Biochemical Abnormalities In Broilers And Native Saso Chicken Serum After Induced Infectious Runting And Stunting Syndrome (IRSS)

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ABSTRACT

Thirty broilers and forty five native saso chicks, were orally inoculated immediately after hatching with intestinal homogenate from field cases suffering from IRSS. Another 10 broilers and 15 native saso chicks served as negative control. Sera samples were collected at 7, 14, 21 and 28 day post inoculation (PI). Total protein, albumen (ALB), alkaline phosphatase ALP, alkaline phosphatase after heat inactivation, calcium, phosphrous, cholesterol and triglycerides were determined, while globulin (G) and ALB/G ratios were calculated

Significant increase in alkaline phosphatase ALP (P<0.05) was recorded in both inoculated chicken breeds. Heat inactivation of serum at 56°C for 30 min had led to a significant reduction (P<0.05) in ALP activity this refers that the increased ALP activity was from osseous source which contradicts the previous findings indicating that ALP in the coarse of (IRSS) was from enteric source. Total proteins were significantly reduced (P<0.05) (PI) during the entire experimental period in both chicken breeds, this reduction was the result of significant reduction (P<0.05) in globulin. Cholesterol and triglyceride increased significantly (P<0.05) in both chickens breeds (PI) during the experimental period. Calcium level was only significantly reduced (P<0.05) 7 days (PI) and remained with non significant changes at 14, 21 and 28 days in broilers and also was non significant at 7, 14, 21 and 28 days (PI) in native saso chickens. Phosphorous was not affected significantly (PI) in both breeds during the experimental period. so they should be disregarded in the interpretation of bone brittleness in the coorse of IRSS. The constellation of these biochemical abnormalities beside the previously reported insulin deficiency after induced (IRSS) is a sure indication for diabetes mellitus . This can interpret some of the observed symptoms and post mortem findings seen in the coarse of (IRSS) such as, (increased feed consumption, reduced body weight, excessive water consumption malaise, lethargy , distended flaccid intestine causing abdominal distension, bone changes , and also can explain previously recorded brain lesion) .

INTRODUCTION

Managing disease abnormalities requires an understanding of how disease process changes the biochemical functions of the body, thus clinical chemistry parameters are necessary to evaluate the cellular changes *(1)*.

Reviewed pathogenesis of IRSS had set four pathogenic event in the coarse of IRSS, these are "proventricular and intestinal pathology, hypothyroidism, pancreas and pancreatic duct pathology, beside maldigestion *(2)*. The separate or simultaneous occurrence of these events beside the magnitude of damage in each can interpret the variation in clinical disease from one flock to another *(2)*. A new pathogenic event in the coarse of (IRSS) involves significant reduction of insulin after induced IRSS was added *(3)*.

In the present study, clinicochemical changes accompanying the previously reported

hormonal disturbance *(3)* were investigated to clarify other facts concerning IRSS.

MATERIAL AND METHODS

Descriptive data concerning the experimental design, clinical signs , postmortem findings, and schedule of blood collection are previously described *(3)*. Collected sera sample were examined without delay for the clinicochemical parameters. Total protein *(4)*, Albumin *(5,6)*, cholesterol *(7,8)*. Triglycerides *(9,10)*, ALP *(11)*, ALP after heat inactivation *(12, 13)*, calcium *(14, 15)* and phosphorus *(16)* were assayed. Globulin and Albumin globulin ratio were obtained via calculation.

The obtained data were statistically analyzed using MSTAT-C computer program *(17)*.

Table 1. Total protein, albumen, globulin, ALB/G ratio, ALP, ALP after heat inactivation, calcium, phosphrous cholesterol triglyuceride in broilers and native saso after induced IRSS 7,14,21, and 28 days PI .

