Senescence and Immunity in some Arthropathy patients

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Shnawa I.M.S And AL-Amidi B.H.H College of Scines / Babylon University *College of Dentistry/ Badylon University Abstracts

Three basic arthritis types were noted among elderly and adults. The first inflammatory lymphocytic, the second autoimmune, while, the third is septic nutrophilie type. The inflammatory and autoimmune were associated with mucosal lymophcytosis. The septic was neutrophilic both at mucosal and systemic arms. The autoimmune was rhumatoid factor positive and the sptices were associated with injured S.aureus and St.pyogenes. senesceance exhibits some what suppressive effects on specific antibody titres as 200/40 (systemic/ mucosal) while in adulthood was 320/32 and leucoyte inhibitory factors as 0.8/0.28 (systemic/ mucosal) and in adulthood as 0.6 / 0.45 thus elderly arthropathic patients showd suppressed antibody and LIF responses as compared to adulthood

الشيخوخة ومناعة بعض مرضح المفاصل

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الخلاصة

لوحظت ثلاث انماط من التهاب المفاصل بين الشيوخ والبالغين. النمط الاول التهابي لمفي ، النمط الثاني هو ناتج من مرض ذاتي المناعة ، النمط الثالث كان تقيحي ذو ارتشاح العدلات . وقد اشترك كل من اللمفي وذاتي المناعة مع زيادة خلايا لمفية على السطح المخاطي المفصلي في حين كان بزيادة عدلات في التقيحي وعلى مستوى الاستجابة البدينة والمخاطية. وكان النمط ذاتي المناعة موجب مع عامل الرثوانية وتبين بان النمط التقيحي مشترك مع مكورات عنقودية ذهبية ومكورات سبحية مقيحة متضررتين. وقد ابدت الشيخوخة بعض الفعل المثبط على الاستجابة المناعية البدينة والمخاطية(0.40/ 200) (مستوى الاضداد) والخلوية (تثبيط هجرة الخلايا(0.8/0.28)

Introduction

Human life span consist of childhood, aldolesence, adulthood and senescence (1).

The humoral and cellular elements for natural and adapted immune responses undergos several changes on aging in comparison to that of youth and adolescant. Some elements are decreasing, others, however, are increasing. Such spectra of increasing and decreasing can be a result of genetic and environmental factors (1, 2). The objective of the present work was at the study the influences of immunoscence on human arthropthy

Materials and Methods

Nine adolescent and Nine aged arthropathy patients and nine other control subjects. Synovial and blood samples were collected Mucosal immunglobulin was separated as in (3). Sera were obtained for total serum protein, serum globulin and specific antibody as well as RF test were made (4, 5). LIF was done for peripheral blood and mucosal leucocytes (6). Bacterial culture study was done as in (7, 8).

Migration of leucocytes with sensitizar

LIF % = X 100

Migration of lecocytes without sensitizer Inhibtion of more than "30% is significant.(6)

Results

- I- Synovial fluid; three major types of synoivial fluid were noted as Inflammatory autoimmune and septic (Table -1)
- II- Aetiology; Bacteria, autoantibody, anti IgG (RF) and all defined (Table-I).
- III- Inflammatory cells; Neutrophilice as in S. pyogenes& St aureus acute arthritis. Lymphocytic responces were evident in those of nonbacterial causes. Mucosal lymphocytosis was 68.4 and higher than these of bacterial arthritis 18.5. Table 1 &2
- IV- Mucosal Globulin (MG) & Serum Globulin SG concentrations of aged arthopathy patients was higher than in normal subjects. In patients serum globulin was higher than controls. aged patients were higher than adolescent and youthood.

- V- Specific antibody; (table 2, 4) pyogenic acute arthritis rise specific antibodies titer to 320 in blood and 32 at mucosa in adolscent ageing suppresses such titres
- VI- Autoantibody; Two sensce and two youthood RF positive cases.
- VII- Leucocyte Inhibitory factors (LIF) (Table 3 &4), LIF at mucosal surface and peripheral blood were signficant in adolescent and nonsignificat in aged patients. (Table 3, 4).

Discussion

The human life spane in heath and disease consist of childhood adolescent adulthood and sensence phases (1,9). Thus in any instance of his life one may expect to fined out healthy and diseased adulthood as well as health and diseased senscences subject (10). The major histocompatibility complex (MHC) and its polymorphic genes experssion products, the human leucocyte antigons (HLA) or major histompatability molecules controls, the immune responses, antigen processing antigen presentation and immune recognition (11).

