CLINICAL ARTICLE

# RECURRENT, IDIOPATHIC URTICARIA IN A TONGA PONY MARE

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## ABSTRACT

A 15-Year old tonga (a light wheeled horse cart) pony mare was presented for the treatment of a condition characterized by generalized urticarial wheals, anemic mucous membranes, concentrated urine, inappetance and unilateral chemosis. Haematological alterations included a marked leucocytosis (WBC = $14x10^3$/ul) with monocytosis ($2.5x10^9$ /L) and moderate anemia (RBC count = $4.1x10^{12}$/L; PCV = 18; hemoglobin = 8.2 gm/dl). There was no history of exposure to agents known to trigger urticaria. Parental administration of a corticosteroid preparation (prednisolone plus dexamethasone) and antihistamine (pheniramine maleate) in 4 rounds of treatment over a 16 days period was associated with rebounding of signs of urticaria after cessation of each round of therapy.

**Key words:** Urticaria, recurrent, idiopathic, pony mare.

## CASE HISTORY

A tonga (a light wheeled horse cart) pony mare, aged approximately 15-years and weighing about 350 kg, was brought to the outdoor clinics of Department of Clinical Medicine and Surgery, University of Agriculture, Faisalabad with a history of 2 weeks standing anemia and a decreased appetite. Anamnesis indicated that the mare had been developing urticarial swellings intermittently for the past five days. Prior to referral, the animal had been unsuccessfully treated with antibiotics, antihistamines and antipyretics.

## CLINICO-PATHOLOGICAL EXAMINATION

Clinical examination revealed a mild fever (103°F), tachypenia (respiration rate 68|min), dullness and innumerable urticarial wheals of various sizes covering the entire abdomen, thorax and neck. Anemic mucous membranes, a dark colored urine and a unilateral chemosis were the other associated signs. Hematological examination revealed a leucocytosis, with monocytosis and a moderate anemia (Table I).

**Table 1. Hematological values of a tonga pony mare suffering from recurrent, idiopathic urticaria**

| Haematological parameters | Observed values | Normal values* |
|---|---|---|
| Red blood cells ($x10^{12}$/L) | 4.1 | 5.5-9.5 |
| Packed cell volume (%) | 18 | 32-52 |
| Hemoglobin (gm/dl) | 8.2 | 8-14 |
| White blood cell Count ($x10^3$/ul) | 14 | 6-12 |
| Neutrophils ($x10^9$/L) | 4.3 | 2.5-7 |
| Lymphocytes ($x10^9$/L) | 2.7 | 1.6-5.4 |
| Monocytes ($x10^9$/L) | 2.5 | 0.6-0.7 |
| Eiosinophils ($x10^9$/L) | 0.5 | 0.1-0.5 |
| Basophil ($x10^9$/L) | 0 | Usually absent |

*(Jhonston, 1994)

Blood films stained with Giemsa and Dip Quick (Difco) stains were negative for extracellular and intracellular parasites. Fecal examination revealed a moderate number of nonmotile protozoan cysts and strongyles eggs.

## DIAGNOSIS AND TREATMENT

On the basis of clinical signs of urticaria together with chemosis, a clinical diagnosis of allergy was reached. Concomitantly, babesiasis was also suspected. In view of above, the following treatment was instituted as 1st round of therapy:

1. Inj. imidocarb (Imizol, ICI, Pakistan Ltd.) 4 ml, I/M,
2. Inj. prednisolone + dexamethasone (Predexa, Shinil Chemical & Livestock Co. Ltd., Korea.) 10 ml, I/M
3. Oxfendazole (Oxanazole, Sanna Laboratories, Pakistan) 60 ml, PO.

On the following day, the animal was found to have resumed almost normal appetite. Rectal temperature reduced to normal (101°F) and chemosis had disappeared. However, the owner returned again after 2 days with the relapse of urticarial swellings. The following treatment was instituted for 4 days as a 2nd round of treatment without any effect:

1. Inj. prednisolone + dexamethasone (Predexa, Shinil Chemical and Livestock Co. Ltd., Korea.) 10, 10, 8, 7 ml. I/M. on day 1, 2, 3 and 4, respectively.

