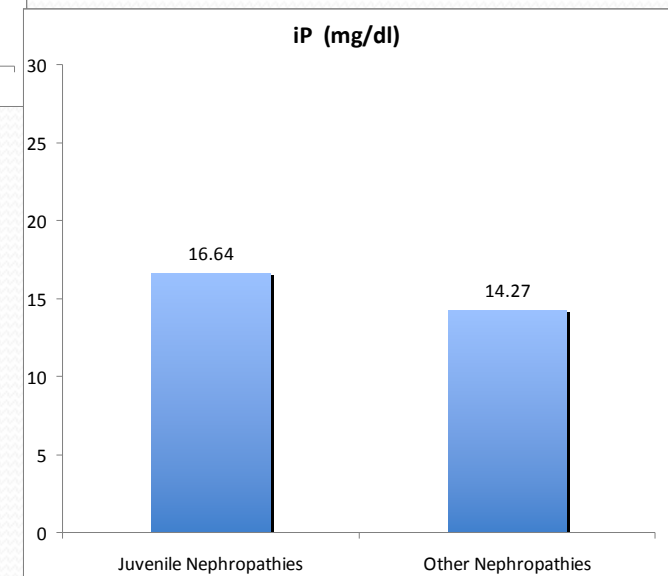
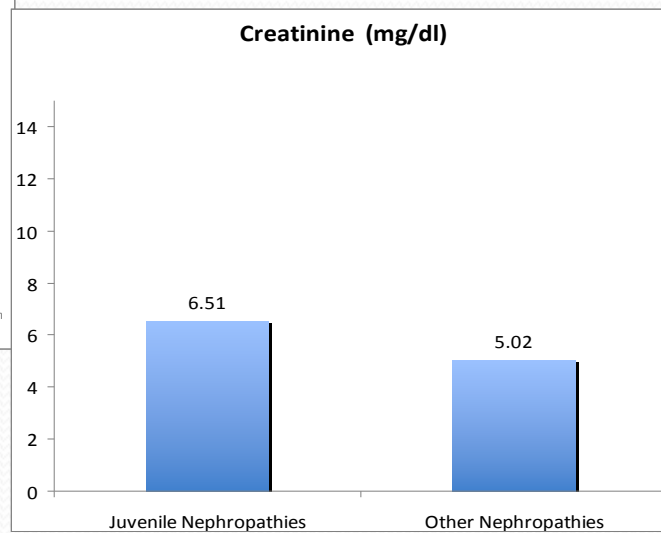
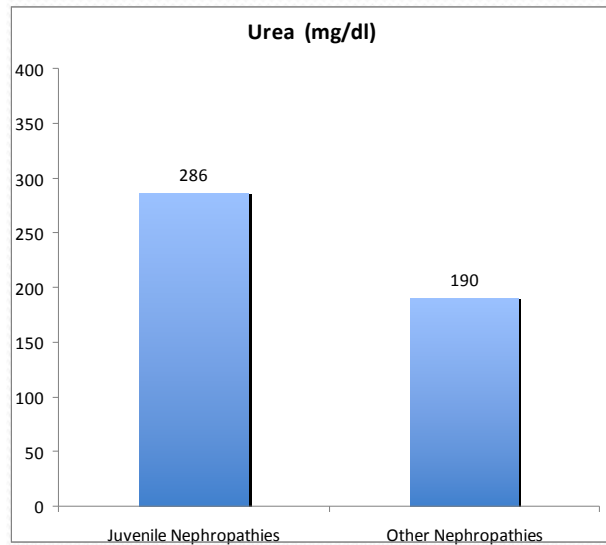
Renal Disease in Boxer dogs: Results

Parameter (time of biopsy)	Juvenile Nephropathy	Others
Urinalysis	Mean	Mean
Specific Gravity	1013.37 \pm 2.33	1012.44 \pm 5.47
pH	6.37 \pm 0.516	6.5 \pm 0.86
Blood stix	1.12 \pm 0.991	1.67 \pm 1.32
Protein stix	1.87 \pm 0.831	1.89 \pm 1.17
UP/UC	2.48 \pm 2.31	3.31 \pm 3.15
Colture pos. (before biopsy)	two dogs	one dog