The childhood and senscence are mostely associated with reduction of immune reactivity and can be attributed to immune conecting lymph vessels, low number of lymphocyte in lymph nodes or lack of lymph ocyte and macrophages surface receptors (12). While in case of senescent changes may be attributed to ultered elements of natural immunity or ultered elementes of adapted immunity (13). Since aging is a multifarcous process that affects different individuals in different ways and affects discrete organ systems in distinct ways within same individual. Furthore more, within complex organ systems, different segments or portions of the system may be affected in different rate and to different degrees by aging (1).

Aging may affect reduction in antigen uptake antigen processing, antigen precsentation, and immune recognition (14). Lower antibody level seen in senscence patients can be attributed impairment in antigen recognition by Th2 or B cell or low antibodyes synthesis by plasma cell, replinishing effect or increased antibody catabolism (15).

Non segnificant LIF in aged patient(table-1) indicate either of the followings(5,6,11,13)

- i. Abscances of approperiate sensitizar (inducer)
- ii. Low synthesis
- iii. Diminished cytokine production
- iv. The produced cytokine involved and exhusted to other reactions(16).

The noted RF positive case means involvement of an autoimmune responses that can be induced by an autogenic epitope or by bacterial derived epitope having portion of mimicking property to an autoantgen (17). Aging however, may facilitate breaking self tolerance barrier leading to an autoimmune arithritis

Higher mucosal and systemic globulin concentration can be due to concamittent environmental exposure to antigen by B lymphocytes or Th2 cells which may ends by polyclonal over production of globulin by plasma cells or due to an inflamatery or infectious process(table-2) (18).

References

- 1- Cohen, H., J.1999 a. Biology and physiology of Aging. American society of Haematology U.S.A., 500-504.
- 2- Cohen, H.J. Biology of aging as related to cancer. Cancer. 1999b. 74 (suppl.): 720-742.
- 3- Shnawa ,I.M.S and Alamidi,B.H.H. Med.J. Babylon. 2004, 1(3&4):
- 4- Garvey, J.S; Cremer, N.E. and Sussdrof D.H 1977. Methods in Immunology 3rd ed Addision Wesley Puplishing company. Massach setts, 517-534.
- 5- Rose , NR. And Bignzzi, P.E 1980. Methods In Immunodiagnosis. 2nd ed A Wiley Medical Puplications. John Wiley and sons new york.
- 6- Soberg, M. Acta. Med. Scand. 1968. 184: 235.
- 7- Baron, E.J.; Peterson, L.R. and Finglod, S.M. 1994. Baely and Scotts Diagnostic Microbiology 9th ed Mosby- Year Book Ine London, 249-257.
- 8- Macfaddin J.F. 2000 . Biochemical Tests for Identification of Medical Bucteni 3rd. Lippinocott Williams & Wilkins, London
- 9- Gerns, D. Hasti Cen. Rep. 2003. 33(4): 31-39.
- 10- Russo, C.; Cherniak, E.p. and Wali, A Proc. Nath Acad. Sci. 1993 90: 11718-11722.
- 11- Caruse, C.; Candore, G. and Romano G.C. Mech. Aging Dev. 2001; 122: 445-462.
- 12- Goodwin, J.S. Nut Rev. 1995. 53(supp): 4-46.
- 13- Hara, H.; Negoro, S. and Miyata, S. Mech. Aging. Dev. 1987.: 38: 245-258.
- 14- High, K.P. clin. Infec. Dis. 1999 28-:717-722.
- 15- Hodes, R.J. Immunal Rev. 1997. 160: 5-15.
- 16- Song, L.; Kim, Y.H.; and Chopra, R.K. Exp. Gerontol. 1993, 28: 313-321.

- 17- Knight J.N. Ann. Clin. Lab. Sci. 1995 25: 1-10.
- 18- Walter, D.K.; Banerjec, M. and Mechr R. Biochm. Soc. Trans. 2003, 31: 447-448.

Table -1 : Systemic versus mucosal immune response of patient with St. pyogenes Active Arithritis

		Statistical Features							
Immune Parameters	Mean	Median	Range	Control mean					
1.Inflammatry cells									
1.1-Neutrophil									
mucosal	66	80	62-88	25					
Systemic	73.6	73	71-77	64					
1.2 Lymphocyte									
Mucosal	20.66	17	12-38	15					
Systemic	21.66	20	21-24	37					
2. Leucoyte Inhibitory Factor									
Mucosal	0.505	0.6	0.44-0.82	0.96					
Systemic	0.672	0.6	0.6-0.82	0.97					
3. Globulin									
Mucosal	1.012	0.99	0.46-1.6	0.5					
Systemic	44.978	45.75	0.6-082	36					
4- St.pyogense specific antibody									
titres									
Mucosal	36	32	8-64	0					
Systemic	293.3	320	80-640	0					
5- Rhumatoid Factor mucosal	-	-	-	-					
Rhamatoid Factor systemic	-	-	-	-					