Sub table	Examined parameters	Group	Broilers				Native saso			
			7 days	14 days	21 days	28 days	7 days	14 days	21 days	28 day
1.1	Mean T. protein (gm/dl)	1	1.638±0.25b	1.96±0.51b	2.024±0.008b	2.40±0.208b	2.08±0.15b	1.977±0.17b	2.02±0.19b	1.17±0.114c
		2	1.788±0.33b	1.657±0.26b	1.735±0.28b	1.911±0.306b	1.95±0.27bc	1.701±0.4b	2.43±0.1b	1.94±0.37bc
		3	1.859±0.206b	1.845±0.17b	2.006±0.47b	2.456±0.253b	1.44±0.11c	1.94±0.17b	1.543±0.16c	3.181±0.7ab
		Cont.	4.007±0.27a*	3.116±0.04a*	3.115±0.05a*	3.425±0.04a*	3.119±0.04a*	3.119±0.03a*	3.119±0.04a*	3.405±0.03a*
1.2	Mean albumin (gm/dl)	1	1.145±0.17	1.655±0.43	1.42±0.17	1.585±0.1	1.288±0.108a	1.578±0.2	1.655±0.21	0.906±0.05b
		2	1.331±0.16	1.438±0.19	1.193±0.11	1.608±0.14	1.144±0.17a	1.132±0.17	1.633±0.26	1.4098±0.3b
		3	1.339±0.09	1.272±0.55	1.88±0.07	1.59±0.06	0.505±0.101b	1.504±0.08	1.182±0.14	2.526±0.4a
		Cont.	1.25±0.07ns	1.166±0.04ns	1.065±0.02ns	1.154±0.02ns	1.283±0.123a*	1.148±0.05ns	1.065±0.02ns	1.159±0.02b*
1.3	Mean globulin (gm/dl)	1	0.44±0.131b	0.306±0.18b	1.06±0.24b	0.869±0.17b	0.796±0.23b	0.399±0.01b	0.378±0.11b	0.26±0.08b
		2	0.45±0.18b	0.218±0.17b	0.599±0.36b	0.306±0.2b	0.819±0.3b	0.691±0.33b	0.942±0.27b	0.55±0.24b
		3	0.594±0.106b	0.564±0.25b	0.967±0.25b	0.615±0.24b	0.968±0.1b	0.476±0.231b	0.486±0.14b	0.654±0.35b
		Cont.	2.75±0.28a*	1.949±0.02a*	2.3±0.23a*	2.26±0.02a*	1.626±0.15a*	1.968±0.02a*	2.3±0.23a*	2.24±0.02a*
1.4	Mean ALB/G ratio	1	1.85±0.28ab	1.533±0.28bc	1.415±0.56	2.185±0.64ab	1.34±0.16a	4.216±1.38a	4.44±1.63a	3.04±1.09
		2	2.77±0.36a	3.21±0.77a	1.218±0.285	1.82±0.68b	1.314±0.36a	2.004±0.84b	2.03±0.96ab	2.25±0.93
		3	3.27±0.92a	2.564±0.555ab	1.717±0.365	4.08±0.91a	0.557±0.14b	1.81±0.37b	2.96±0.82ab	2.2±0.547
		Cont.	0.493±0.06b*	0.584±0.18c*	0.528±0.02ns	0.51±0.06b*	0.719±0.11ab	0.584±0.35b	0.477±0.503c	0.515±0.116ns
1.5	Mean alkaline phsophatase (IU/ml)	1	763.7±25.1b	755.5±40.22b	776±28.49a	624.25±33.28a	858.7±30.7a	817±33.2a	805.5±32.15a	817.25±11.9a
		2	797±23.4ab	856.75±2.57a	741±50.3a	692.3±13.5a	859±32.33a	763±17.8a	885.75±31.6a	847.2±20.6a
		3	848.7±9.01a	725±22.28b	779±21.19a	706±42.58a	754.25±32.4a	785±43.9a	806.5±29.8a	815.7±26.01a
		Cont.	429.35±9.75c*	437.25±16.3c*	425±5.88c*	401±15.26c*	447.25±13.6c*	439.25±10.57b*	453.25±7.3b*	432.25±13.8b*
1.6	Mean alkaline phosphatase after heat treatment	1	170.95±4.9b	167.5±8.1b	205.64±3.3b	235.9±9.6b	230.25±4.86c	228.7±2.1b	289.52±3.7a	310.4±2.7b
		2	161±13.9b	159.75±8.4bc	215.3±4.24b	220.1±7.65a	235.7±9.7b	218.9±9.4b	278.1±2.7b	285.4±1.4c
		3	185.1±13.9c	175.25±8.07b	145.7±5.07c	250.8±9.9b	250.9±8.9h	238.1±4.7a	265.9±3.8c	269.1±5.4d
		Cont.	291.74±3.7a*	293.7±6.5a*	294.5±3.3a*	287.25±7.6a*	299.4±4.8a*	209.08±2.4a*	105.2±2.1d*	352.8±3.8a

Table 1. Continued

| Sub table	Examined parameters	Group	Broilers				Native saso			
			7 days	14 days	21 days	28 days	7 days	14 days	21 days	28 day
1.7	Mean calcium (mg/dl)	1	5.54±0.52b	4.26±0.82	6.77±0.43	8.44±0.72a	4.26±0.82	5.83±1.21	6.591±1.06	7.66±0.58
		2	4.77±0.56b	6.36±0.88	5.35±0.54	5.367±0.61b	5.8±0.57	3.961±0.2	4.71±1.24	7.37±1.46
		3	5.89±0.57b	4.7±0.46	6.56±0.46	8.027±0.84a	4.14±0.13	4.95±0.9	4.8±0.207	7.92±0.83
		Cont.	7.86±1.1a	4.57±1.3ns	6.86±0.054ns	8.207±0.84a*	5.07±1.15ns	5.366±0.54ns	6.09±1.12ns	8.69±0.6ns
1.8	Mean phosphorus (mg/dl)	1	5.55±0.2	5.87±0.22ab	5.16±0.86	5.65±0.15	5.33±0.24	5.5±0.23	5.42±0.15	6.51±0.14
		2	5.86±0.11	6.38±0.16a	5.62±0.32	5.93±0.11	5.24±0.36	5.83±0.29	5.24±0.3	6.57±0.07
		3	5.41±0.4	6.26±0.13a	5.86±0.18	6.09±0.22	5.45±0.29	6.03±0.18	5.24±0.29	6.33±0.24
		Cont.	5.33±0.24ns	5.24±0.4ns	5.5±0.169ns	5.162±0.4ns	6.04±0.21ns	5.95±0.11ns	5.78±0.24ns	6.7±0.16ns
1.9	Mean cholesterol (mg/dl)	1	145.41±14.48a	275.5±38.3a	349.07±4.9a	357.58±26.9a	159.5±10.2bc	278.54±8.9a	270.8±10.7a	273.07±4.3a
		2	143.04±9.7a	240.4±13.7a	247.87±6.6c	267.66±35.6b	142.6±4.8c	233.15±23.0a	265.3±11.8a	2142.2±15.6a
		3	166.8±13.2a	244.25±15.8a	280.58±4.02b	248.68±14.6b	170.32±4.1a	265.3±11.8a	271.33±15.4a	244.9±10.4a
		Cont.	92.64±4.37b*	115.17±15.18b*	183.22±4.8d*	181.9±15.4c*	104.12±6.8d*	111.23±2.14b*	111.5±10.66b*	45.8±4.4b*
1.10	Mean triglycerine (mg/dl)	1	268.32±2.26b	333.8±9.8b	342.18±10.17b	241.16±30.50c	338±9.82b	537.24±8.27a	466.2±1.34b	650.6±18.2a
		2	394.76±2.78a	573.12±37.8a	480.48±4.3b	375.4±32.8b	461.6±19.97a	540.92±10.8a	746.4±16.7a	662.58±10.4a
		3	439.66±18.78a	416.6±12.16ab	541.17±21.2a	855.0±4.96a	288.32±13.25c	522.6±23.2b	708.5±32.8a	499.9±5.49
		Cont.	185.44±3.8c*	244.8±18.4c*	255.76±4.4c*	172.8337±22.9d*	288.32±19.1c*	234.95±10.72c*	380.325±4.0b*	272.84±39.2*

