

Suspected primary immune deficiency in a Donge de Bordeaux dog

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ABSTRACT

A young Donge de Bordeaux dog was presented with chronic intermittent antibiotic responsive gastrointestinal and respiratory disease. Further evaluation showed bacterial lymphadenitis, bacterial tracheitis, normal white cell and differential cell counts, hypogammaglobulinaemia, and the absence of B-lymphocytes but the presence of T-lymphocytes in the lymphoid tissue stained with lymphocyte markers. As the dog came from a narrow genetic base, with related dogs showing similar clinical signs, possible B-cell congenital immune deficiency was suspected.

Key words: canine, congenital, hypogammaglobulinaemia.

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A 2-year-old intact male Donge de Bordeaux was referred for peripheral lymphadenopathy, mass loss and intermittent gastrointestinal signs characterised by diarrhoea. From puppyhood onwards the dog appeared to suffer from recurrent respiratory and skin infections that were responsive to antibiotic therapy. It was one of a number of closely related Donge de Bordeaux dogs that showed similar clinical signs.

On clinical examination the dog showed generalised mild peripheral and mesenteric lymphadenopathy and an irritable trachea. Urine and faecal analyses and complete blood count were all within normal parameters (Table 1). The only significant abnormality in serum biochemistry was hyperglobulinaemia, which on electrophoresis showed elevated α 1- and α 2-, normal β - and low γ -globulins. Toxoplasma serum antibody titre was negative. Bronchial lymphadenopathy was present on survey thoracic radiographs. Trans-tracheal aspirate (TTA) cytology showed an inflammatory reaction characterised by neutrophils and macrophages. Culture of TTA yielded a pure growth of *Klebsiella pneumoniae*. Fungal cultures of the TTA fluid were negative.

Biopsy of the popliteal lymph node showed mild hyperplasia of the cortical tissue and diffuse plasma cell infiltration in the medullary cords and paracortex. Staining for infectious organisms (*Toxoplasma*, *Leishmania* and *Chlamydia*) was negative. Culture of lymph nodes yielded

a pure growth of *Staphylococcus intermedius* whereas culture for fungi was negative. Staining of the lymph node with CD3 and CD79a lymphocyte markers indicated the absence of B-cells but the presence of T-cells. T-cells stain positive with the CD3 marker and negative with the CD79a marker, whereas B-cells stain positive with CD79a and negative with CD3.

From the history of the dog, clinical findings, poor white cell response to systemic bacterial infections, low globulins and staining with lymphocyte markers, primary or congenital immune deficiency was suspected. Possible causes would have included X-linked agammaglobulinaemia, transient hypogammaglobulinaemia of infancy, selective IgA deficiency and common variable immunodeficiency syndrome, also known as

acquired or adult onset hypogammaglobulinaemia³. The 1st condition is unlikely as both males and females showed similar clinical signs, and transient hypogammaglobulinaemia during infancy usually resolves as the animal matures^{1,3}. Common variable immunodeficiency syndrome is a primary immunodeficiency disease characterised by little or no antibody production by B-lymphocytes, normal or decreased numbers of B-lymphocytes, and abnormal T-lymphocyte function⁶. The disease is characterised by a great reduction in or complete absence of immunoglobulins⁸. It is most unlikely that the dog in this report had the classic primary severe combined immunodeficiency syndrome as it responded to treatment, did not show growth failure, and did not have severe bacterial or viral infections⁴.

The serum proteins showed a high globulin level, quantified using serum protein electrophoresis, that was due to an acute phase response with a low gamma globulin level. Acute phase proteins are serum proteins identifiable by electrophoresis in the α -globulin zone². The acute phase response occurs during the early stage of infection, tissue injury or immunological disorders⁷. The systemic reaction of the acute phase response includes great changes in acute phase protein (APP) concentrations, primarily due to increased hepatic APP production⁷.

Table 1: Haematological and clinical pathology parameters.

Parameter	Normal values	Day 1	Day 22	Day 30	Day 70	Day 100
White cell count	6–15 × 10 ⁹ /l	15.19	4.62	8.11	17.15	9.69
Neutrophils	3–11.5 × 10 ⁹ /l	10.3	2.68	6.49	10.63	6.3
Band cells	0–0.3 × 10 ⁹ /l	0	0	0	0	0.1
Lymphocytes	1–4.8 × 10 ⁹ /l	2.13	1.02	1.3	3.43	1.55
Monocytes	0.1–1.4 × 10 ⁹ /l	0.15	0.65	0.16	0.86	0.97
Eosinophils	0.1–1.2 × 10 ⁹ /l	0	0	0.16	2.23	0.68
Basophils	0–0.1 × 10 ⁹ /l	0	0.28	0	0	0.1
Thrombocytes	200–500 × 10 ⁹ /l	302	302	196	372	343
Total serum proteins	53–75 g/l	ND*	84	61	74	78
Albumin	25–35 g/l	ND	27	33	26	37
Globulin	25–35 g/l	ND	57	27	48	41
α 1-globulins	2–5 g/l	ND	ND	ND	ND	13.5
α 2-globulins	3–11 g/l	ND	ND	ND	ND	12.8
β -globulins	6–12 g/l	ND	ND	ND	ND	7.2
γ -globulins	5–18 g/l	ND	ND	ND	ND	5.2

*Not done.

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There have been no reports of immune defects in the Donge de Bordeaux, although an outbreak of *Klebsiella pneumoniae* infection has been reported in kennelled Bordeaux dogs⁵. As *K. pneumoniae* is an opportunistic organism it is possible that those dogs had an immune deficiency that predisposed them to the infection.

The diagnosis of an immune deficiency can be suspected from the history and clinical examination of the patient. Clinical signs that may be indicative of an immune deficiency include chronic, recurrent or partially responsive infections caused by organisms of low virulence¹.

To fully elucidate the problem in this breed, a full investigation in other dogs is required. Further evaluation should

consider serum levels of IgA, IgM, and IgG, lymphocyte transformation tests, lymphocyte subset quantification, and neutrophil function tests.

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