TENOSYNOVITIS OF THE DEEP DIGITAL FLEXOR TENDON IN HORSES

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Introduction

Tenosynovitis of the deep digital flexor tendon (thoroughpin) in horses is manifested by distention of its tarsal synovial sheath due to formation of an excessive synovial effusion. Unless tenosynovitis is acute, signs of inflammation, pain or lameness are absent (1). Tendinitis can and does occur in conjunction with inflammation of the tarsal synovial sheath.

As tendons function they are frequently subjected to considerable strain, peritendinous pressure, and friction between the parietal and visceral layers of the tendon sheath (2). Acute direct trauma or trauma that is multiple and minor can precipitate tenosynovitis. In acute tenosynovitis of the deep digital flexor tendon, the ensuing inflammatory reaction affects the tarsal synovial sheath, which responds to inflammation by formation of an excessive synovial effusion. Chronic tenosynovitis follows the acute form or it can be insidious in onset. It is characterized by persistent synovial effusions, stenosis of the tendon sheath, or adhesions between the parietal and visceral layers of the tendon sheath and the enveloped tendon (6, 8). Riziform bodies may be present in synovial effusions in chronic tenosynovitis.

Therapy in this group of horses was directed at preventing formation of excessive synovial effusion by aspiration of the tarsal synovial sheath and the intrathecal injection of an adrenocortical steroid or one of its synthetic analogues. Presented in this report are clinical signs, response to intrathecal injection of synthetic steroids, and results of synovial effusion analyses. Analyses of synovial effusions were performed to characterize synovial effusions produced in horses affected with tenosynovitis of the deep digital flexor tendon.

Materials and Methods

The tarsal synovial sheaths of the deep digital flexor tendon in eight clinically affected horses were aspirated and injected with synthetic steroids at the time of diagnostic aspiration (Table I). Since synovial fluid was unobtainable from normal synovial tendon sheaths, statistical comparisons were made between certain values determined for synovial effusions from tarsal synovial sheaths of affected horses and synovial fluids from the tibiotarsal joints of control horses.

Control Horses

Five healthy horses ranging in age from four to nine years were used as controls. Four of the horses were Thoroughbreds and one horse was of Quarter Horse breeding. All control horses were geldings. Synovial fluid samples were obtained from the tibiotarsal joint.

Hematologic Determinations

Blood samples for determination of serum sugar content (measured as total reducing substances) were obtained from the jugular vein prior to aspiration of the tarsal synovial sheath in affected horses and the tibiotarsal joint in control horses. The clot was allowed to retract at 5° C and the samples were then centrifuged at 1,600 g at 5° C.

Synovial Effusion Determinations

Synovial effusion samples were transferred from aspirating syringes to screw-capped tubes which contained dried dipotassium ethylenediaminetetraacetate[1] (EDTA) in a ratio of 2 mg of EDTA per milliliter of synovial effusion. Synovial fluid samples from the tibiotarsal joints of control horses were not transferred to tubes containing EDTA. Tests for relative viscosity (RV), mucinous precipitate quality, total protein content, albumin: globulin (A:G) ratio, and sugar content (measured as total reducing substances) were performed on synovial effusions and synovial fluids after centrifugation at 1,600 g at 5° C (5, 12, 15, 18). Total erythrocytic and leukocytic counts were determined from noncentrifuged portions of the synovial effusion and synovial fluid samples (12). Smears for differential counts were stained with Wright's stain. One hundred leukocytes were identified and counted (neutrophils, lymphocytes, monocytes, macrophages, and eosinophils).

Bacteriologic Determinations

A portion of the synovial effusion sample from two of the horses was transferred to a

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[1]Cambridge Chemical Products, Inc., Dearborn, Mich.