$*$ = (P<0.05) ns = non significant changes
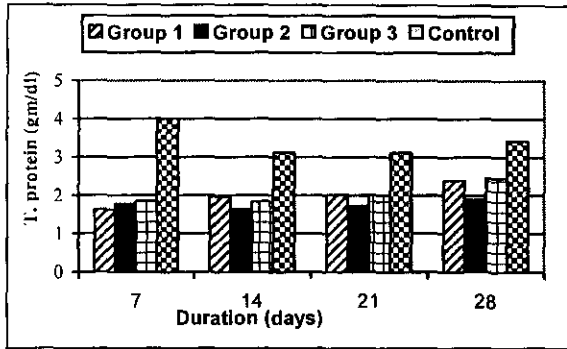
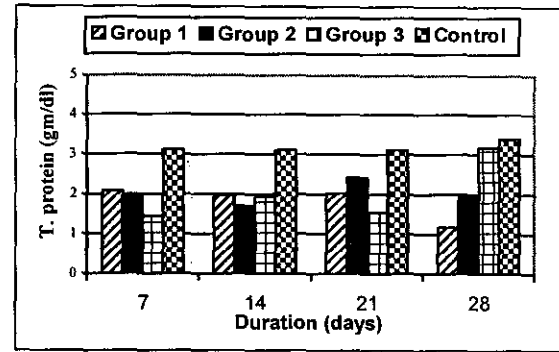Fig. 1. Total protein of broilers in gm/dl (PI).

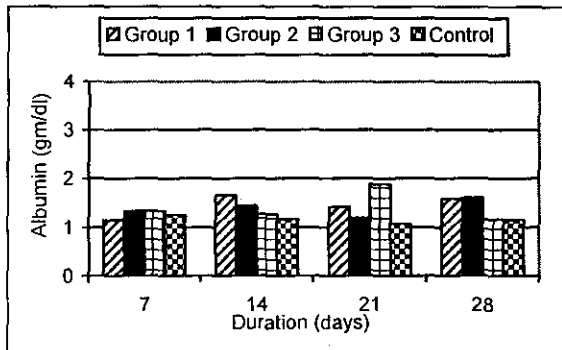Fig.2. Total protein of native Saso in gm/dl (PI).
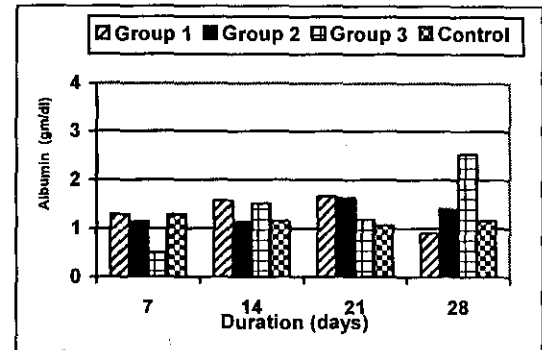
Fig. 3 . Albumin of broilers in gm/dl (PI).
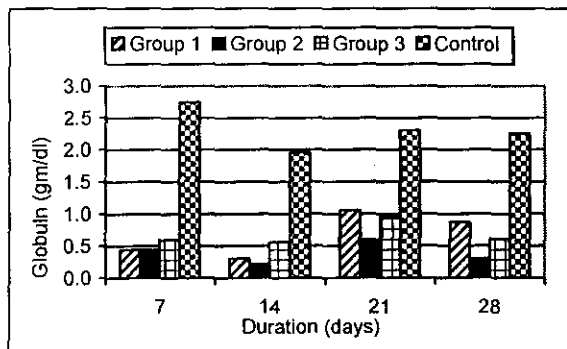
Fig. 4 . Albumin of native Saso in gm/dl (PI).
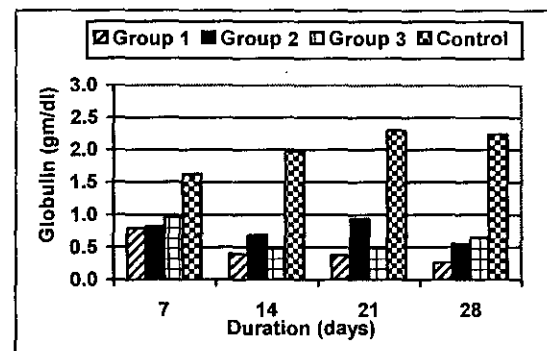
Fig. 5 . Globulin of broilers in gm/dl (PI).

