بعض التغيرات الباثولوجيه والكيميائية فى الأغنـــام
بعـــد انفجـار المثانـة البوليـة

ح • ســـالم ، أ • عامـــر ، ن • مسك ، ط • العـــلاوى

استهدفت الدراسة معرفة التغيرات الباثولوجيه فى أنسجة الأغنام وكذلك التغـــيرات الكيميائية المحتملة فى دم وسيرم هذه الحيوانات بعد انفجار المثانة البولية تجريبيـــا • ولقـــد أظهرت الدراسة مايلى :ـ

١ـ حدوث مواضع التهاب فى أنسجة القلب مصحوبة بتحلل وموت للخلايا العضلية ، وكذلـك أنابيب الكلى وكرات ملبيجى لهـــذه الكلى •

٢ـ ظهور تحللات دهنية حول الوريد المركزى فى فصيات الكبـد ، مع التهاب فى أنسجـة الأمعـــاء •

٣ـ زيـادة مضطردة فى مستوى اليوريا والكرياتينين فى الدم ومســتوى الفوسفور الغـــير عضوى والبوتاسيوم فى سيرم الدم لهذه الحيوانات وكانت الزيادة مرتبطه بالوقت الـــذى مر بعد اجراء العملية وحتى النفوق ، بينما سلك مستوى الصوديوم عكس ذلك •

٤ـ كانت نتائج تحليل البول المتجمع فى تجويف البطن مطابقة لما حدث فى الدم •

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SOME HISTOPATHOLOGICAL AND BIOCHEMICAL STUDIES ON RAMS AFTER EXPERIMENTAL RUPTURE OF THE URINARY BLADDER

(With 2 Tables and 4 Figures)

By

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SUMMARY

Experimental rupture of the urinary bladder was conducted in four rams. Blood, urine and abdominal fluid samples were collected before and at regular intervals following the operation till death. Besides, heart, liver and kidney tissue samples were examined microscopically. The study revealed:-

1- Small few foci of fibrinous inflammation with degeneration in the myocardium, tubular nephrosis with some glamerular changes and centrilobular hepatic necrosis with congestion of the intestine.

2- Progressive elevation of blood urea nitrogen, non-protein nitrogen, serum creatinine serum inorganic phospharus and serum potassium level in relation to time post-operation. Serum sodium and chloride levels behaved on the contrary. Similar data were achieved in urine and abdominal fluid samples.

INTRODUCTION

Rupture of the urinary bladder as a result of urolithiasis constitutes an important economic problem in our domestic farm animals. The condition has been reported in cattle (O'CONNOR, 1974) and sheep (WEAVER, 1969). Other animals such

as dog and horses are also liable to this contion (COLIET et al. 1966 and REWLINGS, 1969). Studies on physiopathological effects (ADO and ISHIOMOVA, 1973, LAVANIA, MISRA and ANGELO, 1973 and others) are sufficiently decomented yet little informations are avaliable on pathological changes under such conditions.

The present work was carried out to investigate the pathological changes in rams with experimental rupture of the urinary bladder.

MATERIALS AND METHODS

A number of four adult rams were used in this study. The abdomen was surgically opened under aseptic conditions and urinary bladder was incised. Full details concerning preparation of animals as well as technique of bladder rupture was reported by ALLAWY et al. (1978).

Whole anticoagulated blood and blood samples were collected before operation (B.O.) and every 12 hrs. post-operation (P.O.) up to the first 72 hrs. then every two days till death. Abdominal paracentasis was performed at 24 & 48 hrs. P.O. to collect abdominal fluid. Post-mortem examination was carried immediately after death. Accumulated peritoneal fluid was measured. Tissue samples were fixed in 10% buffered formalin solution embeded in paraffin, cut at 6 U,stained with H&E stains and examined microscopically. Whole anticoagulated blood samples were used for determination of blood urea nitrogen(B.U.N) and non-protein nitrogen (N.P.N.) as described by RAITASKA, (1970). The gained serum was analysed for creatinine (FOLIN and WU, 1920), chlorides (SCHALES and SCHALES,1941) sodium and potassium (EEL-photometer) and inorganic phosphorus (ANTOROVA

and PLINOVA, 1971). Colleted abdominal fluid was examined physically and its biochemical constituents were analysed on the same basis adopted in blood samples.

RESULTS

Three animals died after 1,3&21 days. The fourth was sacrificied after 22 days p.o.

Pathological Findings:

Morphological changes either grossely or microscopically were more or less the same without any significant differences which can be related to the period of survival after operation.

