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JUNIN VIRUS INFECTION OF *CALITHRIX JACCHUS*: HAEMATOLOGICAL FINDINGS[1]

M. J. FRIGERIO[2], S. N. RONDINONE[2], M. A. CALLELO,
E. R. PARADISI[2], M. C. WEISSENBACHER[2]

Department of Microbiology, Faculty of Medicine, University of Buenos Aires, Paraguay 2155 — Piso 12 — 1121, Buenos Aires, Argetina

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Summary. — Haematological changes produced by experimental Junin virus infection of a platyrrhine monkey, *Callithrix jacchus* were studied. Normocytic and normochromic anaemia appeared after 7 days post infection (p. i.), and increased steadily until day 21 p. i. Reticulocytes and circulating erythroblasts were elevated during the anaemia, reached a peak on day 7 p. i., and disappeared later. Platelets and leukocytes showed a significant decrease from days 14 and 18 p. i., respectively. These alterations could be attributed to the damage of bone marrow and lymphatic tissue.

Key words: *Junin virus, Callithrix jacchus, haematology*

Introduction

In previous reports we have described a fatal disease, which developed in the platyrrhine monkey, *Callithrix jacchus* (Frigerio *et al.*, 1978, Weissenbacher *et al.*, 1979, 1980) experimentally infected with Junin virus (JV), the causative agent of Argentine haemorrhagic fever (AHF). The clinical course in these monkeys was similar to that of severe human illness (Alvarez Ambrosetti *et al.*, 1959; Rugiero *et al.*, 1959).

Haematological alterations due to JV were first demonstrated in natural infection of man by Arribalzaga (1955) and in guinea pigs experimentally infected by González and Mejszenkier (1962), characterized by a severe leukopenia and thrombocytopenia. The leukopenia is due to decreased values of neutrophils, eosinophils and lymphocytes.

Infecting primates with the Machupo virus, the agent of Bolivian haemorrhagic fever, Kastello *et al.* (1976) found haematological changes similar to those described in human disease. In addition anaemia was observed the origin and role of which remained unknown.

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This paper describes the haematological changes developing in *C. jacchus* in the course of experimental infection with the pathogenic strain (XJ) of JV.

Materials and Methods

Virus. The stock virus was a pathogenic prototype strain of Junin virus (JV) passaged in suckling mouse brain (10^8 LD_{50}/ml). The stock was kept at −70°C and diluted at convenience in Hanks' solution before use.

Animals. Seventeen young adult male *Callithrix jacchus*, weighing from 250 to 300 g, were housed in cages in an isolated room at 24−26°C and moisture content of 50%. The monkey were fed with aqueous mixture of Nestum (Nestlé S. A.), Gevral proteina (Lederle Cyanamid Argentina S. A.) and powdered milk, as well as apples and bananas. This diet was supplemented with vitamins and minerals. Water was provided "ad libitum".

Experimental procedures. Six. *C. jacchus* were infected intramuscularly (i. m.) with 1000 LD_{50} of JV diluted in Hanks' solution. The dose was titrated simultaneously in suckling mice. Four other monkeys were similarly sham-inoculated with virus-free solution. Haematological values from seventeen uninoculated monkeys, along with those obtained before virus inoculation of the experimental animals were used to determine the normal ranges.

Daily observation of animals for clinical signs and recording of body weight and rectal temperature at regular intervals were carried out. Immediately after death, necropsies were performed and organ samples were taken for virological and histological examinations.

Blood samples for haematological as well as virological and serological studies were taken from all animals prior to inoculation. Finally, the animals were bled at days 7, 14, 18 and 21 p. i.; the sham-inoculated ones were bled on days 7, 14, 18 and 38. The results of virological and serological studies have been published elsewhere (Weissenbacher *et al.*, 1980).

Haematology. Using a syringe fitted with a 25 gauge needle, blood was obtained from the femoral triangle by puncture through the skin previously disinfected and coated with silicone grease. Heparin (1000 units/ml) was used as anticoagulant for haemoglobin, haematocrit as well as for red blood cell and reticulocyte counts. Blood for counting platelets and white blood cells and for differential smear counts was obtained immediately after puncture, by residual bleeding over the silicone grease coated skin in the absence of anticoagulant. Erythrocytes were counted in an improved Neubauer counting chamber; blood was diluted 1/200 with saline in a standard red blood cell pipette.

Haemoglobin concentration was determined as cyanhaemoglobin and haematocrit estimation was performed by the capillary method.

Reticulocytes were stained with cresyl brilliant blue and counted at 1000 times magnification.