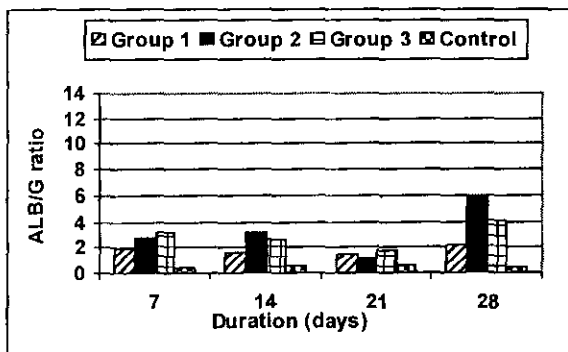Fig. 6 . Globulin of Native Saso in gm/dl(PI).
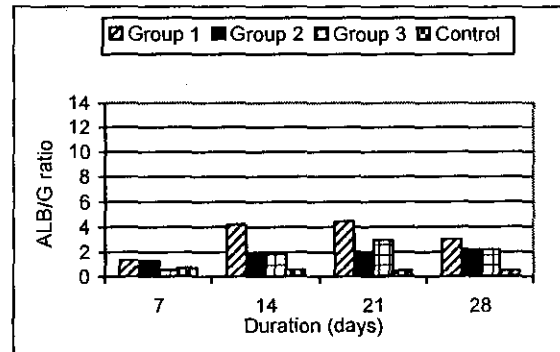
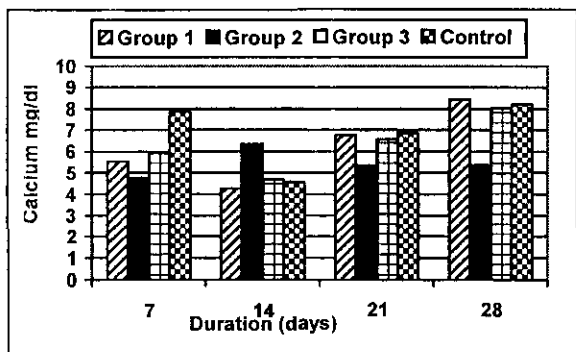Fig. 7 . ALB/G ratio of broilers.

Fig. 8 . ALB/G ratio of Native Saso.

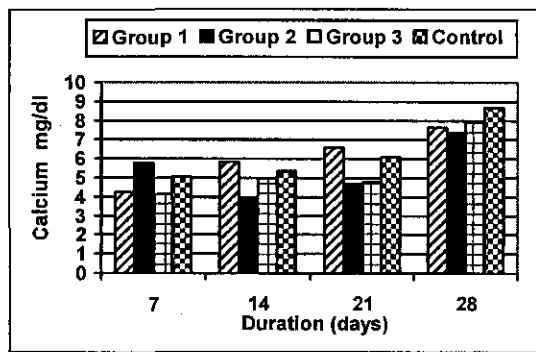Fig. 9 . Mean calcium of broilers in mg/dl (PI).

Fig. 10. Mean calcium of native Saso in mg/dl (PI).
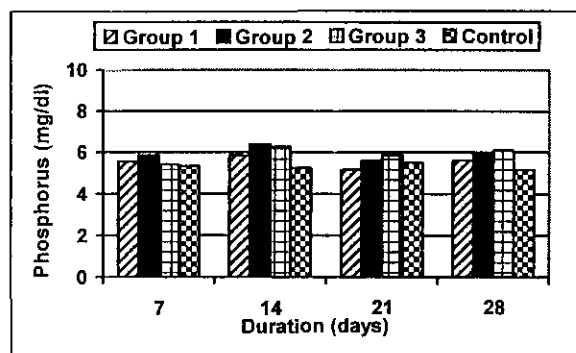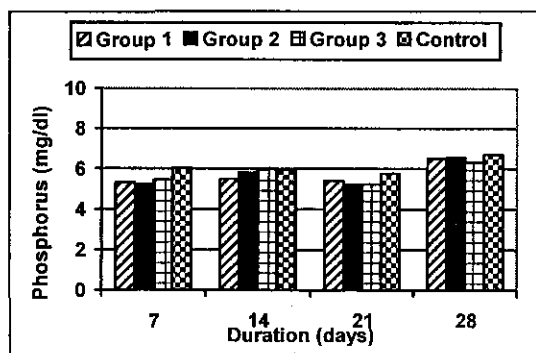

Fig. 11 . Phosphorus of broilers in mg/dl (PI).

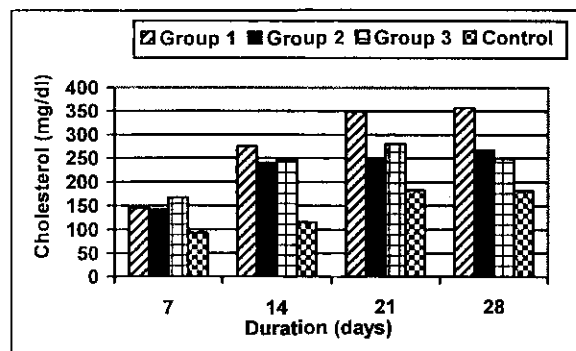Fig. 12 . Phosphorus of native Saso in mg/dl (PI).