Table 2: Arthritis types in Elderly and adulthood

		Se	nescence		Adulthood					
Parameters		Septic	Autoimm	Mean	Septic	Autoimm	Mean			
		Arthritis	Arthritis		Arthritis	Arthiritis				
Mean Age		64.75	63	63.89	19.86	56.33	38.1			
cell	N	73.25%	58.66%	65.9%	73.75%	55.33%	64.54%			
System cell count	L	32%	34.33%	28.2%	22.25%	40.33%	31.29%			
N N	M	4 %	5 %	4.5%	3 %	3 %	3%			
	E	1.5 %	0 %	0.75%	1 %	1 %	1%			
	В	0 %	0 %	0 %	0 %	0.3 %	0.15%			
Mucosal	N	81.25%	26.33%	33.79%	81.25%	27 %	54.13%			
Muc		18.75%	73.66%	46.2 %	18.75%	73 %	45.87%			
Mucosal Globulin		0.78	1.58	1.18	0.9	1.2	1.05			
Serum Globulin		42.22	47	44.6	42.22	42.88	42.55			
Total serum protein		72.97	79.6	76.28	77.17	80.27	79.24			
Agent		S.aureus / St.pyogens	-		S.aureus/ St.pyogen s	-				
RF		-	+	-/+	-	+	-/+			
LIF		0.8/0.78	-		0.6/0.45	-				
Titre		200/40	-		320/32	-				

table 3 Immune parameters in sensanals and Adulthood patient with autoimmune arthritis

Se	A	Diffe	rentia		ount/	Diffe	renti							
q	ge	syste	mic				al		M	S	TS	Ag	R	Ti
		-					Muco	osal	G	G	P	ent	F	tre
Sen	esce	N	L	M	Е	В	N	L						
nt														
1	63	54	37	7	0	0%	37	63	1.	60	82	-	+	-
		%	%	%	%		%	%	5		.0			
									6					
2	60	51	42	5	0	0%	24	76	1.	46	78	-	+	-
		%	%	%	%		%	%	6		.4			
3	65	71	24	3	0	0%	18	82	1.	35	78	-	+	-
		%	%	%	%		%	%	6		.4			
X	63	58.	34.	5	0	0%	26.	73.	1.	47	79	-	+	-
		66	33	%	%		33	66	5		.6			
		%	%				%	%	8					
Adulth											81	-	+	
ood	ı	60	36	2	1	1%	25	75	0.	48	.3			
1	56	%	%	%	%		%	%	9	.9	1			
									9	1				
2	56	52	42	5	1	0%	35	65	1.	32	78	-	+	-
		%	%	%	%		%	%	8	.3	.2			
									2					
3	57	54	43	2	1	0%	21	79	0.	47	81	-	+	-
		%	%	%	%		%	%	7	.4	.3			
									8	4	1			
X	56	55.	40.	3	1	0.3	27	73	1.	42	80	-	+	-
	.3	33	33	%	%	3%	%	%	2	.8	.2			
	3	%	%		-	0.1				8	7			
To	54	56.	37.	4	0.	0.1	26.	73.	1.	44	79	-	+	-
tal	.6	49	33	%	5	1%	66	33	3	.9	.9			
M	6	%	%		%		%	%	9	4				
ea														
n														

Table 4 :- Immune parameters in sensonce and Adulthood with septic bacterial Arthritis

Seq	Age	age Differential count/ systemics Differential				ntial							
_							Mucosa	1	MG	SG	TSP	Agent	
Senceneat		N	L	M	Е	В	N	L					
1	63	72 %	23 %	4%	1%	0%	83%	17%	1.0	36.2	78.4	S. aureus	
2	65	74 %	21 %	3%	1%	0%	75%	25%	0.67	38.6	70.89	S. aureus	
3	65	73 %	20 %	5%	2%	0%	84%	1.6%	1.0	54.0	81.89	St. pyogens	
4	66	74 %	21 %	4%	1%	0%	83%	17%	0.46	40.08	60.7	St. pyogens	
X	64.75	73.25%	22 %	4%	1.5%	0%	81.25%	18.75%	0.78	42.22	72.97		
Adulthood		73 %	24 %	2%	1 %	0%	80%	20%	0.99	43.9	78.3	S. aureus	
1	39												
2	40	75 %	22 %	2%	1 %	0%	78%	22%	0.57	38.61	71.00	S. aureus	
3	47	75 %	20 %	4%	1 %	0%	88 %	21%	0.72	46.3	80	St. pyogens	
4	38	72 %	23 %	4%	1 %	0%	80%	12%	1.3	42	78.8	St. pyogens	
X	19.86	73.75%	22.25%	3%	1 %	0%	81.15%	18.75%	0.9	42.1	77.1		