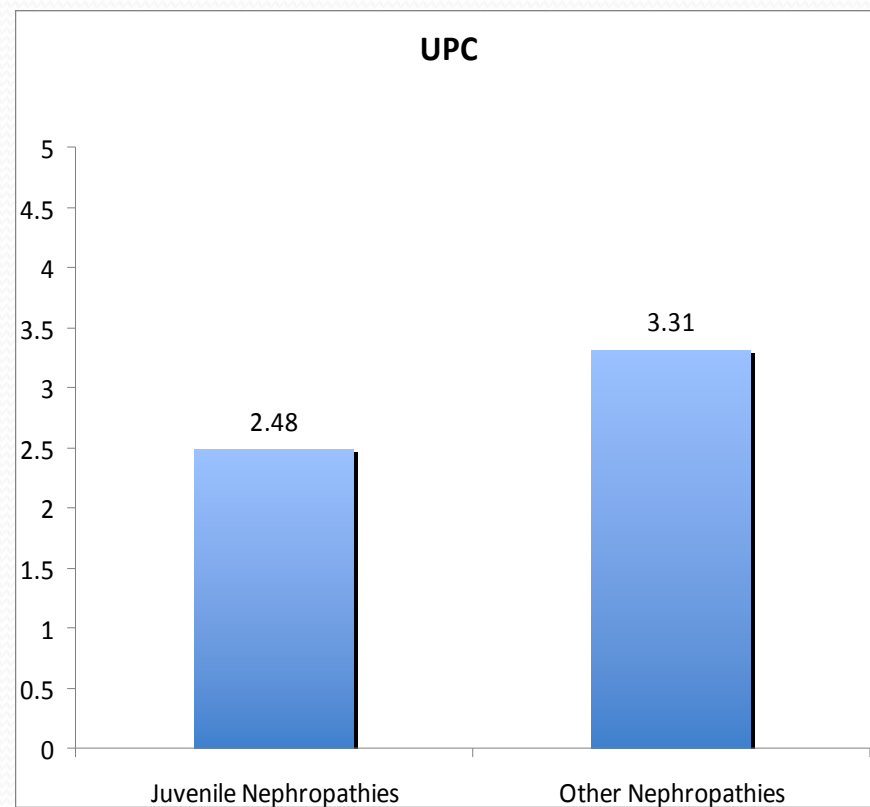
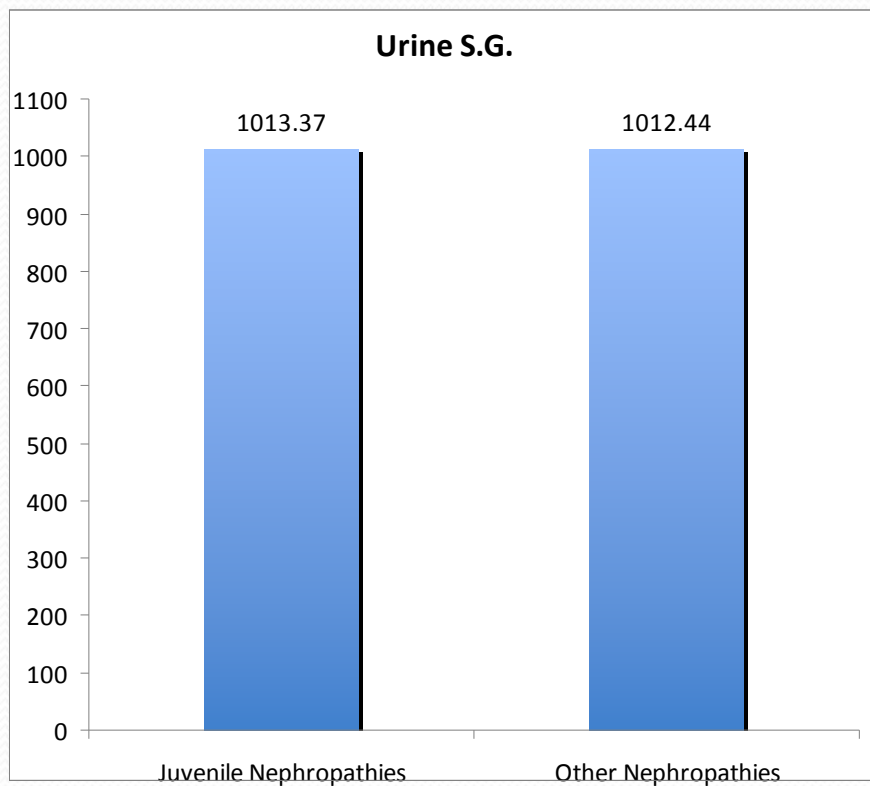
Renal Disease in Boxer dogs: Results



Renal Disease in Boxer dogs: Results



Renal Disease in Boxer dogs: Results



Statistically significant Pearson Correlations ($P < 0,05$) among considered parameters, evaluated all together, in Juvenile Nephropathies, and in Other Nephropathies.

PARAMETER 1	PARAMETER 2	Correlation degree in all Boxer Dogs	Correlation Degree in Juvenile Nephropathy Boxers Dogs	Correlation Degree in Other Nephropathy Boxers Dogs
IRIS Class	BCS	-0,705		-0,754
IRIS Class	Creatinine	+0,814	+0,823	+0,848
IRIS Class	Urea	+0,791	+0,829	+0,842
IRIS Class	iP	+0,658		
IRIS Class	Urine S.G..	-0,491		
IRIS Class	Age			+0,753
BCS Class	Creatinine	-0,665		-0,797
BCS Class	Urea	-0,679		-0,727
BCS Class	Age			-0,797
Creatinine	Urea	+0,960	+0,968	+0,965
Creatinine	Age			-0,849
iP	Creatinine	+0,752	+0,947	+0,705
iP	Urea	+0,757	+0,975	
iP	Albumin	-0,561		
Urinary PH.	Albumin	-0,725		-0,894
Urinary PH	Urinary Protein	+0,582		
Urinary PH	Sodium		-0,957	
Urinary PH	Protein stix		+0,786	
Urinary S.G.	Age			-0,865
Total Protein	Sodium		+0,764	