Fig. 13 . Cholesterol of broilers in mg/dl (PI).
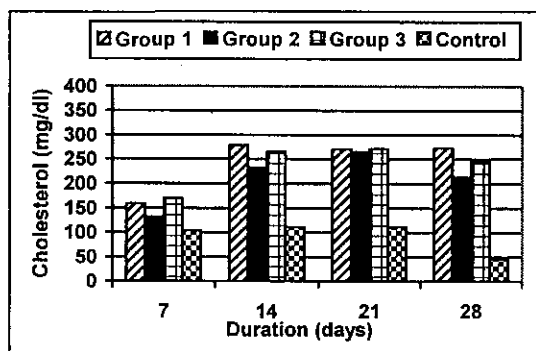

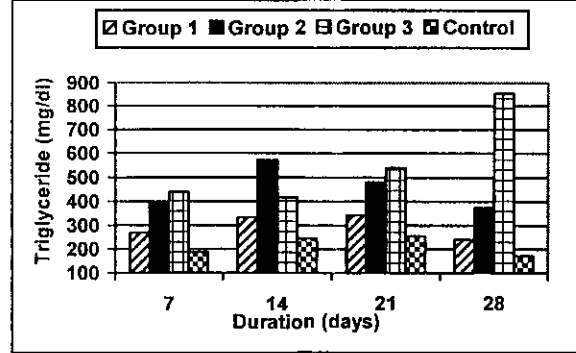Fig. 14 . Cholesterol of Native Saso in mg/dl (PI).

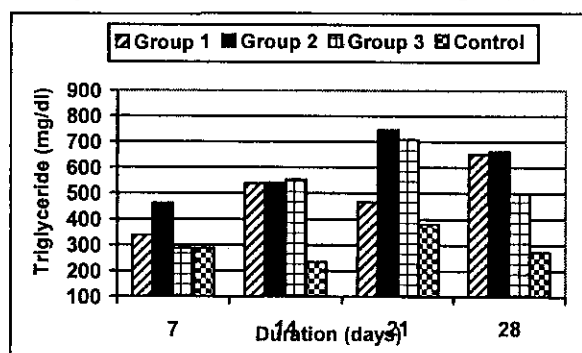Fig. 15 . Triglyceride of broilers in mg/dl (PI).

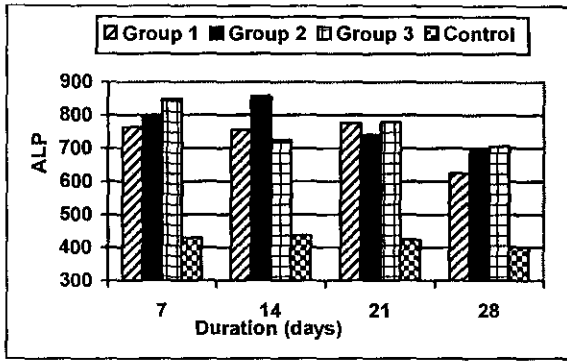Fig. 16 . Triglyceride of Native Saso in mg/dl (PI).

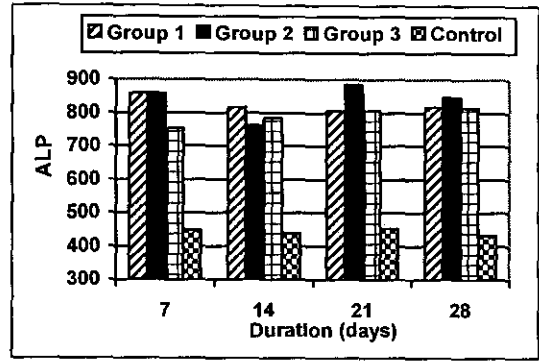Fig. 17 . Alkaline phosphatase of broilers (PI)..

Fig. 18 . Alkaline phosphatase of native Saso (PI)..
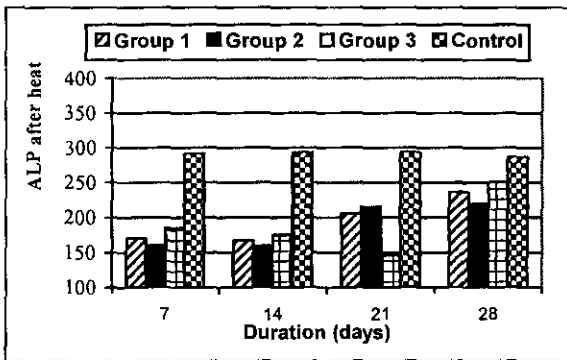

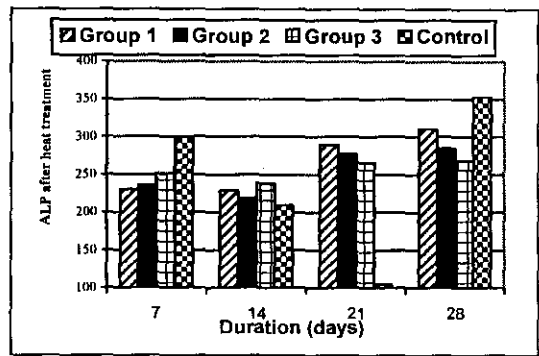Fig. 19. Alkaline phosphatase after heat treatment in broilers (PI).

Fig. 20. Alkaline phosphatase after heat treatment in native Saso (PI)..