TABLE I

HORSES AFFECTED WITH TENOSYNOVITIS OF THE DEEP DIGITAL FLEXOR TENDON, AFFECTED TARSAL SYNOVIAL SHEATH, SYNTHETIC STEROID, AND DOSAGE

Horse No.	Breed	Age (yrs.)	Sex	Affected tarsal synovial sheath	Interval since previous injection (days)	Synthetic steroid and dosage per tarsal synovial sheath
1	Standardbred	4.0	Mare	Right	.....	200 mg 6 α-methylprednisolone acetate
2	Thoroughbred	10.0	Stallion	Left	.....	200 mg 6 α-methylprednisolone acetate
3	Standardbred	2.0	Stallion	Left	.....	120 mg 6 α-methylprednisolone acetate
4	Standardbred	3.0	Mare	Left	.....	200 mg 6 α-methylprednisolone acetate
5	Standardbred	6.0	Gelding	Right	.....	200 mg 6 α-methylprednisolone acetate
6	Arabian	11.0	Stallion	Left	.....	100 mg 6 α-methyl, 17 α-hydroxyprogesterone acetate
7	Quarter Horse	5.0	Mare	Right	.....	150 mg 6 α-methyl, 17 α-hydroxyprogesterone acetate
8	Standardbred	3.0	Gelding	Left	.....	30 mg 9-fluoroprednisolone acetate 300 mg 6 α-methyl, 17 α-hydroxyprogesterone acetate
					34	30 mg 9-fluoroprednisolone acetate 300 mg 6 α-methyl, 17 α-hydroxyprogesterone acetate

sterilized test tube immediately following aspiration of the tarsal synovial sheath. Synovial effusion samples were inoculated into brain heart infusion[2] semisolid (0.15% agar) and bovine blood agar plates, and incubated at 37° C. Negative cultures were held up to 10 days.

Results

Clinical Signs

Tenosynovitis of the deep digital flexor tendon was unilateral in all horses and was manifested by a fluctuant, palpable distention of its tarsal synovial sheath. The tarsal synovial sheath was distended on both medial and lateral sides of the tarsus; however, the greatest degree of distention was limited to the medial aspect of the tarsus. Degree of distention was variable, dependent on the capacity of individual tarsal synovial sheaths for retention of excessive synovial effusion. Tenosynovitis was chronic in all horses at the time of clinical examination, and signs of inflammation, as manifested by heat and pain on palpation, and lameness were not apparent. None of the horses had developed adhesions between the parietal and visceral layers of the tarsal synovial sheath, and the tendon of the deep digital flexor.

Clinical Response to Intrathecal Injection

Aspiration of affected tarsal synovial sheaths provided symptomatic relief from intrathecal pressure and had a therapeutic effect by removing excessive synovial effusion. Intrathecal injection of synthetic steroids controlled formation of excessive synovial effusions in tarsal synovial sheaths. Dosage and choice of injectable synthetic steroid in each horse was determined by the degree of distention of the tarsal synovial sheath and the approximate duration of the synovial effusion (Table I). Acute inflammation was not a factor in determining therapeutic dosages of synthetic steroids in this group of horses.

Response to intrathecal injection of various synthetic steroids was manifested clinically by resolution of excessive synovial effusion and a concomitant reduction in distention of the affected tarsal synovial sheath. Clinically, favorable response to the intrathecal injection of synthetic steroids generally required from three to six weeks. Despite intrathecal injection of synthetic steroids, a minor palpable peritendinous fibrosis persisted to some degree in the more chronically affected tarsal synovial sheaths. Advanced peritendinous fibrosis made it difficult to achieve complete reduction of chronically affected tarsal synovial sheaths. When possible, the affected tarsal synovial sheath was kept under a pressure bandage until resolution of excessive synovial effusion was achieved.

Five tarsal synovial sheaths were injected with 6 α-methylprednisolone acetate[3] (40 mg / cc) and two tarsal synovial sheaths received injections of 6 α-methyl, 17 α-hydroxyprogesterone acetate[4] (50 mg / cc). Excessive synovial effusion was controlled in a three-year-old Standardbred gelding (Table I, horse 8) affected with chronic tenosynovitis of the tarsal synovial sheath following the second intrathecal injection of 30 mg of 9-fluoroprednisolone acetate[5] (2 mg / cc) and 300 mg of 6 α-methyl, 17 α-hydroxyprogesterone acetate.

Intrathecal injection of 6 α-methyl, 17 α-hydroxyprogesterone acetate did not alter the estrous cycle of one mare or impair fertility in one stallion (Table I, horses 6 and 7).