Grossely the carcasses were somewhat debilitated. There was a nasal discharge with congested m.m. The mucosa of the buccal cavity was pale and had a somewhat ammoniacal odor. The abdominal walls were rigid from each side. The wool was easily detached and comparatively rough. Ammoniacal smell appeared directly after opening the skin.

Subcutanoeus gelatinous change was found in some parts of the pale abdominal wall. Haemorrhagic patches in some areas of peritoneal surface were observed. In the abdominal caivty there was an accunulation of fluid which had a characterstic of odor of urine and coagulated directly after exposure to air. In addition diffuse congestion of blood vessels of the whole serosa of the digestive system was present.

The texture of the liver was friable and greasy.The liver was apparently enlarged and had a bulging character of the cut surface with oozing of blood.

The abomasum showed a thick oedematus wall with the presence of haemorohagic patchs in the mucosa. Its content was more fluidy and yellowish in color. The same picture was observed in the duodenum. The centent of the large intestine was more fluidy and the serosal blood vessels were more congested.

The kidneys revealed gelatinous transformation of the perinephric adipose tissue together with the superficial petecheal haemorrhages. Their texture was more softer than normal and on longtidunal cut section, they showed pale areas in the cortex and dilated pelvis which contianed urine mixed with gelatinous mass. The urinary bladder of three rams showed superficial haemorrhagic patches. Slight congestion was shown in the ureters.

Opening of the chest cavity showed congested thoracic muscles with the presence of a bloody serous fluid in the cavity larynex, trachea and bronchi had congested blood vessels. All the lobes of the lung were rigid, enlarged and uniformely discoloured purplish red. On cut section, frothy bloody fluid oozed by pressure. Faetures of oedema and congestion of broncheal and mediastinal lymph nodes were also present.

The heart was enlarged than normal and its muscles were flabby. Opening of its chambers showed clotted blood in the left ventricle and pulmonary artery. There were petecheal haemorrhages subendocardially especially in the left ventricle.

Microscopically, the lung revealed changes related to slight cattarrhal bronchitis. Destruction of broncheal epithelium with the presence of plasma cells, lymphocytes and macrophages in broncheal lumen were frequently observed. (Fig. 1). Hyperaemia of the cappillaries surrorunding the alveoli was

accompanied by occasional neutrophelic infilteration.Sometimes hyperplasia and increased activity of broncheal epithelium occured (Fig. 2). Small amounts of serofibrirous exudate with the presence of some red cells was observed in few alveoli. Areas of collapse surrounded by areas of compunsatory emphysema was a common finding. Oedema of the interstitial tissue of the lung was prominant.

In the heart, features of proteineous dystrophy of the heart nuscles fibres which appeared granular and swollen together with some haemorrhages were present.

In the kidney some tubules showed epithelial necrobiotic changes while others were dilated with flattened epilhelial lining. Scattered ruptured tubules were also observed. Glomerular changes in the form of widely dilated Bowman's capsules with shrinked glomerular tuft in some parts and severe congestion in others were found.

The liver showed centrolobular necrobiosis with well developed fatty change and lymphocytic infilteration in the portal areas,sometimes thrombosis in the central and portal veins and hepatic artery was observed (Fig. 4).

Necrobiotic changes appeared in lymphocytic cells of the follicles of the spleen.

Patchy necrosis and desquamation of the epithelial lining of the villi in the abomasum were observed while the submucoa revealed congested blood vessels with lymphocytic and neutrophilic accumulations. The intestinal mucosa also showed erosions and focal necrosis of the villar epithelium.

Blood and Serum changes:

Blood urea nitrogen, N.P.N. and serum creatinine levels were increased directly at the first 12 hrs post-operation. This increase continued and was directly proportional to the time the animal survived (Table 1).At times od death(24 hrs-22 days) B.U.N. level reached 57.9-92.3 mg%. and serum creatinine level ranged from 0.80-4.50 mg%.

Serum sodium level in experimental animals decreased proportionally to the time the animal survived. Pre-operative sodium level amounted 150.3-190.2 mEq/l while hyponatraemia was evident an time of death (84.2-140.5 mmEq/l).

Table (1) showed that serum chloride level decreased in rams P.O. in comparison to pre-operative levels. At time of death serum chloride level ranged from 70.0-85.8 mEq/l while its level at the pre-operative time constituted 108.2-130.3 mEq/l. Such decrease of chloride level was paralled to the decreased level of sodium in post-operative period.