Table 2. Mean ALP (PI) before and after heat inactivation in inoculated and control chicks

Sub table	Item	Broilers				Native saso			
		7 days	14 days	21 days	28 days	7 days	14 days	21 days	28 day
2.1	X	803.13	779.06	766.33	674.25	823.98	788.33	832.58	826.7
2.2	Y	172.35	167.5	205.5	235.6	238.95	228.56	277.84	288.3
2.3	x-y	630.78	611.56	560.83	438.56	585.03	559.77	554.74	538.4
2.4	x-y%	78.54%	78.49%	73.18%	65.04%	71%	71%	66.68%	65.04%
2.5	Xc	429.35	437.25	425	401	447.25	439.25	453.25	432.25
2.6	Yc	291.74	293.7	294.5	287.25	299.4	309.08	305.2	352.8
2.7	xc-yc	137.61	143.55	130.5	113.75	147.85	130.17	148.05	79.45
2.8	Net osseous ALP (N.O. ALP)	493.17	469.01	430.33	324.81	437.16	429.6	406.69	458.95
2.9	% of (N.O ALP)	61.4%	60.2%	56.15%	48.1%	53.05%	54.49%	48.84%	55.5%

X = Mean ALP of inoculated groups (G1, G2, G3)   Y= Mean ALP of inoculated groups post heat inactivation
X-y= ALP from osseous source in inoculated groups.   Xc= Mean ALP of control group
Yc= Mean ALP of control post heat inactivation   Xc-Yc= ALP from osseous source in control
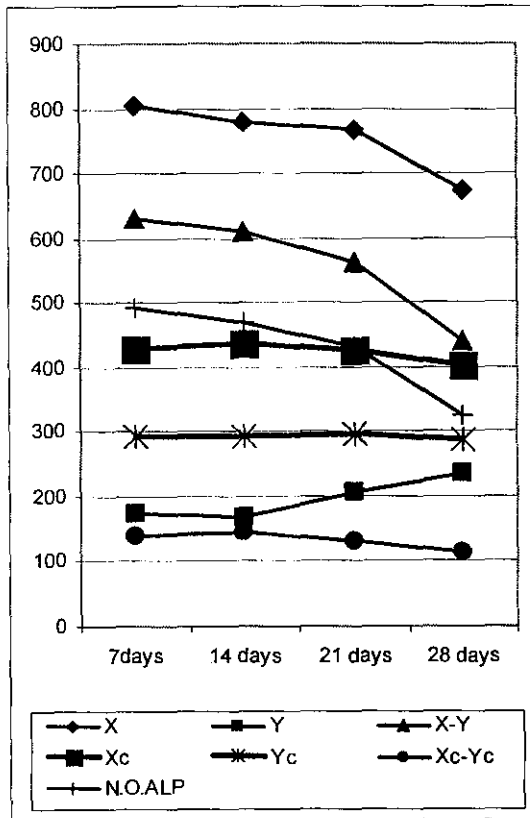(N.O.ALP)= Net osseous ALP in inoculated birds = (X-Y) – (Xc – Yc)

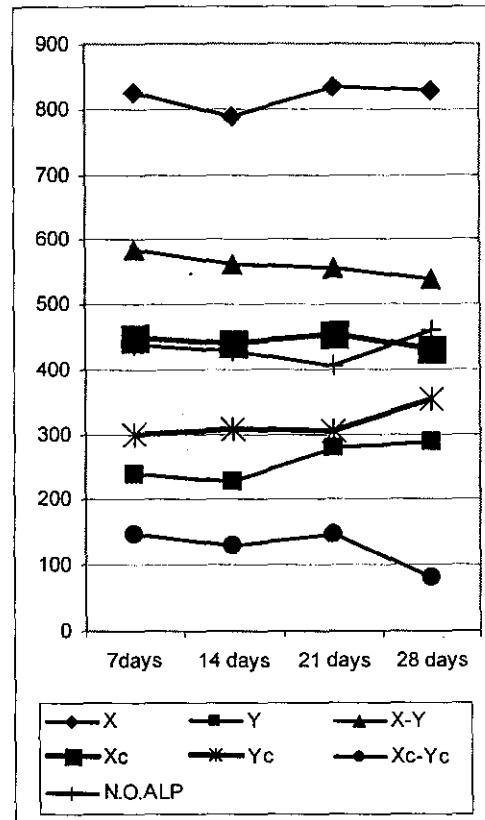Fig. 21. Mean ALP (PI) before and after heat inactivation in broilers

Fig. 22. Mean ALP (PI) before and after heat inactivation in native saso

RESULTS AND DISCUSSION

Calcium level was significantly low in inoculated broilers 7 days PI, and was only significantly low at 28 day PI in broiler group inoculated with (int. homog.b) , as for the inoculated native saso chicken breed no significance could be demonstrated during the entire experimental period. Similar results were reported after induced infection *(12)*, this was also true in the natural field cases of IRSS affecting native breeds *(18)* (Table 1.7, Figs. 9,10).

Total calcium should be interpreted along with albumin concentration *(1)*, in the present study albumen levels showed non significant changes in the inoculated broilers. The native saso chicken the group inoculated with intestinal homogenate c *(3)* showed significant hypoalbumenaemia at 7 days (PI) and showed hyperalbumenaemia at 28 days

(PI) (Table 1.2 , Fig. 3,4). This may reflect the state of hydration or dehydration *(1)*, but calcium was not significantly affected at the two time points. Calcium plays several important functions such as its role as a major constituent of bones, transmission of nerve impulses, the permeability and excitability of cell membranes, muscular contraction and activation of enzyme systems *(1)*.