Synovial Effusion Findings

All synovial effusions from horses affected with tenosynovitis of the deep digital flexor tendon were transudative. Quantity and gross appearance of synovial effusions were recorded at the time of aspiration and intrathecal injection (Table II). Potential capacity of individual tarsal synovial sheaths and the duration of tenosynovitis determined the quantity of retained excessive synovial effusion. Grossly, synovial effusions were either pale yellow and clear, or amber and clear, or amber and opaque in appearance. One synovial effusion contained some flocculent material. Synovial xanthochromia was more pronounced in amber synovial effusions due to continued intrathecal hemorrhage and subsequent hemolysis antecedent to aspiration.

Relative viscosity and mucinous precipitate quality were determined for each synovial effusion sample (Table III). These procedures determined the relative viscosity of synovial effusions and, qualitatively the concentration and polymerization of the hyaluronic acid moiety in synovial effusions from horses affected with tenosynovitis of the deep digital flexor tendon. There was no significant ($P > 0.05$) difference between the relative viscosity of synovial effusions from the tarsal synovial sheaths of affected horses (RV = 2.44) and synovial fluids from the tibiotarsal joints of control horses (RV = 3.72). Mucinous preci-

[2]Difco Laboratories, Inc., Detroit, Mich.

[3]Depo-Medrol, Upjohn Company, Kalamazoo, Mich.

[4]Depo-Provera, Upjohn Company, Kalamazoo, Mich.

[5]Predef 2X, Upjohn Company, Kalamazoo, Mich.

TABLE II

GROSS APPEARANCE AND QUANTITY OF SYNOVIAL EFFUSIONS

Horse No.	Affected tarsal synovial sheath	Gross appearance	Quantity (ml/tarsal synovial sheath)
1	Right	Pale yellow, clear	140.0
2	Left	Amber-opaque	50.0
3	Left	Amber-clear	30.0
4	Left	Pale yellow, clear	110.0
5	Right	Amber-opaque, some flocculent material	10.0
6	Left	Amber-opaque	24.0
7	Right	Pale yellow, clear	70.0
8	Left	Pale yellow, clear	250.0
Mean			85.5
S.E.			27.9

S.E. = Standard error of the mean.

TABLE III

RELATIVE VISOCITY, MUCINOUS PRECIPITATE QUALITY, TOTAL PROTEIN CONTENT, AND ALBUMIN: GLOBULIN RATIO OF SYNOVIAL EFFUSIONS

Horse No.	Relative viscosity (at 37 °C)	Mucinous precipitate quality*	Total protein (Gm/100 ml)	Albumin: globulin ratio
Affected horses				
1	1.32	Fair	....	....
2	1.57	Fair	....	....
3	2.54	Poor	....	....
4	2.07	Fair	1.25	7.33
5	2.16	Very poor	4.00	1.86
6	6.47	Very poor	2.29	1.06
7	1.81	Poor	1.70	0.83
8	1.59	Poor	2.73	2.21
Mean	2.44		2.39	2.66
S.E.	0.59		0.47	1.20
Control horses				
1	3.55	Normal	1.04	5.50
2	3.91	Normal	1.57	0.31
3	2.61	Normal	1.39	1.62
4	4.54	Normal	1.54	8.06
5	3.97	Normal	1.37	1.58
Mean	3.72		1.38	3.41
S.E.	0.32		0.10	1.45

S.E. = Standard error of the mean.

*Mucinous precipitate quality: normal = tight, ropy clump in a clear solution; fair = soft mass in a slightly turbid, xanthochromic, or amber solution; poor = small, friable masses in a turbid, xanthochromic, or amber solution; and very poor = few flecks in a very turbid, xanthochromic, or amber solution of 7 N glacial acetic acid (2.5%).

pitate quality ranged from fair (soft mass in a slightly turbid, xanthochromic, or amber solution) to very poor (few flecks in a very turbid, xanthochromic, or amber solution) for synovial effusions from affected tarsal synovial sheaths. All synovial fluid samples from the tibiotarsal joints of control horses were normal (tight, ropy clump in a clear solution). Decreased concentration and loss of polymerization in the hyaluronic acid moiety in most synovial effusions was denoted by formation of small, friable precipitates when 1 ml of synovial effusion was added to 4 ml of a 2.5% aqueous solution of 7 N glacial acetic acid (12).