For white blood cell and platelet counts, blood was diluted in a white cell pipette using Feissly and Ludin solution modified by Milani and Milani (1977) as diluent and counted simultaneously in a Neubauer counting chamber with phase contrast.

Both the differential counts of leukocytes and the evaluation of morphological characteristics of erythrocytes and platelets were performed in smears stained with May-Grünwald-Giemsa.

The bone marrow samples were obtained from four infected and one uninfected animal during necropsies and stained with May-Grünwald-Giemsa. During this work serial bone marrow samples were impracticable, due to the severe trauma of so small sick animals.

Data obtained from sham-inoculated and infected monkeys were analyzed by Student's test in order to calculate the means of differences between the values obtained on day 0 (before inoculation) and at intervals given above.

Results

Clinical findings

By day 14−15 p. i. six infected monkeys were obviously sick as compared to controls. The weight curve of virus-inoculated animals closely approximated the mean weight curve of sham-inoculated ones until day 16 p. i., but

Table 1. Normal haematological values in Callithrix jacchus (n = 17)

Cells	$\bar{x}$	SD	Range
Erythrocytes (µl)	7,176,470	209,565	5,400,000—8,300,000
Haemoglobin (g/100 ml)	15.13	0.47	11.68 — 17.44
Haematocrit (%)	52.08	1.40	41.00 — 60.40
Reticulocytes (%)	2.39	0.33	0.86 — 5.59
Indices:			
M.C.V. (μm^3)	73.08	0.93	68.50 — 84.20
M.C.H. (pg)	21.06	0.25	19.20 — 23.00
M.C.H.C. (%)	29.11	0.50	23.80 — 32.10
Platelets (µl)	464,823	52,515	201,000—1,164,000
Leucocytes (µl)	9,194	636.50	5,700— 14,300
Granulocytes:			
Neutrophils (µ')	2,163.61	357.44	840— 6,084
Eosinophils (µl)	227.23	52.88	0— 903
Basophils (µl)	102	41.26	0— 585
Monocytes (µl)	289.58	36.23	88— 615
Lymphocytes (µl)	6,411.60	598.92	2,143— 12,584
Erythroblasts (µl)	260.35	32.60	59— 516

afterwards it fell below control values. Signs of illness started with general depression and anorexia that progressed steadily until death. As compared to controls, the temperature of sick monkeys failed to show significant differences until a few hours before death. Temperature then decreased abruptly and the animals either died spontaneously with a marked terminal hypothermia or were killed at its onset between 19—24 days p. i. A few days before death all the monkeys suffered haemorrhages, mainly in gums. Besides, marked spasms and motor disorders were observed in two of them.

Haematological findings

The values given in Table 1 are coming from seventeen normal animals. In nearly each case the normal range was quite wide. The haematological values obtained for sham-inoculated animals failed to show significant alterations.

The values obtained from infected animals can be seen in Table 2. Red blood cell count, haematocrit and haemoglobin continuously decreased from day 7 p. i. onwards, dropping to 40% of the original values at day 21 p.i. Normocytic and normochromic indices were obtained.

Increased reticulocyte count was detected initially on day 7 p. i., while that of erythroblasts increased between days 7—14 p. i. Both declined lated practically to zero values. By erythrocyte counts over 1500—2000 no reticulocytes were found.

Due to the increase in neutrophils the leukocyte count at day 7 p. i. was higher than on day 0 and declined steadily until death. After initial icnrease of segmented neutrophils on day 7 p. i. these cells together with lymphocytes

Table 2. Haematological values in Callithrix jacchus infected with 1000 LD_{50} of JV at different days post infection