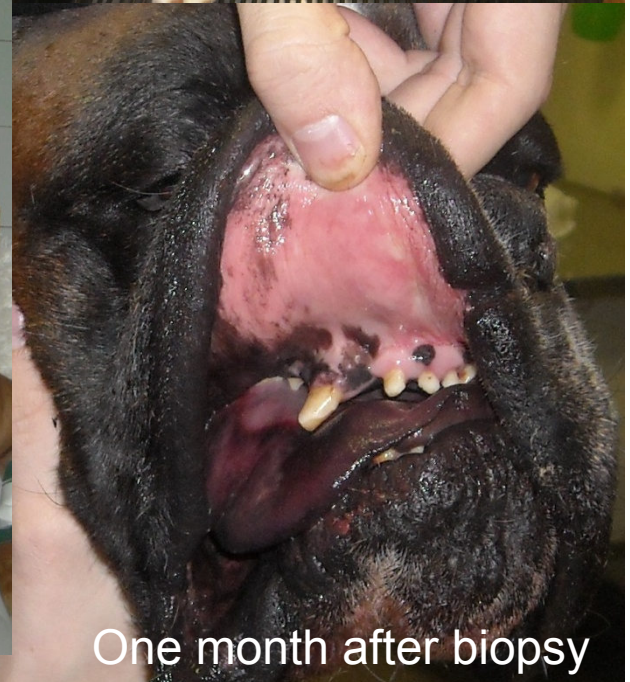
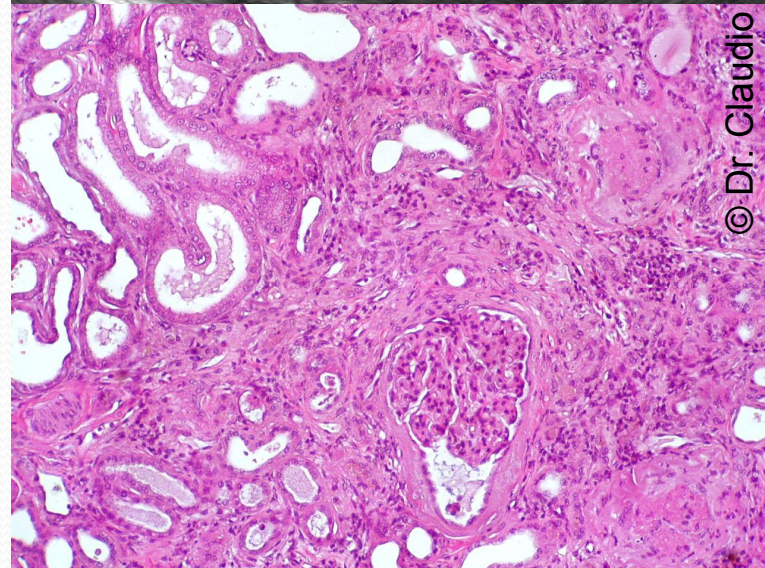
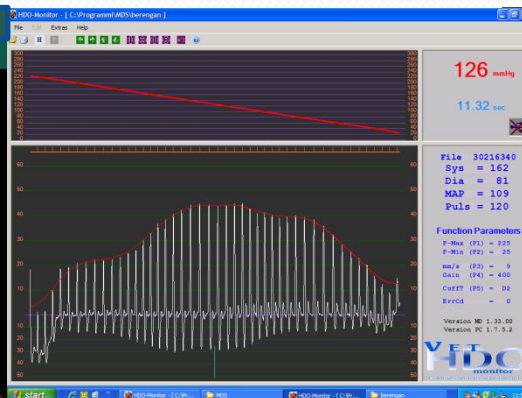
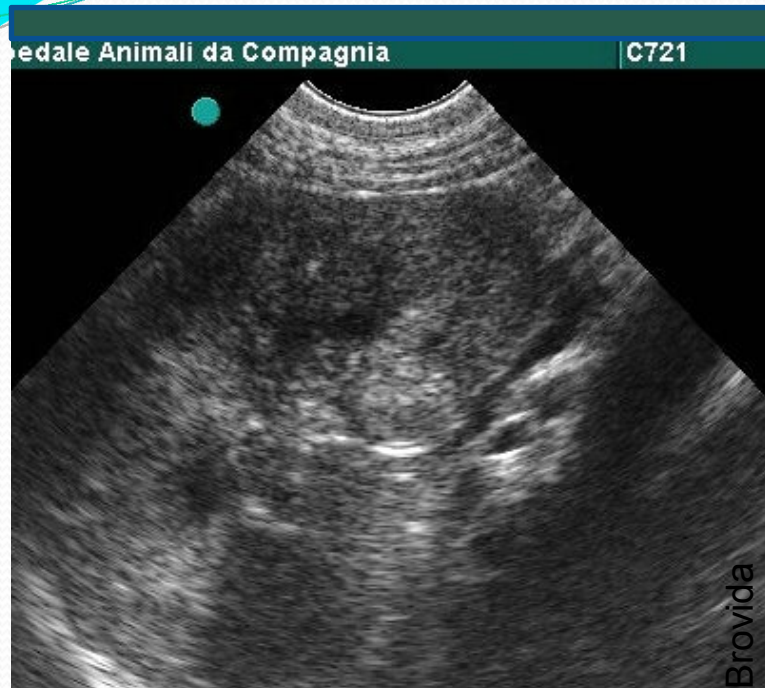
B., M-nc, 6yrs. Juvenile Nephropathy

Referred for evaluation of uremic signs (already treated):

BCS:	3 / 9
CKD IRIS Stage:	4 P, H (178/104)
Hct (%):	36.7
Creatinine (mg/dL / μ mol/L):	9.53 / 842.45
Urea (mg/dL / mmol/L):	442 / 73.59
Albumin (g/dL / g/L):	3.2 / 32.0
iP (mg/dL / mmol/L):	29.7 / 9.59
Sodium (mmol/L):	152
Potassium (mmol/L):	4.83
Urine S.G.:	1015
UPC:	0.75
Culture:	not done



B., M-nc, 6yrs. Juvenile Nephropathy



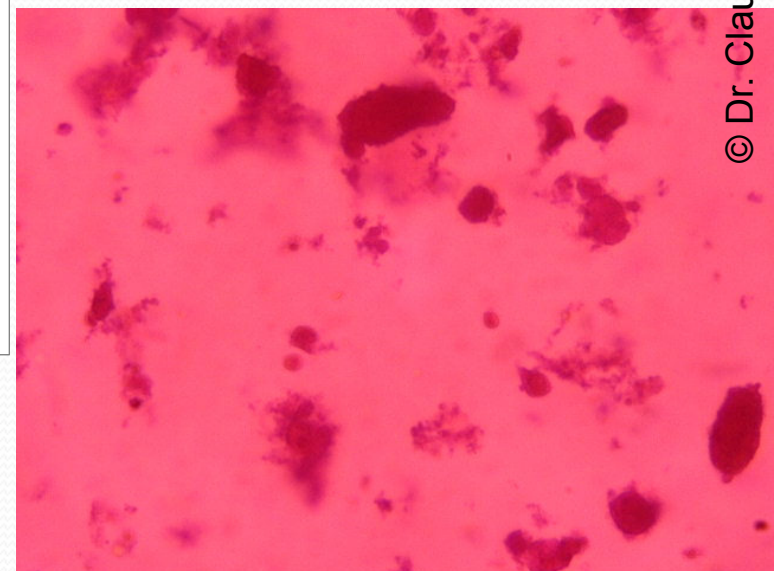
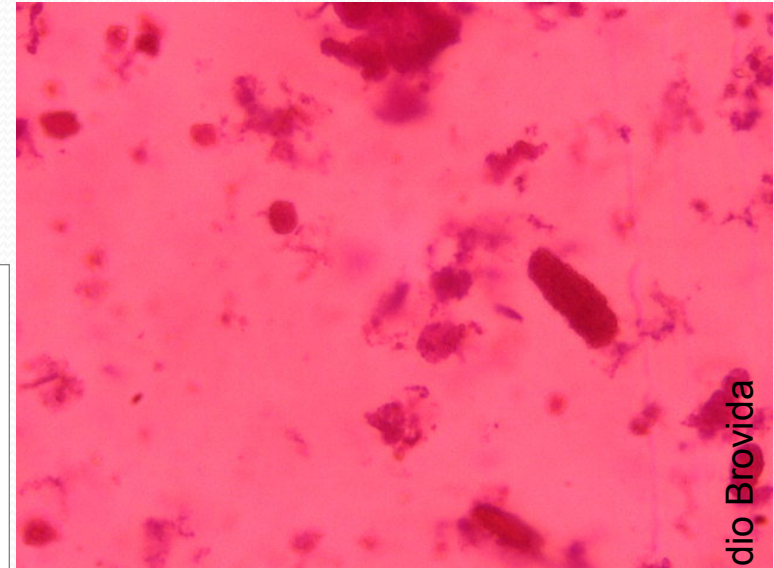
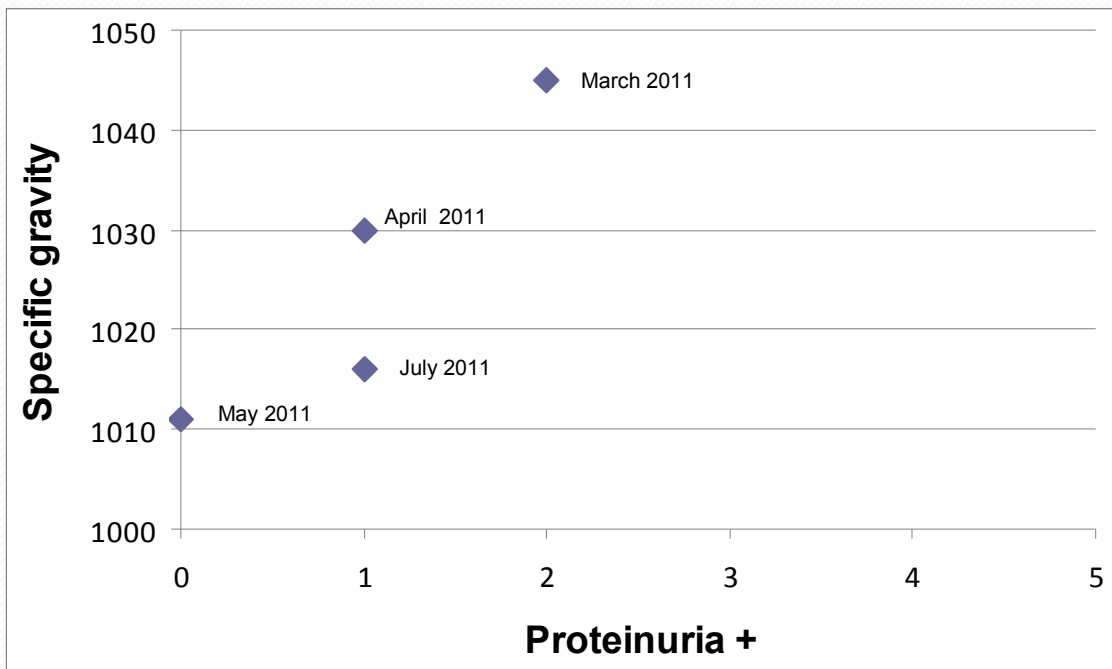
One month after biopsy