Results of total protein determinations and A:G ratios partially confirmed the transudative properties of synovial effusions (Table III). The mean total protein content in synovial fluid from control horses (1.38 Gm/100 ml) was slightly less than in synovial effusions from affected horses (2.39 Gm/100 ml). The mean A:G ratio (3.41) was slightly higher in synovial fluid from control horses than the mean A:G ratio (2.66) in synovial effusion from horses affected with tenosynovitis of the deep digital flexor tendon.

Serum and synovial effusion sugar content (measured as total reducing substances) were determined simultaneously (Table IV). The sugar content in synovial effusions ranged from 97 mg/100 ml less than serum sugar content to 18 mg/100 ml greater than serum sugar content. The mean synovial effusion sugar content in affected horses was 40 mg/100 ml less than their mean serum sugar content. Mean synovial effusion sugar content in horses affected with tenosynovitis of the deep digital flexor tendon was 29% less than their mean serum sugar content. This difference was significant ($P < 0.05$). The mean sugar content in synovial effusion from tarsal synovial sheaths was significantly ($P < 0.05$) less than in synovial fluid from tibiotarsal joints. The sugar content in synovial fluid from tibiotarsal joints of control horses ranged from 32 mg/100 ml less than sugar content in serum to 12 mg/100 ml greater than sugar content in serum. There was a mean synovial fluid sugar difference for control horses of 9 mg/100 ml less than their mean serum sugar content. Mean synovial fluid sugar content in control horses was 4% less than their mean serum sugar content, which was not significant ($P > 0.10$). These differences in sugar content established a serum:synovial effusion sugar ratio of 1.4:1 for horses affected with tenosynovitis of the deep digital flexor

TABLE IV

COMPARISON OF SERUM AND SYNOVIAL EFFUSION SUGAR CONTENT

Horse No.	Serum sugar (mg/100 ml)	Synovial sugar (mg/100 ml)	Serum: synovial sugar difference (mg/100 ml)*
Affected horses			
1	218	162	<56
2	100	105	> 5
3	105	32	<73
4	109	57	<52
5	...	121	....
6	64	82	>18
7	94	69	<25
8	171	74	<97
Mean	123	88**	<40
S.E.	20	14	16
Control horses			
1	99	67	<32
2	103	105	> 2
3	65	69	> 4
4	107	100	< 7
5	138	150	>12
Mean	102	98	< 9
S.E.	12	15	6

S.E. = Standard error of the mean. *Synovial sugar content less than or greater than the simultaneously determined serum content. **Significantly ($P < 0.05$) less than corresponding serum sugar content.

tendon. Serum:synovial fluid sugar ratio for control horses was 1:1.

Total erythrocytic counts ranged markedly from one synovial effusion to another (Table V). There was a close relationship between gross appearance of the synovial effusion and the total erythrocyte count (*i.e.*, the highest erythrocytic counts were in amber and clear or amber and opaque synovial effusions). No erythrocytes were present in synovial fluid from the tibiotarsal joints of control horses. Total leukocytic counts did not vary as markedly as total erythrocytic counts (Table V). Leukocytic counts for synovial fluid from control horses were low. Increased relative numbers of neutrophils were associated with increased numbers of erythrocytes. Numerous laked erythrocytes were noted on synovial effusion smears. Many degenerated leukocytes and a few synovial intimal cells were noted, but excluded from both total and differential leukocytic counts.

Bacteriologic Findings

The two synovial effusion samples submitted for bacteriologic studies were negative on culture (Table I, horses 5 and 8).

Discussion

Tenosynovitis of the deep digital flexor tendon in horses was manifested by marked distention of its tarsal synovial sheath due to formation and retention of an excessive transudative synovial effusion. Distention of the tarsal synovial sheath of the deep digital flexor tendon can be confused with tarsal hydrarthrosis (bog spavin) when the medioplantar and lateroplantar pouches of the tibiotarsal synovial sac are visibly distended (1, 6, 16). Distention of the tarsal synovial sheath is generally greatest on the medial aspect of the tarsus. In contrast to the location of the medioplantar pouch of the tibiotarsal synovial sac, the tarsal synovial sheath of the deep digital flexor tendon extends 5 to 7 cm proximal to the medial malleolus of the tibia and 5 to 7 cm distal to the proximal end of the third metatarsal bone (10). Inflammation of the large bursa interposed between the gastrocnemius and superficial digital flexor tendons (intertendinous calcaneal bursitis) can also present a problem in differential diagnosis (17).