Serum potassium level was markedly increased by the time elapsed after operation in comparison with levels of pre-operative ones. At time of death serum potassium level reached 5.7-11.3 mEq/l instead of 3.9-5.0 mEq/l at pre- experimental time.

A relatively elevated serum inorganic phosphorus level was detected at the first 12 hrs. P.O. (5.1-5.5 mg%) . More higher levels (up to 8.8 mg%) were observed at time of death. (Table 1).

Abdominal Fluid Examination:

The colour of the abdominal fluid, obtained by paracentasis of living rams and that observed at necropsy was generally, pale yellow to yellowish but pink in only one ram. It ocagulated and became gelationus soon after exposure to air in two rams. Turbidity varied from slight to obvious. The amount of the abdominal fluid cellected at necropsy, ranged from 3.00-7.25 litres with a pH ranged from 6.5-7.9 and specific gravity from 1.012-1.020.

Table (2) presented biochemical constituents of abdominal fluid during the experiment till death. General increase of urea nitrogen, N.P.N. and creatinine levels in abdominal fluid was evident either at 36 hrs.P.O. or at time of death. At that time recorded levels were 100.2 mg% for N.P.N., 80.4 mg% for urea nitrogen and 4.8 mg% for creatinine instead of 28.1, 16.2 and 0.4 mg% respectively before operation. At time of death chloride concentration reached 80.0 mEq/l while sodium and potassium levels were 270.3 and 21.8 mEq/l respectively while inorganic phosphorus level reached 17.3 mg%.

DISCUSSION

Pathological lesions observed in heart, kidneys and liver consisted mainly of degenerative changes which can be attributed to the toxic effect of increased urea level in blood or the liberation of ammonia from the present urea. Lesions in the kidneys and heart are comparable to the lesions that have been reported by other investigators in cases of uraemia following chronic renal failure.

Patchy necrosis in renal tubules was recorded by CAPPELL and ANDERSON (1975) in humans while degenerative changes have been described ANDERSON and SCOTTI,(1968) in the outer portion of the myocardium. The presence of sero-fibrinous exudate into the alveolar tissue of the lungs was described by CAPPELL and ANDERSON, (1975) and WALTER and ISRAIL, (1961) while pulmonary haemorrhage was reported by ANDERSON and SCOTTI, (1968). Haemorrhagic alterations and pseudonembranous enterocolitis, observed by GAPPELL and ANDERSON, (1975), in uraemic persons are more or less similar to our results. The aetiology of gastero-intestinal inflammation was beleived to be due to the decomposition of urea by gasterointestinal microflora which results in liberation of ammonia that irritate the mucous membranes.

The biochemical results in the present study are not different from those reported by earlier investigators. RAO et al. (1972) stated that the blood urea nitrogen level ranged between 76-190 mg% at time of death of ox with experimental rupture of the urinary bladder. Similarily WEAVER, (1969) considered that B.U. values of 150 mg% or more generally indicated a ruptured bladder in sheep. EL-SHEIKH and SHOKRY, (1973) and RAO et al. (1972) mentioned that B.U.N. exceeded the phyiological levels in ruptured bladder in ox.

Hyponatraemia observed here,has explained by COLES,(1974) on the basis that in renal diseases sodium concentration mechanism was operating defeciently beacuse of tubular damage. Hyperkalaemia my occur with renal failure,particularily acute, especially if accumpanied by anuria and oliguria (COLES,1974). It is accepted also that serum potassium levels does not reflect the degree of uraemia, however deviations beyond the

normal levels goes side by side with B.U.N. elevation (BIRZA, 1961). The relatively elevated serum phosphorus level observed here, was similar to those observed by HOE et al. (1965) and SMART et al. (1972). These authors demonstrated that in renal insuffeciency the rise in serum phosphorus level seems to be parallel to that of urea.

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Table (1): Blood and Serum Biochemical Changes In Rams With Experimental Rupture of Urinary Bladder.