If we consider the non significance of the calcium level after induced IRSS in the present study (Tables 1.7, Figs. 9, 10) or as previously reported *(12, 13)* and the non significant changes in calcium level in naturally affected birds with IRSS *(18)*. An important question will imposes its self, what is the real cause for bone alterations that lead for the synomous nomenclature of IRSS as Brittle bone disease and femur head necrosis *(19)?*. Another question., why previous investigations didn't demonstrated an increase

in the total calcium concentration as it should be with osteolytic bone changes *(1)?*.

ALP is involved in energy transfer for Ion exchange across the cell membrane *(1)* ALP activity may be elevated due to irritation of cells in different tissues. Pathological elevation of ALP is common in liver diseases, induced stimulation of osteoplastic activity and enteritis. Increased ALP is one of the criteria required for fulfillment of successful induction of IRSS *(20)*. In the present study ALP activity was significantly higher in inoculated broilers at 7,14, 21 and 28 day PI (Tables, 1.5, 2, Figs. 17, 18). This elevation was also recorded *(12, 13, 20)*, inoculated native saso at 14 and 21 day PI showed elevation similar to the previous finding *(18)*.

ALP inhibition after L- phenylalanine treatment signifies that, the inhibited ALP is from intestinal source *(13, 21)*, while ALP inhibition after heat inactivation signifies osseous source *(12, 13, 22 -25)*. In the present study ALP activity was reduced in both control and inoculated birds after heat inactivation (Table 2.2, 2.5). The heat inactivated ALP was constituting larger portion of the total ALP (PI) (Table 2.3 and 2.4). Since negative control had ALP activity from osseous source so we had to subtract these values from those of inoculated groups (Table 2.5, 2.7, 2.8) to obtain a net ALP from osseous source due to the inocula used (Table 3.8), the percent of this net ALP was calculated (Table 2.9), this net ALP portion from osseous source was constituting a great percent of the total ALP activity (Table 2.9, Fig. 21,22) these findings contradicts the previously reported results *(12, 13)* which refers that the encountered ALP was from intestinal source . It is worth to mention that increased ALP in control birds in the first week is due to physiologic bone activity *(1)*.

Reoviruses causing IRSS, differs in the *in vivo* and *in vitro* characteristics from reoviruses causing viral arthritis *(26)*. The same mentioned author found that reoviruses causing IRSS dosen't move beyond the inoculation site. So, orally inoculated viruses remains in the gastrointestinal tract, on the contrary orally inoculated arthrotropic viruses causes viral arthritis. Thus osseous damage due to viral replication is not expected in reoviruses causing IRSS. The reported insulin deficiency after induced IRSS *(3)* may be the reason for the bone changes and the easily splitting of femur head articular surface via its effect on carbohydrate metabolism with special reference for the, hyaluronic acid which acts as bone cement *(27)*.

On the other hand phosphorus also showed non significant changes after induced IRSS during the experimental period in both chicken breeds (Table 1.8, Fig. 11, 12), this is in agreement with several previous studies *(12, 13 and 18)*. For this reason calcium and phosphorus role in bone brittleness found in the coarse of IRSS should be disregarded.

Determination of total plasma proteins can be used as an indicator for heath status, it is also valuable in the diagnosis of gastrointestinal, renal or hepatic diseases, but it seldom leads to a specific disease diagnosis; it can be used as indicative for the severity and progression of diseases *(1)*. In the present study total protein was significantly reduced in inoculated birds in both chicken breeds, at 7,14, 21 and 28 day PI compared to control (Table 1.1, Fig. 1,2) these results disagree with the previously reported studies *(18, 28)*.

Hormones that influence carbohydrate metabolism can indirectly influence lipid and protein metabolism. Insulin by its effect on glucose metabolism helps to provide the energy necessary for protein synthesis , and it may have a direct effect on amino acid transport *(29)*.

Globulin level showed significant reduction in inoculated birds compared to control. This reduction was detected at 7,14, 21 and 28 day PI in both breeds (Table 1.5 , Fig. 5,6). It seems that globulin reduction is the point responsible for total protein reduction, this globulin reduction may be acceptable in view of the known immunosuppressive potential of reovirus *(30 - 34)*. Specially if the long viral shedding period of reoviures is consider *(3, 33-37)*.

Immunoglobulin are synthesized by the cells of the reticuloendothelial cells in response to a variety of antigenic stimuli *(29)*. Lowered immunoglobulin can result from liver diseases, malabsorption and malnutrition *(38)* all these are reported in IRSS *(3)*.

Lipid circulating in blood are derived from intestinal absorption, synthesis in the liver or mobilization from fat depots., They are classified into triglycerides, phospholipids, cholesterol esters, free fatty acids and fat soluble vitamins *(29)*. In the present study cholesterol and triglycerides were significantly higher than control in both chicken breeds at 7,14, 21 and 28 days PI (Table 1.9, 1.10 , Figs. 13-16). Glucose is a major precursors of lipid synthesis in the body specially in birds and in animals obtaining high carbohydrates through its action on glucose transport. Insulin plays a role in the regulation of lipogenesis; with increased insulin output glucose uptake increases. Insulin also influence triglycerides synthesis by regulating esterification through the provision of α- glucose phosphate substrate. The required fatty acid can be supplied by plasma fatty acids, through synthesis from glucose or through hydrolysis of the existing triglycerides. Triglycerides hydrolysis by extra hepatic tissue is mediated by lipoproteinlipase which in an enzyme mediated by Insulin and directly or indirectly by glucose, thus insulin acts as an important antilipolytic hormone through inhibiting the release of glycerol and fatty acid in adipose tissues through the conversion of glucose to fat *(39)* these physiological facts can justify elevation of cholesterol and triglycerides after induced IRSS as a sequallae for insulin deficiency *(3)*.