The pathogenesis of tenosynovitis of the deep digital flexor tendon wherein excessive transudative synovial effusions are formed and persist in its tarsal synovial sheath is a poorly understood syndrome in the horse (1, 6, 8). Inflammation of the tendon of the deep digital flexor can and does occur in conjunction with inflammation of its tarsal synovial sheath. Acute tenosynovitis can be caused by tendon strain, friction between the parietal and visceral layers of the tarsal synovial sheath, peritendinous pressure, and acute direct trauma to the tendon of the deep digital flexor and its tarsal synovial sheath (2). Acute tenosynovitis of the deep digital flexor tendon is manifested by rapid filling of the tarsal synovial sheath, heat, pain, and lameness (1). Acquired hypersensitivity to an unknown foreign agent or agents can precipitate acute tenosynovitis (8).

TABLE V

Cytologic Properties of Synovial Effusions

Horse No.	Erythrocytes (per cmm)	Leukocytes (per cmm)	Neutrophils (%)	Lymphocytes (%)	Monocytes (%)	Macrophages (%)	Eosinophils (%)
Affected horses							
1	0	175	7	73	13	7	0
2	8,000	500	30	20	50	0	0
3	3,000	600	52	1	44	1	2
4	0	44	3	85	12	0	0
5	20,500	367	61	27	12	0	0
6	21,500	200	8	65	22	1	4
7	111	311	0	95	5	0	0
8	1,975	511	5	93	2	0	0
Mean	6,886	339	20.8	57.4	20.0	1.1	0.8
Control horses							
1	0	44	0	30	64	6	0
2	0	144	2	52	40	6	0
3	0	78	4	64	26	6	0
4	0	78	0	82	18	0	0
5	0	67	0	66	31	0	3
Mean	0	82	1.2	58.8	35.8	3.6	0.6

Chronic tenosynovitis may follow the acute condition or develop from trauma that is multiple and minor. It is possible that repeated subclinical strain of the tendon of the deep digital flexor or inapparent trauma could develop into chronic tenosynovitis. Chronic tenosynovitis is manifested by a fluctuant distention of the tarsal synovial sheath due to continued formation and retention of an excessive synovial effusion. Fibrosis of the parietal layer of the tarsal synovial sheath further enhances chronicity. Tenosynovitis of the deep digital flexor tendon can develop in an insidious manner (8). Horses in this investigation developed an insidious form of tenosynovitis without signs of inflammation. In the absence of an accurate history, it proved impossible to distinguish between chronic tenosynovitis and the insidious form of tensynovitis. A low degree of tarsal joint angulation (*i.e.* too straight in the tarsal joint) can predispose horses to tenosynovitis of the deep digital flexor tendon (9).

Aspiration of synovial effusions and intrathecal injection of synthetic steroids provided a palliative means of effectively controlling production and retention of excessive synovial effusions, and reducing fibrosis in tarsal synovial sheaths. The mechanism is unknown whereby adrenocortical steroids or their synthetic analogues suppress formation and retention of excessive transudative synovial effusions (13, 14). Adrenocortical steroids suppress inflammation without regard to etiology or type (*i.e.* suppurative or nonsuppurative) (2, 11). Chronic tenosynovitis or the insidious form of tenosynovitis did not respond rapidly to the intrathecal action of synthetic steroids. Intrathecal injection of 6 α-methyl, 17 α-hydroxyprogesterone acetate suppressed formation of synovial effusions and revealed a prolonged duration of action when compared to 6 α-methylprednisolone acetate. These data reflected either an intrathecal depot effect of 6 α-methyl, 17 α-hydroxyprogesterone acetate or a delayed rate of tissue inactivation. The addition of 9-fluoroprednisolone acetate to 6 α-methyl, 17 α-hydroxyprogesterone acetate in a 1:10 ratio hastened this antitransudative effect without interfering with its prolonged duration of activity. These results indicated that 6 α-methyl, 17 α-hydroxyprogesterone acetate possesses features characteristic of 11 β-hydroxylated, C_{21} acetate steroids (3, 4). Addition of the 6 α-methyl group to the progesterone molecule enhances its biologic activity. Apparently oxygen functions at C-11, C-17, and C-21 are not obligatory for adrenocortical steroid activity (4).