The following treatment was instituted as the 3rd round of therapy:

1. Inj. prednisolone + dexamethasone (Predexa, Shinil Chemical and Livestock Co. Ltd., Korea.) 5 ml, I/M.
2. Inj. pheniramine maleate (Antivil, Orient Lab. Ltd.) 20 ml, I/M.

Even after 3rd round of treatment, the animal developed urticarial wheals again 24 hours after treatment.

The following treatment was recommended as the 4th round of therapy:

1. Hydroxyzine hydrochloride (Allerid, Pharmatec, Pakistan (Pvt) Ltd.) 500 mg, PO, t.i.d.

But this treatment could not be instituted due to high cost of about Rs.150/dose and the owner declined any further treatment.

## DISCUSSION

Urticaria is a rare problem affecting different animals and human. The major causes of urticaria that have been incriminated in horses are reactions to drugs, insects, ingestants and inhalants (Evans, 1993). Ostensibly, there was no exposure to these triggering factors in the subject of the present report. As such, the urticaria was designated idiopathic. According to Evans (1993), this type of urticaria is common in horses.

An immunoglobulin (Ig) E-mediated type-1 hypersensitivity reaction is frequently incriminated in the pathogenesis of equine urticaria, even though critical investigations into the pathogenesis have not been performed. At best, the pathophysiology of urticaria in horses is poorly understood. In humans, mast cells are the primary effector cells in urticaria (Schwartz, 1991). Although, no studies have been performed to clarify the role of mast cells in equine urticaria, similarities in the pathomechanism are expected. Mast cells activation results in secretion of an array of mediators, which can be grouped into three classes of biologically active molecules: preformed mediators, newly generated lipid mediators and cytokines.

Mast cells are activated by immunologic and non-immunologic factors. Immunogenic activation may involve classic type-1 hypersensitivity (in which there is cross-linking of the high-affinity receptors for immonoglobulin E in the mast cell plasma membrane) or may be a non-immunoglobulin E-dependant immunologic reaction mediated by cytotoxic or immune-complex interaction with mast cells.

Non-immunogenic activation of mast cells can occur via direct degranulation, perturbation of arachidonic acid metabolism, physical stimuli, idiosyncratic reactions, and histamine-releasing factors (Schwartz, 1991; Soter, 1991). Non-immunogenic activation of mast cells, by definition, does not involve an allergic mechanism and thus may occur without previous exposure and sensitization to the triggering agent. Regardless of the sequence of events that precedes mast cell degranulation, the result is the same: the release of mediators that produce urticarial eruption (Evans, 1993).

The most effective therapy for urticaria is identification and removal of the cause. Many cases of equine urticaria are idiopathic; therapy thus is directed towards control of the clinical signs. Corticosteroid preparations (in particular those containing prednisone and prednisolone) and antihistamines are commonly used in the therapy of urticaria in horses. According to Evans (1993), most patients with chronic idiopathic urticaria fail to respond to antihistamines labeled for use in horses (i.e. pyrilamine maleate and tripelennamine hydrochloride).

By contrast, the antihistamine hydroxyzine HCl is reportedly very effective in treating chronic urticaria in equines (Fadock, 1990; Evans, 1991; Rozenkrantz and Griffin, 1986). Repeated administration of a corticosteroid preparation (Predexa, Shinil Chemical & Livestock Co. Ltd., Korea.) as well as antihistamine preparation (Antivil, Orient Lab. (Pvt) Ltd.), during 4 rounds of therapy over 16 days failed to affect a cure of idiopathic urticaria in the subject of present report. The antihistamine hydroxyzine HCl could not be tried in the subject of the present report because it was not affordable by the client. Whether or not this unique antihistamine would have affected a cure in the present case is not known.

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