Days p. i.	0 n=6	7 n=6	14 n=6	18 n=6	21 n=4
Erythrocytes (µl)	7,175,000	6,595,000[b]	5,866,00[d]	5,515,000[d]	4,367,000[c]
	414,420	*374,320*	*380,070*	*330,900*	*74,480*
Haemoglobin (g/100 ml)	14.49	13.39[a]	12.03[d]	11.52[d]	8.98[d]
	1.04	*1.03*	*0.85*	*0.84*	*1.28*
Haematocrit (%)	52.95	48.60[d]	42.97[d]	40.45[d]	32.50[c]
	2.66	*2.86*	*2.90*	*2.66*	*5.15*
Reticulocytes (%)	3.29	4.29	1.79	0.07[d]	0[a]
	0.70	*0.60*	*0.55*	*0.01*	
Indices:					
M.C.V. (µm³)	73.80	73.70	73.23	73.23	74.78
	1.30	*1.78*	*1.75*	*1.58*	*1.17*
M.C.H. (pg)	20.20	20.22	20.47	20.80	20.75
	0.31	*0.62*	*0.49*	*0.49*	*0.52*
M.C.H.C. (%)	27.30	27.46	27.65	28.38	25.93
	0.76	*0.58*	*0.29*	*0.56*	*1.45*
Platelets (µl)	521,330	431,080	334.500[a]	209,917[b]	30,437[c]
	134,870	*62,818*	*101,956*	*52,807*	*8,442*
Leukocytes (µl)	9,650	12,450	9,417	6,159[d]	4,205[a]
	1,243	*2,506*	*1,446*	*1,093*	*121*
Granulocytes:					
Neutrophils (µl)	3,018	4,962[b]	3,508	2,333	1,752
	817	*1,066*	*1,069*	*862*	*649*
Eosinophils (µl)	125	219	72[a]	8[b]	0[a]
	38	*79*	*48*	*6*	
Basophils (µl)	83	167	155	88	51
	66	*42*	*59*	*23*	*40*
Monocytes (µl)	236	407	295	207	133
	47	*102*	*97*	*27*	*46*
Lymphocytes (µl)	6,188	6,695	5,387	3,523	2,269
	1,534	*2,716*	*613*	*295*	*511*
Erythroblasts (µl)	*252*	3,975[a]	1,388[c]	108[a]	2[a]
	58	*1,610*	*274*	*48*	*2*

Values represent mean ± 1 SE (*in italics*)
Significance: a: $0.05 > p > 0.025$ – b: $0.025 > p > 0.01$ – c: $0.01 > p > 0.005$ – d: $p > 0.005$

declined until day 18–21 p. i. when the lowest level was recorded. Although in two monkeys, eosinophils disappeared between days 7–14 p. i., in all of them these cells were absent by day 21 p. i.

Platelets decreased by day 21 p. i. to 5 or 10% of the initial count.

Bone marrow findings

Four bone marrow smears from experimental animals were analyzed. Percentage values of the principal types of cells are given in Table 3. Infected animals exhibited an obvious drop in myeloid cell count in two samples and in erythroid precursor count in three samples of bone marrow smears.

Table 3. Different types of cells in the bone marrow of infected and control monkeys

Bone marrow		Cells					
		Lymphoid	Myeloid	Erythroblasts	Blasts	Megakaryocytes	M/E ratio
Control		44.4*	31.0	17.0	1.6	Normal maturation	1.9
Infected animal	No. 1	67.7	17.3	3.3	1.3	Abundant Abundant immature forms	5.6
	No. 2	49.0	8.2	8.6	2.9	Isolated	1.5
	No. 3	40.5	21.4	29.0	0.8	Abundant immature forms	0.66
	No. 4	65.6	6.1	1.1	9.0	Isolated	15

* Percentages related to 1000 cells.

Two animals (2 and 4) had their three major bone series well below control values. Most bone marrow cells of two animals (1 and 4) were of lymphoid type. Furthermore, the latter (4) showed monocytoid cell infiltration of varied morphology, as well as a considerable increase of blasts.

The megakaryocytes found in 2 animals (1 and 3) were morphologically akin to those found in human idiopathic thrombocytopenic purpura of immunological etiology and other viral thrombocytopenias. They exhibited smooth edges, were free from inner granules and proved more basophilic than the mature cells.

Discussion

The clinical findings in infected animals showed the existence of a severe illness with remarkable haematological changes, as reported (Frigerio *et al.*, 1978; Weissenbacher *et al.*, 1979, 1980). The observed signs were similar to those found in guinea pigs infected with JV (Boxaca *et al.*, 1961; Guerrero *et al.*, 1977) and to those detected in various clinical forms of haemorrhagic fever in humans (Alvarez Ambrosetti *et al.*, 1959; Rugiero *et al.*, 1959). Of the six infected monkeys, two suffered severe haemorrhages, another two showed CNS involvement with minor haemorrhages, while the last two suffered slight gum hemorrhages only. No alterations were observed in uninfected animals. In contract, these animals gained weight and kept active during observation for four months.