© Dr. Claudio Brovida

© Dr. Claudio Brovida

F., M-nc, 1yrs. No CKD, cilindruria

Case included in a study of a group of young Boxer dogs monitored for one year. He very often presented granular casts in the urinary sediment. But was always clinically normal



© Dr. Claudio Brovida

F., M-mc, 1yrs. No CKD, cilindruria

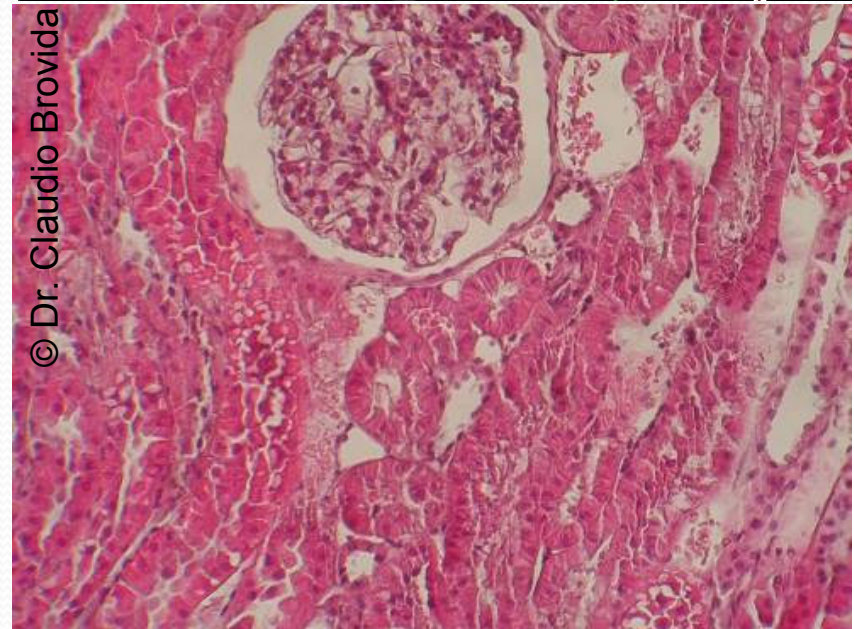
BCS:	5 / 9
CKD IRIS Stage:	No CKD
BP:	140 / 90
Hct (%):	37.7
Creatinine (mg/dL/ $\mu\text{mol/L}$):	1.15 / 101.66
Urine S.G.:	1011
UPC	< 0.2



© Dr. Claudio Brovida

Control on 07/09/2013, the dog is clinically normal, hyper-dynamic.

