

Heterophils exhibiting toxic changes in a juvenile green sea turtle (*Chelonia Mydas*)

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Abstract

One green turtle (*Chelonia mydas*) was accidentally captured by a fisherman. The preliminary blood film revealed heterophils exhibiting toxic changes. Following medical treatment, blood smears no longer detected toxic changes in heterophils. The heterophilia observed in this turtle may have been caused by inflammation. This case report increases the body of knowledge related to treatment of the injured green sea turtle.

Key words: green sea turtle, blood smear, toxic change, heterophil.

Introduction

Five species of sea turtle have been discovered in Taiwan (Fong et al., 2010). The green sea turtle (*Chelonia mydas*) has been deemed critically endangered by the World Conservation Union (IUCN red list). This turtle is also the species most commonly found stranded on the coast of Taiwan. Stranded sea turtles, as well as those obtained through incidental capture, are routinely announced by the Taiwan Coast Guard Authority and transferred to nearby rehabilitation facilities. Following

medical treatment, rehabilitated sea turtles are released back into their natural environment. Blood values are an important tool for assessing the condition of sea turtles during hospitalization. Previous studies have reported on the hematological and plasma biochemical parameters of green sea turtles (Anderson et al., 2011); however, information related to toxic changes in heterophils is limited. This paper reports on changes in blood values in a juvenile green sea turtle. Blood values were recorded at first presentation and immediately following hospitalization.

Materials and Methods

A fisherman accidentally captured a juvenile green sea turtle off the coast of Taiwan. The animal was transferred to the rehabilitation facility at the National Museum of Marine Biology and Aquarium to receive care by the Coast Guard Administration on June 14, 2012. The length of the curved carapace measured 41.8 cm and the animal weighed 8.8 kg, indicating that it was a juvenile (Samour et al., 1998). The sea turtle was initially placed in a rehabilitation pool. The preliminary physical examination revealed that the turtle was alert, responsive, anorexic, and bore no external lesions. No barnacles were observed covering the plastron, carapace, or skin of the turtle.

Enrofloxacin was administered by

intramuscular injection (5mg/kg of body weight) every 24 hrs as a prophylactic antibiotic. Hope-B drops (1.5 mL PO q 24 hr for 5 days) were also administered as a nutritional supplement. First measure of heterophil, blood was collected from the dorsal cervical sinus in EDTA Vacutainer® tubes with sodium citrate buffer for haematological and biochemical analyses. The total leukocyte count was found to be within the reference interval, and the packed cell volume (PCV) (37%) was normal. The leukogram was characterized by heterophilia ($7.731 \times 10^3 / \mu\text{l}$), and the lymphocyte count ($1.079 \times 10^3 / \mu\text{l}$) was low normal, compared with hematologic data for wild green sea turtles (Komoroske et al., 2011). Moreover, toxic changes in heterophils were observed in the blood film (Fig. 1).



Fig. 1. Heterophil with toxic changes (arrow) consisting of abnormal staining in a green sea turtle blood smear.

We further observed increased plasma concentrations of aspartate aminotransferase (AST)(873U/L), creatine kinase (CK) (>2000U/L), lactate dehydrogenase (LDH) (>900U/L), uric acid (UA) (9.9mg/dl), phosphorus (>15mg/dl), and potassium (8.25 mg/dl). Hypoproteinemia was also

present in the profile.

On Day 5 of the treatment period, the sea turtle began eating and behaving normally. On day 23, second measure of heterophils by blood smears indicated that no longer detected toxic changes in heterophils (Fig. 2), and enrofloxacin therapy

