Glanzmann Thrombasthenia in Otterhounds

A bleeding disorder called Thrombasthenic thrombopathia was first described in Otterhounds in 1967.[1] Affected dogs had mucosal bleeding and prolonged bleeding times. Platelet aggregation responses were minimal or lacking and clot retraction was severely impaired. Platelet aggregation and clot retraction tests both look at the ability of a patient's platelets to form a strong hemostatic plug at the site of blood vessel injury. Abnormal clot retraction tests and platelet aggregation studies were used as screening tests in an effort to eliminate Thrombasthenic thrombopathia from the Otterhound breed in the 70's and 80's. As a result of this testing, it was thought that the platelet disorder had been largely eliminated from the breed; however, in the early 1990's, descendents of the dogs originally described with Thrombasthenic thrombopathia were identified with platelet dysfunction. Because Thrombasthenic thrombopathia closely resembled the Glanzmann thrombasthenia platelet disorder that had been described in Great Pyrenees dogs in 1996 and 2000,[2-3] molecular studies were begun in Dr. Boudreaux's laboratory to determine whether a mutation could be found that caused Thrombasthenic thrombopathia in Otterhounds. Blood samples were collected from the affected Otterhounds identified by Dr. James Catalfamo at Cornell University in the early 1990's. As a result of this work, it was determined that Otterhound Thrombasthenic thrombopathia was identical to Glanzmann thrombasthenia (GT),[4] based on the finding that affected Otterhounds had a mutation in the gene encoding for platelet glycoprotein IIb (mutations in either of the genes encoding for glycoproteins IIb or IIIa have been documented to cause GT in human beings). Although Otterhounds and Great Pyrenees both have mutations in the gene encoding for platelet glycoprotein IIb and both breeds are affected with GT, the mutations that cause the disease are different for the two breeds. The mutation causing GT in Otterhounds is in Exon 12 in the glycoprotein IIb gene, while the mutation causing GT in Great Pyrenees is in Exon 13.

GT has been recognized for many years in humans and is due to a congenital/inherited membrane defect in platelets. Platelets are small, circulating cytoplasmic fragments that are the first line of defense in stopping the flow of blood from injured blood vessels. An important aspect of platelet function is their ability to stick to each other and plug holes in damaged vessels until blood clotting and tissue repair can occur. The platelets of people and dogs with GT are defective in their ability to stick to each other. Therefore, these individuals are at increased risk for spontaneous hemorrhage and they are also at high risk for excessive hemorrhage as a result of injury or surgery. The type of spontaneous bleeding that occurs with GT includes excessive gingival bleeding during tooth eruption, nose bleeds, and superficial skin bleeds. Young dogs less than 18 months of age are especially prone to excessive, spontaneous bleeding.

By using DNA testing, affected and carrier animals can now be identified by submitting a blood sample through the mail. By using DNA testing, carriers can be accurately identified before breeding to avoid spreading the mutation and to avoid producing affected puppies. Carrier detection is vital in controlling spread of inherited defects and DNA testing is the only reliable method of detecting these animals.

1. Dodds WJ. Familial canine thrombocytopathy. Thromb Diath Haemorrh Suppl 26:241-248, 1967.

2. Boudreaux MK, Kvam K, Dillon AR, Bourne C, Scott M, Schwartz KA, Toivio-Kinnucan M. Type I Glanzmann's Thrombasthenia in a Great Pyrenees Dog. Veterinary Pathology 33:503-511, 1996.

3. Lipscomb DL, Bourne C, Boudreaux MK: Two genetic defects in alpha IIb are associated with Type I GT in a Great Pyrenees dog: a 14-base insertion in exon 13 and a splicing defect of intron 13. Veterinary Pathology 37:581-588, 2000.

4. Boudreaux MK and Catalfamo JL. Molecular and genetic basis for thrombasthenic thrombopathia in Otterhounds. Am J Vet Res 62(11):1797-1804, 2001.

Specimen requirements: At least 1ml EDTA whole blood (purple top tube). Do not cross contaminate samples during collection particularly if more than one dog is collected at the same time. Label all specimens clearly. Protect the tubes to prevent breakage during shipping. All methods of shipping are acceptable. **Blood samples do not require ice**.

Ship to: Hemostasis Laboratory, Peter W. Christopherson
166 Greene Hall
Auburn University, AL 36849-5519

Fee for testing: $130.00 (payment options listed below)

Invoice payments are due within 30 days from the invoice date and can be made securely online: https://www.aub.ie/payinvoice, by mailed check payable to: Pathobiology Diagnostic Services, or through wire transfer (email weldolm@auburn.edu for wire transfer instructions)

Questions regarding invoicing and/or payments: weldolm@auburn.edu

Hemostasis Laboratory
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Dr. Peter W. Christopherson, DVM, PhD, DACVP
166 Greene Hall
Auburn University, AL 36849-5519
PH: 334-844-2797 / 334-844-2697, Fax: 334-844-2652
Email: chrispw@auburn.edu

OFFICE USE ONLY

ACCESSION _____

DATE _____

HEMOSTASIS LABORATORY

Glanzmann Thrombasthenia in Otterhounds

SAMPLE DATE: _____ AGE AT TIME OF SAMPLING OR DATE OF BIRTH: _____

ANIMAL NAME: _____ BREED: _____ SEX: ___ MALE ___ FEMALE

ANIMAL REGISTRATION NUMBER (if applicable): _____

NAME OF SIRE (if applicable): _____

REGISTRATION NUMBER OF SIRE (if applicable): _____

NAME OF DAM (if applicable): _____

REGISTRATION OF DAM (if applicable): _____

PERTINENT HISTORY:

OWNER INFORMATION	VETERINARIAN'S INFORMATION (BILLING INFORMATION)
	REFERRING VETERINARIAN _____
NAME _____	CLINIC _____
ADDRESS _____	ADDRESS _____
CITY/TOWN _____	CITY/TOWN _____
PROVINCE _____	PROVINCE _____
POSTAL CODE _____	POSTAL CODE _____ COUNTRY _____
COUNTRY _____	PHONE _____ FAX _____
PHONE _____	EMAIL _____
	FAX RESULTS ____ EMAIL RESULTS ____

RESULTS (if you would like the results sent to additional emails and/or faxes please list below)

EMAIL 1: _____ FAX 1: _____

EMAIL 2: _____ FAX 2: _____

SPECIMEN REQUIREMENTS: EDTA WHOLE BLOOD (1ML)

TURNAROUND TIME FOR RESULTS: TYPICALLY 8 TO 10 WORKING DAYS UPON ARRIVAL HARD COPIES OF REPORTS AVAILABLE UPON REQUEST

SAVE AS A PDF

RESET FORM