Urine S.G.:	1018
UPC	< 0.2
Urine sediment:	negative

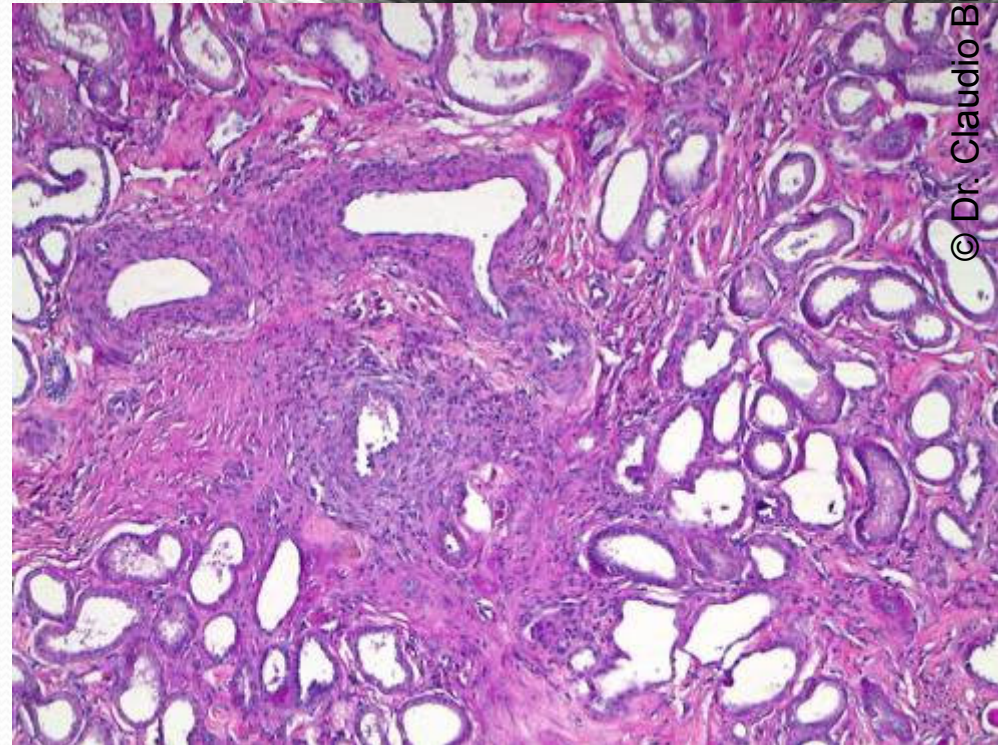
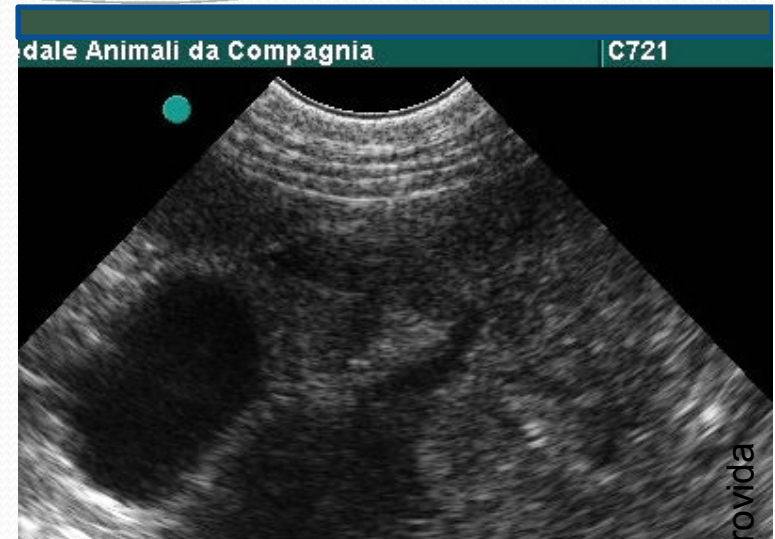


© Dr. Claudio Brovida

Z., F. not sp., 1yrs. Juvenile Nephropathy

Referred for evaluation of advanced uremic signs
(already treated):

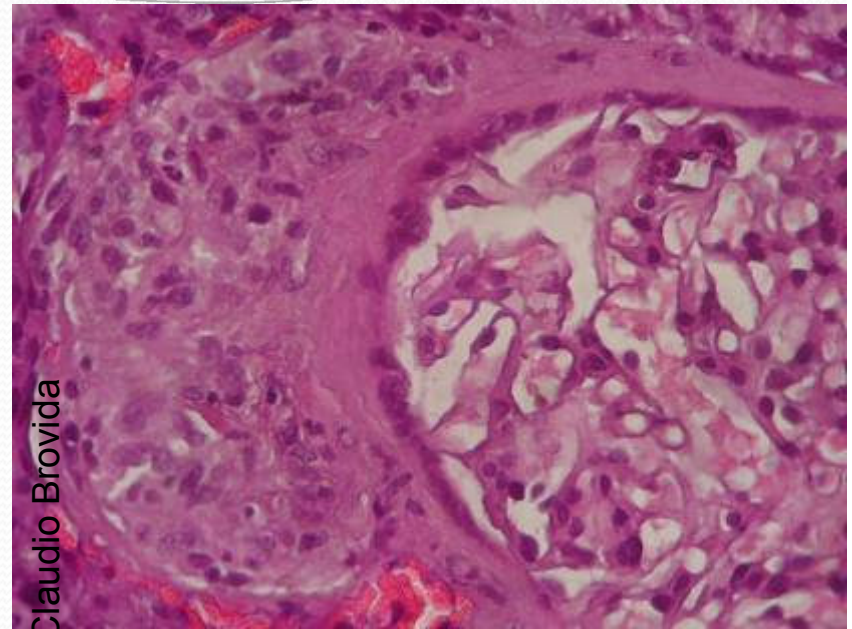
BCS:	2-3 / 9
CKD IRIS Stage:	4 P, H (190/110)
Hct (%):	24.2
Creatinine (mg/dL/ μ mol/L):	9.06 / 800,9
Urea (mg/dL / mmol/L):	480 / 79.9
Albumin (g/dL / g/L):	2.4 / 24
Pi (mg/dL / mmol/L):	26.29 / 8.48
Sodium (mmol/L):	145.4
Potassium (mmol/L):	4.79
Urine S.G.:	1015
UPC:	2.93
Colture:	not done