The haematological values from seventeen normal animals agrees with those obtained from other members of *Callithricidae* family (*Saguinus fuscicollis nigriformis*, *S. f. illigeri*, *S. f. desillei*, *S. nigricollis* and *S. oedipus*), as described by Anderson *et al.* (1967). On the other hand, in the normal animals, we detected circulating erythroblasts mainly of the orthochromatic type. The fundamental haematological changes in infected animals appeared in the erythrocytic, thrombocytic and leukocytic series. Statistically significant normocytic, normochromic anaemia appeared as from day 7 p. i., followed by variations in the circulating reticulocyte and erythrocyte counts. Therefore, we may recognize two periods in these haematological changes. In the first stage, between 0 and from 7—14 days p. i., both reticulocyte and erythroblast counts increase, while erythrocyte, haemoglobin and haematocrit values decrease. This could be due to fast erythrocyte destruction, haemorrhages, or inefficient erythropoiesis, resulting from bone marrow failure caused by viral action.

In the second period (from 14 to 21 days p. i.) erythrocytes dropped sharply, the partial or total absence of reticulocytes and erythroblasts leads us to believe in the existence of hypoplastic or even aplastic bone marrow, more so if we tok into account the thrombocyte and leukocyte values. Reticulocytopenia also exists in humans (Suárez *et al.*, 1970), but anaemia appears only in patients who bleed copiously or, less often, it occurs during convalescence. This would be understandable if the human erythrocytes had a longer mean life than those of *C. jacchus*. The erythroblastosis observed in *C. jacchus* has not been detected in human AHF.

In guinea pigs, the appearance of a discrete normochromic normocytic anaemia without reticulocytopenia was described by day 11 p. i. (González and Mejszenkier, 1962). The ranges of leukopenia and thrombocytopenia observed in *C. jacchus* in peripheral blood are most likely due to various degrees of bone marrow failure. This conjecture is based on previous reports (Languens *et al.*, 1979) as well as on preliminary observations made on four bone marrow smears from infected animal necropsies which were compared with one from a normal monkey. In the latter comparison of bone marrow smears we have observed a remarkable decrease in one or more bone marrow series.

It must be stressed that in two animals there was an increase in immatur-megakaryocytes morphologically resembling those observed in other diseasee as dengue and measles of viral origin, as well as in idiopathic thrombocytopenic purpura with immunological etiology. One bone marrow smear exhibited a wide range of abnormal monocytoid cells, from macrophage-like vacuolated elements to typical monocytoid blast cells. Similar alterations in bone marrow were observed also in humans (Pirosky *et al.*, 1959; Gallardo, 1970; Elsner *et al.*, 1970, 1973; Ponzinibbio *et al.*, 1979) and guinea pugs (Carballal *et al.*, 1976, 1977; Oubiña *et al.*, 1980) infected with JV. In the latter, large amounts of viral particles in the platelet demarcation chanels of megakaryocytes were also detected. These alterations may explain the peripheral haematological changes (González and Mejszenkier, 1962; Nota *et al.*, 1969; Martínez Segovia and De Mitri, 1971) observed during the illness.

Unfortunately, the failure to obtain serial bone marrow samples in *C. jacchus* in the course of the disease in order to compare values with the findings in peripheral blood, seriously hindered the interpretation of results.

The observed thrombocytopenia in man, guinea pig and marmoset could equally well arise from disseminated intravascular coagulation phenomena.

It is not impossible that both mechanisms were simultaneously at work. However, only in a few severely ill humans do clinical signs and laboratory results indicate action of intravascular mechanisms (Agrest *et al.*, 1969). In a larger series of patients this mechanism could not been demonstrated (Molinas *et al.*, 1981).

Discussing similar haematological signs in guinea pigs infected with JV, Molinas (1978) rejected the coagulation mechanism, since thrombocytopenia had appeared before any visible changes in blood-clotting factors became manifest, pointing out the absence of fibrinogen degradation products in blood or urine and the lack of fibrin deposits in tissues. Likewise, Laguens *et al.* (unpublished data) failed to detect any histological evidence of intravascular coagulation in *C. jacchus* necropsies. Due to the low number of monkeys studied, no definitive conclusion regarding the intravascular mechanism can be reached at predsent time. Further work is required to clarify the situation.

The lymphopenia of infected *C. jacchus* could be explained by lymphoid tissue alterations. Preliminary histopathological studies in *C. jacchus* detected

multiple necrotic foci of the germinal centres and of the splenic red pulp (Languens *et al.*, 1979). Similar alterations of this tissue has been observed in humans (Elsner *et al.*, 1970, 1973; Gallardo, 1970) and in guinea pigs (Besuschio, 1966; Elsner *et al.*, 1976).

The findings described in this paper, together with those previously reported, support our contention that *C. jacchus* seems to be a suitable model for the study of pathogenesis, pathology, prophylaxis and therapeutic measures in AHF.

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