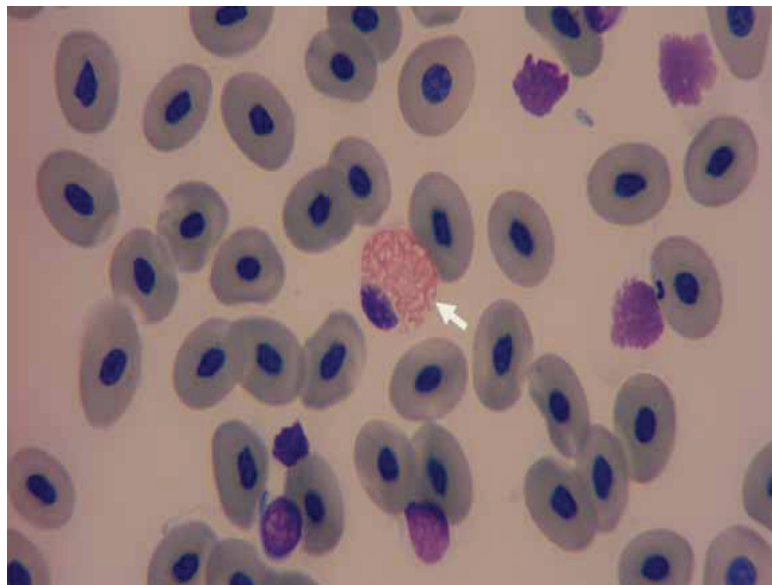


Fig. 2. Normal heterophil (arrow) in a green sea turtle blood smear.

was discontinued. At that time, the values of total plasma protein (TP) and UA had returned to a normal range. Phosphorus and potassium also presented a marked decrease in this profile. Unfortunately, blood work revealed high plasma concentrations of AST, CK, and LDH. Clinical treatment with dexamethasone (0.25mg/kg body weight q 24 hr for 6 days) was subsequently initiated to control inflammation. Following

6 days of therapy, the dosage of dexamethasone was gradually reduced by 0.05 mg per day. After approximately 2 months of treatment, AST, CK, and LDH values returned to within normal limits. In the meanwhile no heterophils with toxic changes were present.

Discussion

This report presents a characterization

of heterophils exhibiting toxic changes due to stress or disease in a green sea turtle undergoing rehabilitation. Compared to the first blood smear, the heterophil morphology found in the second blood film presented no toxic changes. Previously, Caliendo et al., (2010) reported toxic changes in heterophils of severely compromised hawksbill turtles. Heterophils exhibiting toxic changes are often observed in association with inflammatory response, and their disappearance may indicate an improvement in prognosis.

The heterophilia observed in this case may have been caused by inflammation of muscle injury, indicating increased heterophils migration to the injured tissue sites, particularly in light of the elevated plasma AST, CK, LDH, potassium, and phosphorus recorded during the initial observation. Injured turtles generally present higher levels of AST than those found in healthy turtles (Whiting et al., 2007). Nevertheless, plasma AST is not considered an organ-specific enzyme in reptiles. Increased AST activity suggests hepatocellular leakage, muscle cell injury, or mild tissue damage to the dermis or shell (Stamper et al., 2005).

As with AST, plasma LDH is not considered to be organ specific in reptiles. The elevated LDH levels observed in this animal could be associated with injury to the liver or muscles. Harris et al., (2011) reported that elevated AST, LDH, and potassium levels in foraging leatherbacks

are likely associated with muscle damage or increased metabolic activity .

However, plasma ALT is far more liver specific than AST (Allison et al., 2012). As a result, the normal ALT values in this animal eliminate the possibility that liver damage contributed to the increase in AST.

High CK values were previously observed in a leatherback sea turtle missing half a flipper with evidence of muscle damage (Deem et al., 2006). However, animals suffering from capture myopathy commonly present increased CK, AST, and LDH values (Businga et al., 2007).

In this report, elevated levels of primary enzymes associated with muscle damage may have been caused by entanglement in fishing nets during accidental capture. These enzyme levels returned to normal following treatment. In a previous study, sea turtles with decreased AST and CK values during rehabilitation were found to have a better prognosis (Caliendo et al., 2010).

This case report increases the body of knowledge related to treatment of the endangered green sea turtle. Additionally, this report could help veterinarians to improve their interpretations on blood profiles of injured sea turtles.

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