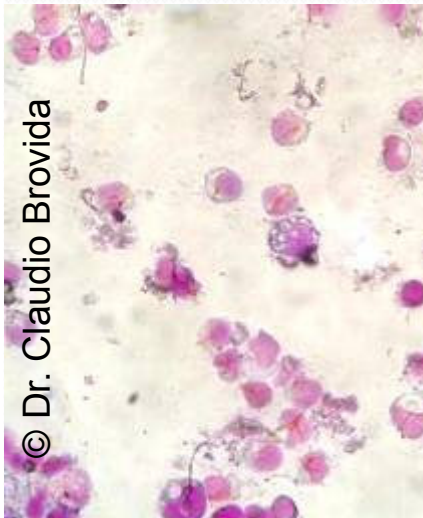
P., M-nc, 3 yrs. Renal lymphoma

Referred for kidney biopsy, polycythemia

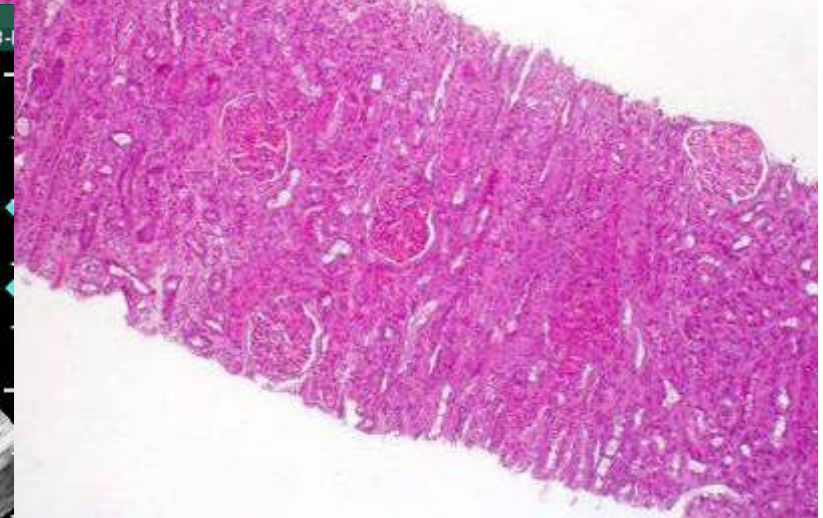
BCS:	4 / 9
CKD IRIS Stage:	2 P,
RBC (M/ μ L):	12.6
Hct (%):	76.5
Creatinine (mg/dL/ μ mol/L):	2.01 / 177.68
Urea (mg/dL / mmol/L):	66 / 0.9
Albumin (g/dL / g/L):	3.1 / 31.0
iP (mg/dL / mmol/L):	4.5 / 1.45
Sodium (mmol/L):	146.0
Potassium (mmol/L):	3.19
Urine S.G.:	1009
UPC:	1.58



© Dr. Claudio Brovida



© Dr. Claudio Brovida



U., M-nc, 18Mths. Juvenile Nephropathy

Referred after a control for a minor surgery

BCS:	5 / 9
CKD IRIS Stage:	2 P, H (170/90)
Hct (%):	41.6
Creatinine (mg/dL/ μ mol/L):	2.37 / 209.5
Urea (mg/dL / mmol/L):	89 / 14.8
Albumin (g/dL / g/L):	2.7 / 27.0
iP (mg/dL / mmol/L):	3.82 / 1.23
Sodium (mmol/L):	145.4
Potassium (mmol/L):	4.61
Urine S.G.:	1014
UPC (after UTI resolution)	3.97
Colture:	<i>Staph.spp</i>

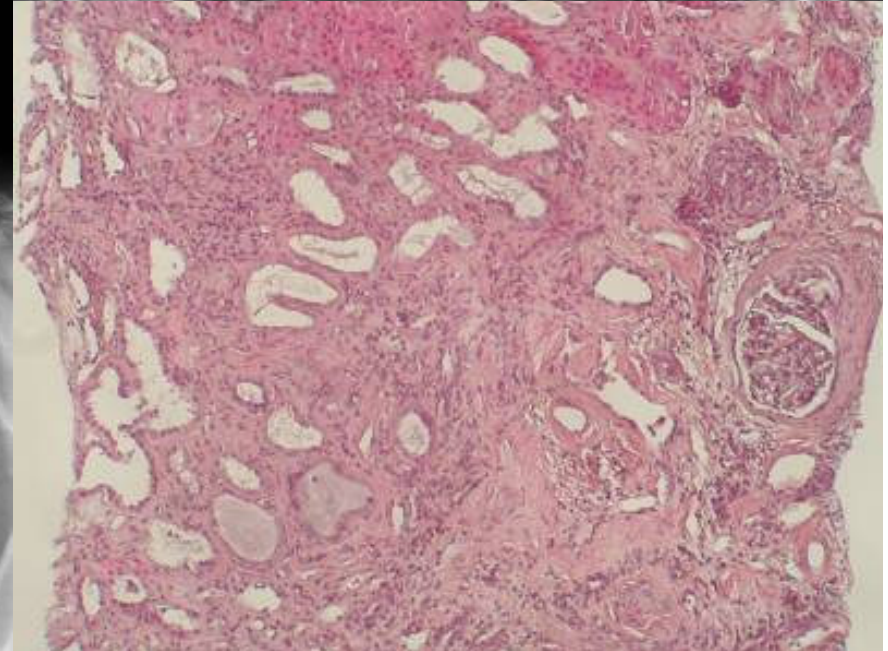
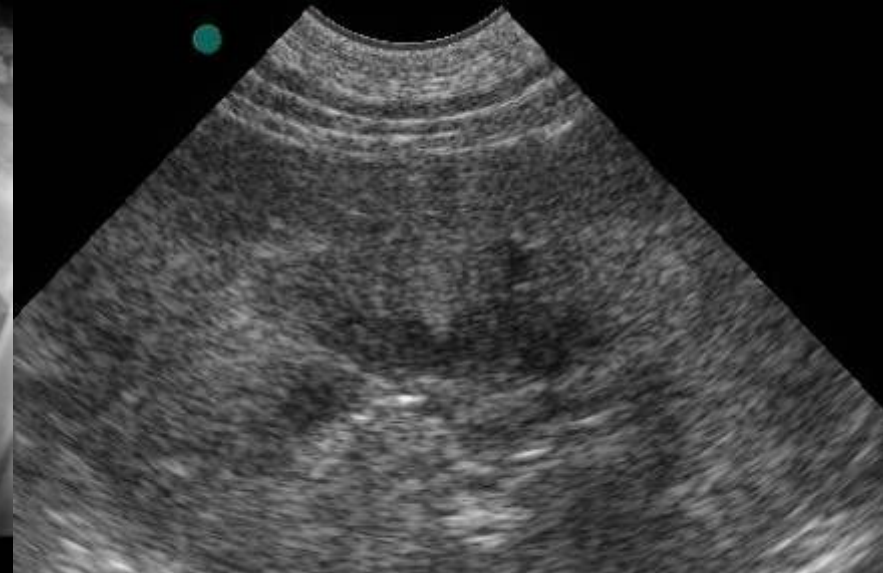


U., M-nc, 18Mths. Juvenile Nephropathy



pedale Animali da Compagnia

C721



DISCUSSION

17 CKD (IRIS Stage 1-4) in Boxer Dogs, age 1-6 yrs, have been evaluated:

8 with Juvenile Nephropathy, 9 with different causes of CKD

All evaluated with clinical examination, blood work, urinalysis, ultrasound and kidney biopsy. Clinical aspects and lab test were similar in the two groups.