Indices	Anim. No	Pre- operative	Time Post - Operation														
			12hrs	24hrs	36hrs	48 hrs	60hrs	72hrs	5 days	7 days	9 days	11 days	13 days	15 days	17 days	19 days	21 days	22 days
Blood Urea Nitrogen %	1	44.3	45.5	57.9														
	2	37.9	47.2	59.2	67.7	80.0	86.0	92.3										
	3	35.6	39.7	40.7	46.6	47.6	47.9	50.8	52.0	54.3	62.6	67.2	80.2	86.2	86.0	85.8	86.8	
	4	20.7	27.3	29.3	36.4	37.9	47.4	59.3	64.4	67.8	67.3	70.2	73.3	75.4	76.0	75.8	76.0	78.0
Blood Non-protein Nitrogen (B.N.P.N.) mg%	1	66.2	65.6	77.8														
	2	57.8	67.2	78.2	87.8	100.0	106.2	112.4										
	3	55.6	59.8	60.8	66.6	67.8	70.8	72.0	74.4	82.6	87.2	100.2	106.0	105.8	106.0	105.0	105.8	
	4	30.4	47.7	50.7	58.2	59.7	59.0	66.7	78.6	82.2	83.9	83.7	96.1	96.7	97.0	97.3	97.9	98.0
Serum Creatinine mg%	1	0.75	0.79	0.80														
	2	0.80	0.83	0.85	0.95	1.00	1.50	1.80										
	3	0.60	0.96	1.00	1.10	1.20	1.50	1.80	2.20	2.60	2.80	3.00	3.30	3.40	4.00	4.10	4.50	
	4	0.60	0.90	1.00	1.20	1.30	1.50	2.00	2.20	2.40	2.60	2.80	3.40	3.50	3.70	4.00	4.20	4.30
Serum Potassium mEq /1	1	5.0	5.6	5.7														
	2	4.3	4.8	7.8	8.8	8.5	8.6	8.9										
	3	4.0	4.5	4.8	4.9	5.1	5.5	5.9	6.2	6.3	8.3	8.7	8.7	9.7	10.1	10.8	11.3	
	4	3.9	4.0	4.2	4.6	5.0	5.1	5.5	5.8	6.0	6.0	6.5	7.3	7.5	7.8	8.0	8.1	8.0
Serum Sodium mEq /1	1	190.2	160.5	140.5														
	2	150.0	140.2	130.3	126.0	122.0	122.0	16.0										
	3	150.5	135.0	130.7	120.8	116.7	112.5	10.6	110.5	90.2	110.0	110.6	100.5	90.7	90.7	90.1	90.1	90.2
	4	150.3	130.3	126.0	120.5	114.1	110.2	99.8	95.9	90.9	90.7	87.5	86.3	82.6	80.0	84.6	86.6	84.2
Serum chloride mEq /1	1	110.2	90.6	85.8														
	2	119.8	104.4	102.2	126.0	95.4	85.4	80.0										
	3	130.3	110.8	98.3	95.3	93.0	90.6	87.1	85.0	75.7	75.0	75.0	72.4	71.0	70.8	70.6	70.0	
	4	108.2	103.8	100.8	97.3	95.3	94.0	92.0	90.8	85.4	80.4	80.8	80.3	80.0	80.2	80.5	80.0	78.0
Serum Inorganic Phosphorus mg%	1	5.0	5.5	5.8														
	2	4.5	5.0	5.2	5.8	6.0	6.8	7.2										
	3	5.0	5.1	5.3	5.5	5.5	5.8	6.1	6.2	6.4	7.1	7.3	7.6	7.6	7.8	7.8	7.9	7.9
	4	5.2	5.4	5.6	5.9	6.1	6.2	6.2	6.6	6.9	6.9	7.8	7.9	8.0	8.3	8.5	8.6	8.8

Table 2. Abdominal Urine Constituents.

Time of Sampling / Indices	Before operation (Urine Sample)	36 hrs. P.O.	After death
Urea Nitrogen mg%	16.2	50.5	80.4
N.P.N. mg%	28.1	80.4	100.2
Creatinine mg%	0.4	3.0	4.8
Potassium mEq/l	3.8	8.9	21.8
Sodium mEq/l	100.2	180.6	270.3
Chloride mEq/l	62.4	73.8	80.0
I. P. mg%.	3.1	5.8	17.3

N.B. Figures stated here represents average of data for four animals.

Fig. (1): Alveolar septa with evidence of cellular reaction (40 X 10).