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الملخص العربى

التغيرات البيوكيميائية الحادثة فى سيرم كتاكيت التسمين والبلدى الساسو
بعد العدوى الاصطناعية بمتلازمة التقزم وإعاقة النمو

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معهد بحوث صحة الحيوان بالزقازيق وحدة الدواجن والباثولوجيا – المعمل المركزى للنظائر المشعة بمستشفيات جامعة الزقازيق**

أجريت هذه الدراسة لمعرفة التغيرات البيوكيميائية الحادثة فى سيرم كتاكيت التسمين والبلدى الساسو بعد العدوى الاصطناعية بمسببات متلازمة التقزم وإعاقة النمو. واستكمالاً لدراسة سابقة تم دراسة الخلل الهرمونى بها وكذلك للحصول على نظرة أكثر شمولاً عن كيفية الإحداث المرضى فى ضوء انخفاض مستوى هرمون الأنسولين فى الدجاج المصاب بمتلازمة التقزم وإعاقة النمو كما هو ثابت من دراستنا السابقة.

تم فى هذه الدراسة استعمال عينات سيرم من كتاكيت تسمين وبلدى ساسو محقونة بالمزيج المعوى من ثلاث قطعان مصابة حقلياً بمتلازمة التقزم وإعاقة النمو عند الفقس. هذه العينات جمعت فى عمر ٧, ١٤, ٢١ و٢٨ يوماً.

أظهرت النتائج أن العدوى الصناعية بمسببات متلازمة التقزم وإعاقة النمو قد أحدثت زيادة معنوية فى إنزيم الفوسفاتيز القلوى فى الكتاكيت التسمين عند ٧, ١٤, ٢١ و ٢٨ يوماً. والبلدى الساسو عند عمر ١٤ و ٢٨ يوم وقد وجد أن المعاملة الحرارية لعينات السيرم عند ٥٦م لمدة ٣٠ دقيقة قد أحدثت انخفاضاً معنوياً فى مستوى إنزيم الفوسفاتيز القلوى مما يشير إلى أن هذا الأنزيم ناتج من التغيرات الباثولوجية بالعظام مخالفاً بذلك نتائج الغير التى كانت تعتبران مصدر هذا الإنزيم هو التغيرات الباثولوجية بالأمعاء.

كذلك أظهرت النتائج حدوث انخفاض معنوى فى البروتين الكلى فى نوعى الكتاكيت عند عمر ٧, ١٤, ٢١, ٢٨ يوم. هذا الانخفاض كان ناتجاً عن الانخفاض المعنوى فى مستوى الجلوبيولين فى هذه الأعمار ويمكن تفسير ذلك فى ضوء التثبيط المناعى الناتج من عدوى فيروس الريو وخصوصاً اذا ما وضعنا فى الاعتبار طول فترة تكاثره بداخل الكتاكيت. ولم يتم ملاحظة فروق معنوية فى مستوى الزلال فى كتاكيت التسمين والبلدى فى عمر ١٤, ٢١ يوم إلا أنه كان مرتفعاً فى المجموعة المحقونة بالمزيج المعوى Int. homog.c فى عمر ٧, ٢٨ يوم فقط.

أمكن تسجيل ارتفاعاً معنوياً فى مستوى الكلويسترول والدهون الثلاثية فى عمر ٧, ١٤, ٢١, ٢٨ يوم فى الكتاكيت البلدى والتسمين. بعد العدوى الصناعية بمسببات متلازمة التقزم وإعاقة النمو.

لوحظ وجود انخفاض معنوى فى مستوى الكالسيوم فى الكتاكيت التسمين فى عمر ٧ أيام فقط. إلا أنه لم تكن هناك فروق معنوية عند تقديره فى كلا الهجينين فى باقى المراحل العمرية. وكذلك لم نتمكن من تحديد فروق معنوية فى مستوى الفسفور فى الطيور المحقونة بمسببات متلازمة التقزم وإعاقة النمو وعلية يمكن الجزم بعدم وجود أى دور للكالسيوم والفوسفور فى التغيرات الحادثة فى العظام .

التغيرات الكيميائية الحادثة فى هذه الدراسة منطقية جداً فى ضوء انخفاض مستوى هرمون الأنسولين فى كتاكيت التسمين والبلدى. ومناسبة لطبيعة مرض السكر ومن خلال ذلك يمكن تفسير العديد من المشاهدات الحقلية والآفات التشريحية الخاصة بمتلازمة التقزم وإعاقة النمو ، مثال ذلك زيادة استهلاك العليقة وانخفاض معدلات الأوزان – زيادة استهلاك المياه – انخفاض حيوية الطيور – امتلاء الإمعاء بالغذاء الغير مهضوم والغازات وانتفاخ البطن – التغيرات الحادثة فى العظام – وكذلك بعض التغيرات فى نسيج المخ التى تم رصدها فى دراسة سابقة.