Ultrasound aspects may be different in the two groups and could be worthy of particular attention

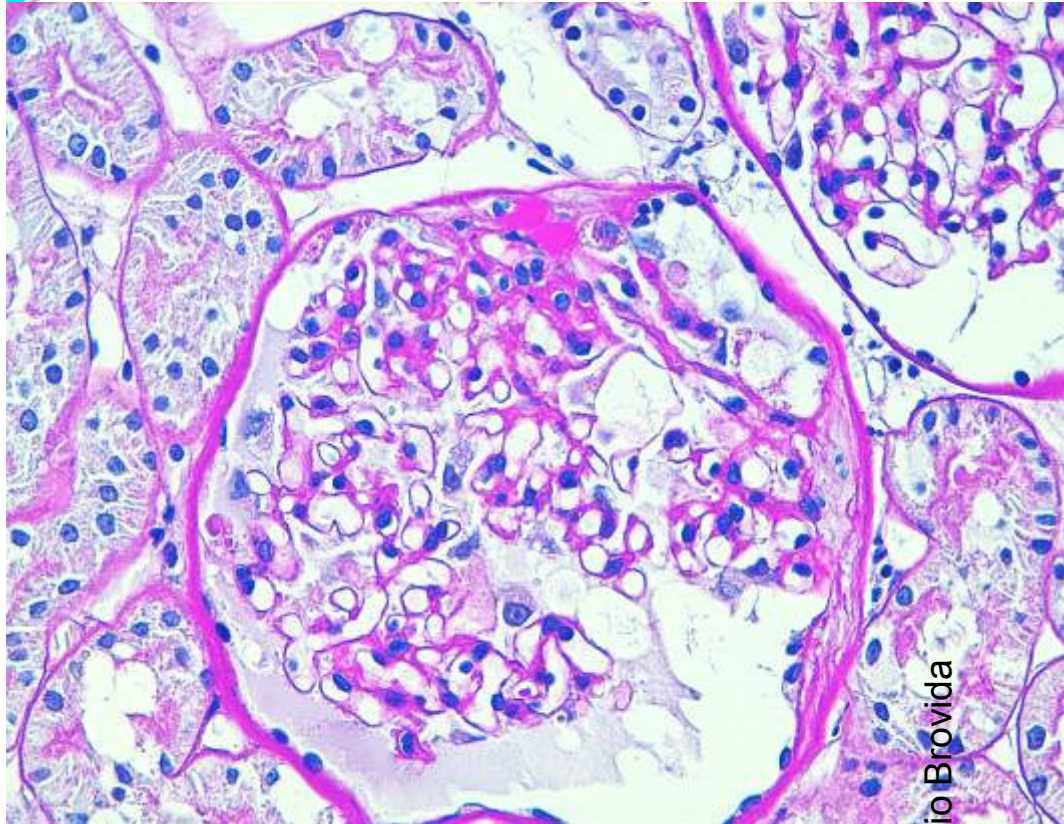
We did not look at genetic linkage, related to J.N. in the evaluated cases, however on the basis of our experience it would not be very difficult in future

The histopathology with LM and EM are specific methods to differentiate J.N. from other causes of CKD in Boxer Dogs

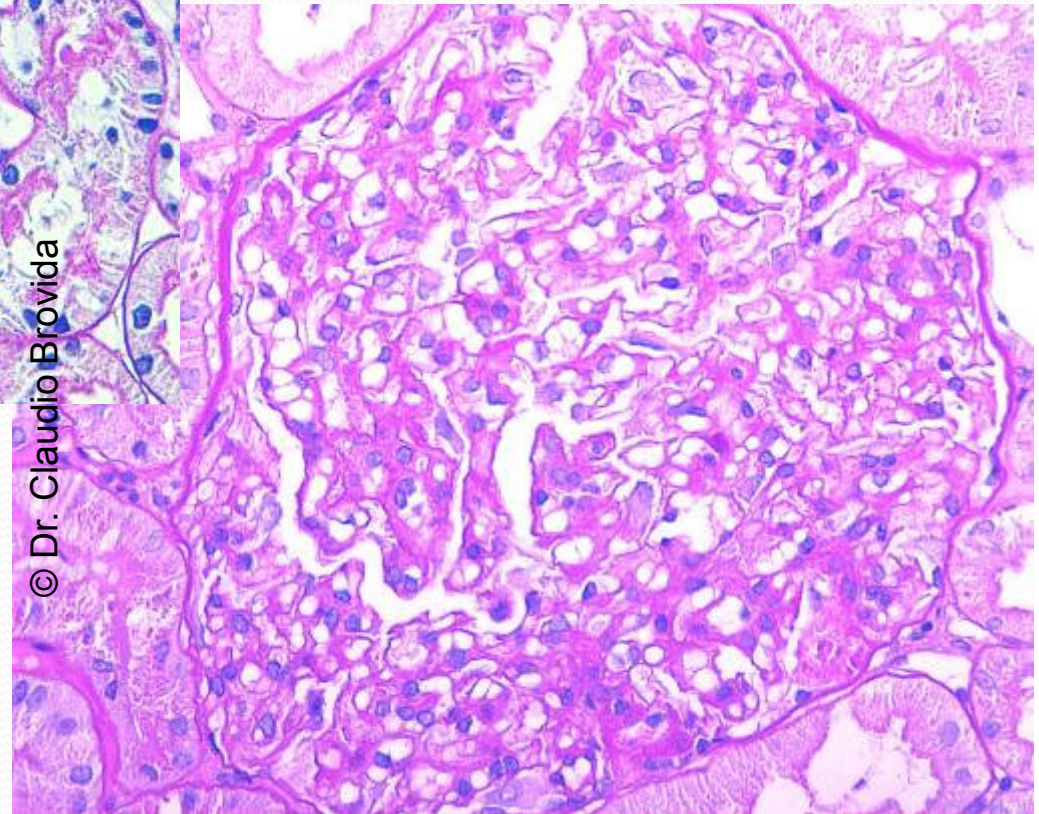
About aetiopathogenesis:.... vesicoureteral reflux, remains a valid hypothesis that needs further validation

Unlike some other breeds in which the aetiology of familial vs. genetic renal disease has been well demonstrated, in Boxer Dogs much more work needs to be done