Tenosynovitis implies inflammation of the synovial membrane lining the tarsal synovial sheath as opposed to its fibrous outer layer; however, the fibrous layer is usually affected (6). Inflammation is not a prerequisite for formation and retention of excessive transudative synovial effusions in tarsal synovial sheaths (8). Synovial effusions from all eight horses in this investigation were chronic at the time of aspiration; however, these synovial effusions reflected no inflammatory changes in the synovial membranes lining these tarsal synovial sheaths. Chronic tenosynovitis and insidious forms of tenosynovitis of the deep digital flexor tendon were indistinguishable, both clinically and by synovial effusion analyses. The primary difference between the two forms of tenosynovitis being one of etiology, viz. chronic tenosynovitis of the deep digital flexor tendon followed the acute form of tenosynovitis, whereas the insidious form of tenosynovitis developed without a history or signs of previous injury or inflammation. Both forms of tenosynovitis were characterized by retention of self-perpetuating transudative synovial effusions. Pathologic changes in synovial effusions of chronic tenosynovitis were similar to those in tarsal hydrarthrosis in horses, wherein excessive transudative synovial effusions develop and persist (16).

Analysis of synovial effusions and interpretation of findings can provide an excellent means of differentiating between acute and chronic forms of tenosynovitis (11). If infection is suspected, bacteriologic cultures of the synovial effusion should be performed. Since little information is available on normal synovial fluid from tendon sheaths, it must be assumed that it is analogous to the synovial fluid of diarthrodial joints. Pathologic changes in joint diseases may therefore have a corollary in tenosynovitis. Synovial xanthochromia was most pronounced in amber synovial effusions due to continued intrathecal hemorrhage and subsequent hemolysis. Grossly, pale yellow synovial effusions from tarsal synovial sheaths most closely resembled normal synovial fluid from the tibiotarsal joints of control horses.

Relative viscosity of synovial effusions (RV = 2.44) from tarsal synovial sheaths was lower than the relative viscosity of synovial fluid (RV = 3.72) from the tibiotarsal joints of control horses. Despite fair, poor, or very poor mucinous precipitates (indicative of reduced hyaluronic acid concentration and polymerization), synovial effusions maintained sufficient viscousness to ameliorate the deleterious effects of friction within tarsal synovial sheaths. Highly polymerized hyaluronic acid in synovial fluid is not necessary for normal joint or tendon

sheath lubrication (7). The proteins of blood have no lubricating properties; however, a protein moiety forms an intrinsic component of the hyaluronic acid-protein complex. This protein moiety may serve as a prosthetic group for adsorption of mucin onto a moving surface (*i.e.* the articular cartilages or synovial membranes lining bursas and tendon sheaths). Adsorption of hyaluronic acid to synovial intimal cells lining tendon sheaths forms an essential prerequisite for boundary lubrication.

Serum:synovial effusion sugar ratio was 1.4:1 in affected horses, compared to the 1:1 serum:synovial fluid ratio in control horses. Sugar levels in synovial effusions suggest that the equilibrium between a bursa and blood differs in comparison to the sugar equilibrium regulated by the blood-joint barrier (11). Low sugar levels in the absence of bacteria or marked leukocytosis are unusual in nonsuppurative synovial effusions in joints. The rate of sugar utilization by tendon sheaths and bursas apparently differs from that of joints. Articular cartilages in diarthrodial joints may require higher levels of readily available sugar in the synovial fluid.

Erythrocytic and leukocytic counts in synovial effusions from horses affected with tenosynovitis of the deep digital flexor tendon were increased in comparison to erythrocytic and leukocytic counts in synovial fluid from the tibiotarsal joints of control horses. Amber synovial effusions had the highest erythrocytic and leukocytic counts. Increased numbers of leukocytes either gained entrance to the tarsal synovial sheath when hemorrhage occurred or migrated into tendon sheaths in response to irritation arising from the products of erythrocytic destruction (*i.e.* bilirubin and hemosiderin) (11).