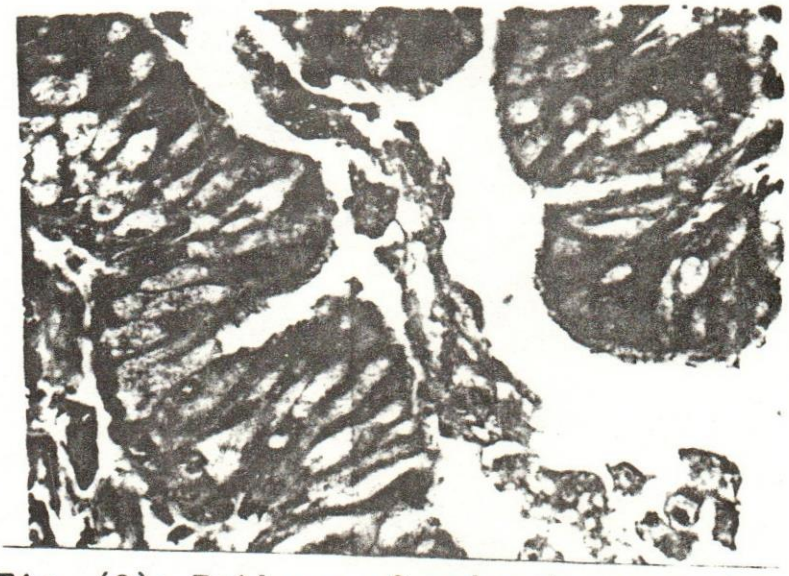

Fig. (2): Evidence of moderate secrotory activity of branceal epithelium (40 X 10).

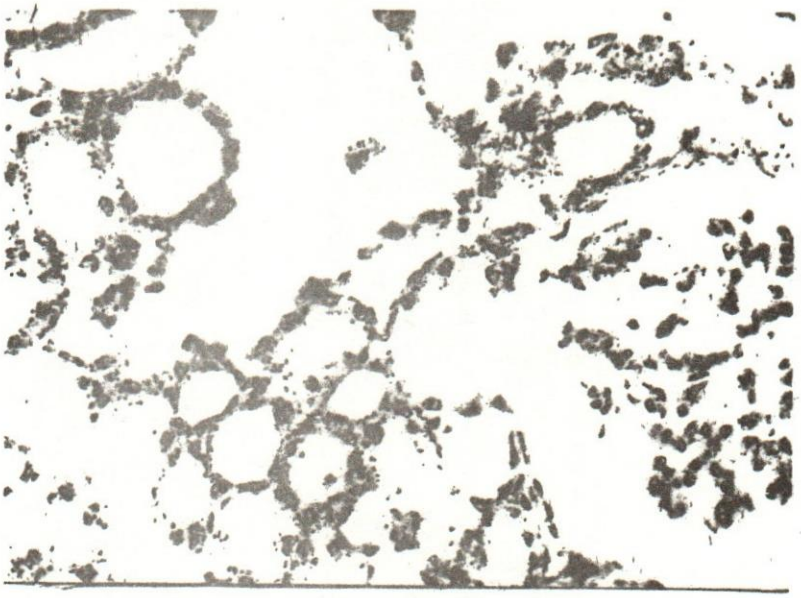

Fig. (3): Kidney showing necrobiotic changes in tubular epithelium with obvious diltation of the Bowmann's capsules (40 X 10).

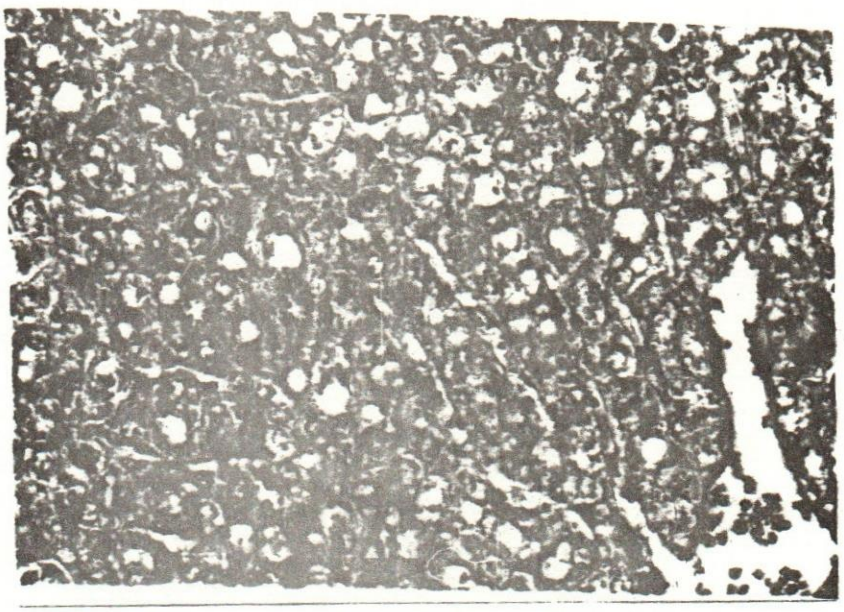

Fig. (4): Liver showing fatty change. (40 X 10).