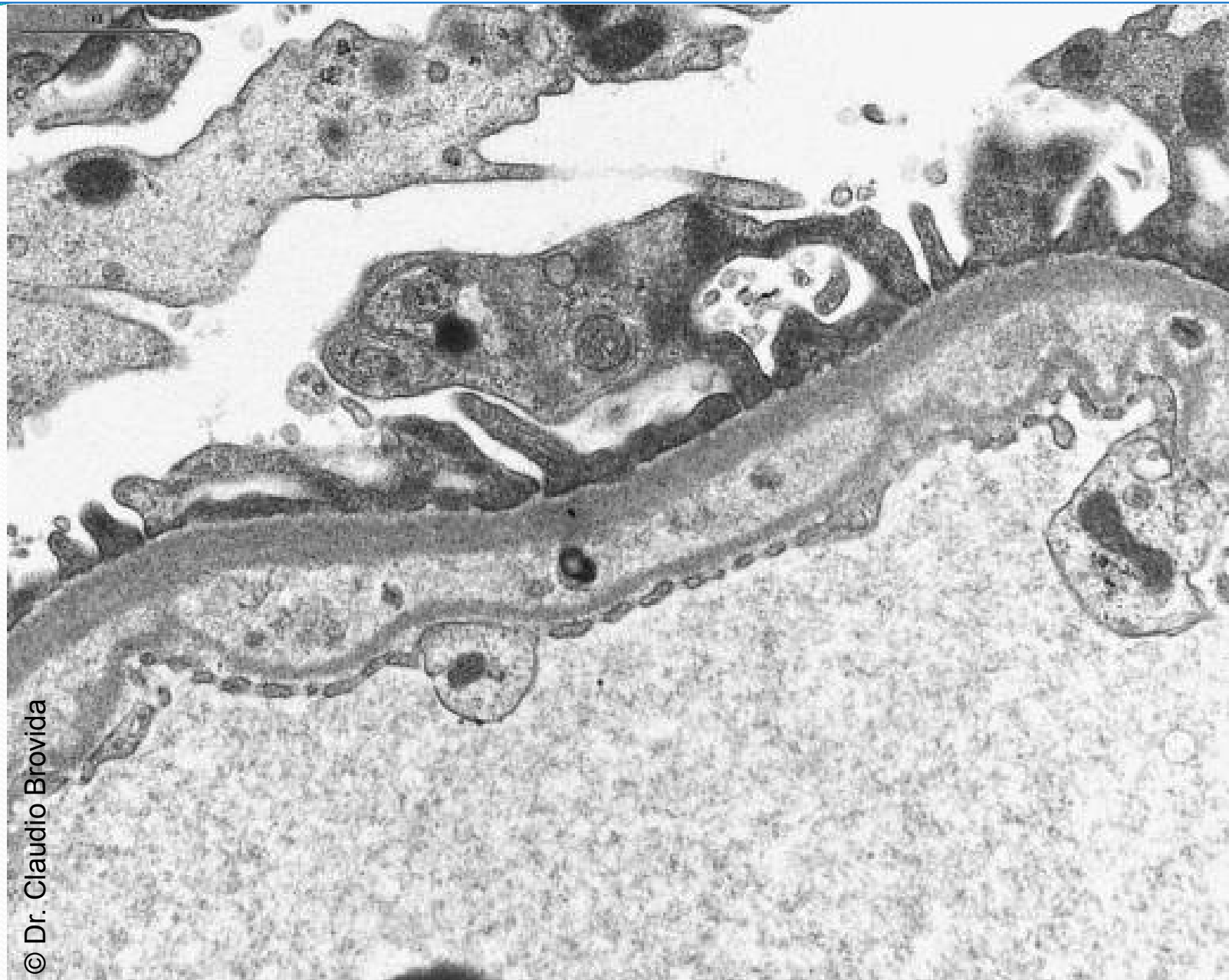
Renal biopsies in early stage of the disease show focal synechiae, podocytes hypertrophy, mesangial hypercellularity and reduction of capillary lumen



© Dr. Claudio Broida



Electron microscopy shows primary podocyte damage: foot process effacement, podocyte loss, prolapse into the urinary space and degeneration

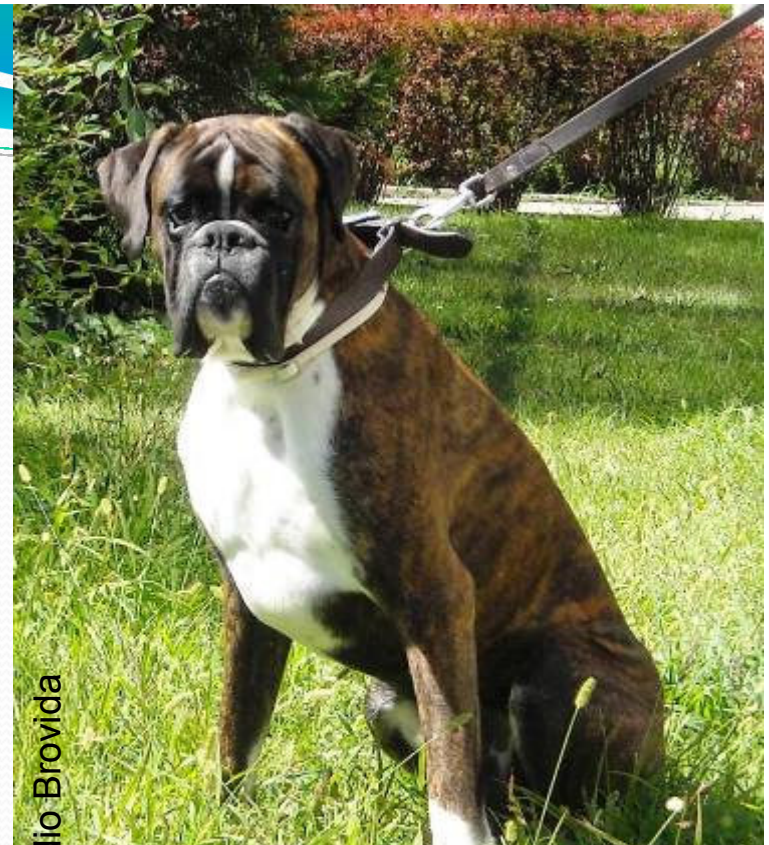


CONCLUSIONS

CKD in young boxers may be multifactorial and while reflux nephropathy may be one pathophysiological mechanism, another mechanism might be a primary podocytopathy.

Early detection of the disease, with systematic control of urinalysis, blood tests, diagnostic imaging, ultrasound and X Rays with positive contrast, and kidney biopsy, will allow a more precise definition of terms related to this disease

Genetic studies on the relationships among the affected dogs and the possibility to analyze the genome in these animals will help to define the origin of the disease



© Dr. Claudio Brovida