Summary and Conclusions

Tenosynovitis of the deep digital flexor tendon was manifested by distention of its tarsal synovial sheath due to formation and retention of an excessive transudative synovial effusion. Tenosynovitis of the deep digital flexor tendon was chronic at the time of clinical examination. Tendon strain, peritendinous pressure, trauma, and intrathecal friction were considered predisposing causes. Absent were signs of inflammation, as manifested by heat and pain, and lameness. The cause of an insidious form of tenosynovitis of the tarsal synovial sheath of the deep digital flexor tendon was undetermined. Synovial effusions were analyzed for their physical, biochemical, and cytologic properties. Pathologic changes in synovial effusions were minor.

Therapeutic benefit was derived from aspiration of all available synovial effusion. Intrathecal injection of 6 α-methylprednisolone acetate and 6 α-methyl, 17 α-hydroxyprogesterone acetate provided a palliative means of suppressing formation and retention of excessive effusions. Results indicated that 6 α-methyl, 17 α-hydroxyprogesterone acetate had a prolonged duration of antitransudative activity and possessed features characteristic of 11 β-hydroxylated, C_{21} acetate steroids. Clinical response to intrathecal injection of synthetic steroids was manifested by resolution of excessive synovial effusion and concomitant reduction in distention of tarsal synovial sheaths. A palpable peritendinous fibrosis persisted to some degree in all horses.

Résumé

Une inflammation de la synoviale du fléchisseur profond des phalanges s'est manifestée par une distention de la gaine tarsienne causée par l'accumulation de synovie. Au moment de l'examen clinique, l'inflammation était chronique. On considérait comme causes prédisposantes les efforts et les pressions sur le tendon, ainsi que les trauma et les frictions à l'intérieur de la gaine tendineuse. On n'a observé aucun signe d'inflammation tel que la chaleur, la douleur ou une boiterie. La cause de l'affection n'a pas été déterminée. Une analyse physique, biochimique et cytologique du liquide synovial n'a révélé que des changements pathologiques mineurs.

L'aspiration de tout le liquide synovial possible a apporté une certaine amélioration. On pallia à la surproduction de synovie en injectant dans la gaine affectée de l'acétate de 6-x-methylprednisolone et de l'acétate de 6-x-méthyl, 17-x-hydroxyprogesterone. Les résultats prouvèrent que cette dernière substance a une action prolongée sur la sécrétion de la synovie et, en outre, qu'elle possède des caractéristiques des acétates C21 de stéroides, 11-B-hydroxylés. L'injection de stéroides de synthèse provoqua la disparition de l'excès de synovie et, conséquemment, une diminution de la distension de la gaine tarsienne. Une fibrose péritendineuse, décelable à la palpation, persista chez tous les chevaux.

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BOOK REVIEW

Animal Agents and Vectors of Human Disease. Third Edition. E. C. Faust, P. C. Beaver and R. C. Jung. Published by Lea & Febiger, Philadelphia and the Macmillan Company of Canada, Toronto. 1968. 461 pages. Price $12.50.

This is the newly revised edition of an established text written principally for medical and public health workers. Although it is not a text book of veterinary parasitology, it does hold some interest for workers in the field of veterinary epidemiology. It is a rather comprehensive volume, covering general principles of parasitology followed by sections on Protozoa, Helminths and Arthropods. A further section, headed "Technical Aids" covers procedures useful in the diagnosis of parasitic diseases. Full author and subject indices are included.

As would be expected from a revised edition, there are few typographical errors. The spelling of "species" on page 322 happens to be one noted. The illustrations are generally excellent, including the grouped set of color plates of amoebae and *Plasmodium spp.* One or two drawings are less useful, for example, a line drawing of a larval ascarid on page 232.

Of the various sections, it is felt that Section IV (Arthropods as Agents and Vectors) might be rearranged slightly to arrive at a more systematic subdivision of headings. In this same section there are one or two common names of insects given which differ from those usually attached to the species.

These, however, are very minor items, and the book remains a most useful volume which serves as an excellent reference in the field of human parasitology. *D. P. Gray.*