Case report

Platelet satellitism in infectious disease?

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Abstract

Background: Platelet satellitism is a phenomenon of unknown etiology of aggregating platelets around polymorphonuclear neutrophils and other blood cells which causes pseudothrombocytopenia, visible by microscopic examination of blood smears. It has been observed so far in about a hundred cases in the world.

Case subject and methods: Our case involves a 73-year-old female patient with a urinary infection. Biochemical serum analysis (CRP, glucose, AST, ALT, ALP, GGT, bilirubin, sodium, potassium, chloride, urea, creatinine) and blood cell count were performed with standard methods on autoanalyzers. Serum protein fractions were examined by electrophoresis and urinalysis with standard methods on autoanalyzer together with microscopic examination of urine sediment. Erythrocyte sedimentation rate, blood culture and urine culture tests were performed with standard methods.

Results: Due to typical pathological values for bacterial urinary infection, the patient was admitted to the hospital. Blood smear examination revealed phenomenon, which has persisted for three weeks after the disease has been cured. Blood smears with EDTA as an anticoagulant had platelet satellitism whereas the phenomenon was not observed in tubes with different anticoagulants (Na, Li-heparin) and capillary blood.

Discussion: We hypothesize that satellitism was induced by some immunological mechanism through formation of antibodies which have mediated platelets binding to neutrophil membranes and vice versa. Unfortunately we were unable to determine the putative trigger for this phenomenon. To our knowledge this is the second case of platelet satellitism ever described in Croatia.

Key words: blood platelets; thrombocytopenia; EDTA; urinary infection

Received: February 27, 2014 *Accepted: May 01, 2015*

Background

The phenomenon of "platelet satellitism" (PS) was first presented by Field and McLeod in 1963, in the British Medical Journal (1). The phenomenon has been observed in 14-year-old boy with neurological problems. Until now, this phenomenon has been described in about one hundred cases in people of different ages, suffering from various pathological conditions, including infectious diseases (2-4), as well as in healthy individuals (5-7). Under the influence of ethylenediaminetetraacetic acid (EDTA) as an anticoagulant, the platelets have the ability to create formations around polymorphonuclear neutrophils (PMN) (mostly granulocyte), monocytes and around basophilic and eosinophilic granulocytes (non-lymphocitic white blood cells), granulated lymphocytes (8) and even atypical lymphocytes (9).

In most cases, hematology analyzers recognize such formations as the white blood cell lineage, and consequently give a falsely low platelet count or pseudoplatelet syndrome (10). Furthermore, to our knowledge blood taken without the addition of EDTA (capillary blood) does not display the above in vitro phenomenon, nor venous blood taken with the addition of any other anticoagulant, such as sodium-citrate or lithium-heparin (5,6,11-14). *In vitro* grouping of the platelets around polymorphonuclear neutrophils (PMNs) and other

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cells imparting a rosette-like appearance is a phenomenon popularly called "platelet satellitism", and it occurs in blood samples anticoagulated only with K_3EDTA at room temperature. The phenomenon is only partially explained so it still remains a major challenge in the everyday routine work of specialists in laboratory medicine.

Pseudothrombocytopenia in general and as a consequence of "platelet satellitism" is a widespread problem present in a number of diseases, and sometimes remains undiscovered. It is particularly necessary to identify it and exclude possible *in vitro* interference. In Croatia, only one case of this phenomenon has been described so far, in a trauma patient treated for left femur fracture. Just like our patient, the patient from Trauma Clinic, among other, suffered from a urinary tract infection (15).

Our case involves patient with a urinary infection, in whom platelet satellitism persisted even after the successful treatment of the disease.

To date, the phenomenon of PS has not been completely clarified with certainty, but there are hypotheses involving both immune and non-immune mechanisms. Bizzaro *et al*. have presented evidence suggesting immunological binding of EDTA-dependent antiplatelet and anti-neutrophil IgG antibodies directly on glycoprotein complex IIb/IIIa of the platelet membrane as well as the Fc gamma receptor III (FcγRIII) of polymorphonuclear neutrophils, that indicates the existence of IgG antibodies that directly form a connection or "bridge" between platelets and neutrophils. In addition, the antibody did not react with platelets from a patient with type I Glanzmann's disease, nor with neutrophils from a patient with congenital Fc gamma RIII absence (NAnull phenotype), thus confirming both specificities. As in other literature cases, a clear correlation between the presence of IgG and a specific clinical situation, disease, or use of drugs could not be shown. Therefore, these antibodies, which are present in some normal, healthy individuals, might occur naturally. Because of the exposure of particular cryptoantigenic structures present on EDTA-modified platelet and PMNs, they may manifest themselves by triggering the PS phenomenon (16).

The working hypothesis of the PS phenomenon is that EDTA, by chelating Ca^{2+} ions, changes the platelet membrane and unmasks (crypto) antigenic structures on platelet GPIIb/IIIa and PMN neutrophils, which are then been recognized by IgG enabling a formation of a "bridge" between the two cells. Why PS patients produce these IgG (auto) antibodies and what is the mechanism of their interaction with target molecules on platelet and PMN membranes, is still unclear (15).

The functions of GP IIb/IIIa in platelet physiology are diverse. Although most functions are manifested after platelet stimulation, there are approximately 40 000 antibody molecules bound to the surface of platelets, indicating that there are probably 40 000 to 80 000 GPIIb/IIIa receptors on the surface of one unstimulated platelet, depending on whether the antibodies bind bi- or mono-valently. Thus, GPIIb/IIIa is probably the most dense adhesion/aggregation receptor present on any cell.

Christopoulos and Mattock suggest non-immunological process which includes linking of platelets via thrombospondin or some other alpha granule proteins such as P-selectin (11). Adhesion of platelets to neutrophils is possible by existence of an active stimulus presented on the surface of platelets (11,12,17).

According to Moreselli *et al*. at lower temperatures EDTA can accelerate the whole process by binding calcium ions, based on the fact that the GPIIb / IIIa antagonists binding depends on the calcium ions concentration in the plasma (6).

It is an interesting fact that kanamycin added to EDTA blood shows dispersion of platelets from neutrophils (18).

Espanol and colleagues in their study have shown that combination of morphologic studies of granulated lymphocytes with PS and immunophenotypic studies displaying CD16 positivity indicates that lymphocytes with natural killer cell activity could exhibit PS. CD16 is cell surface antigen also known as neutrophil antigen NA, HNA1 and has been identified as Fc receptors FcγRIIIa (CD16a) and FcγRIIIb (CD16b). These receptors bind to the Fc portion of IgG antibodies which then activate

the NK cell for antibody-dependent cell-mediated cytotoxicity. A lack of CD16 in a given population of neutrophils may indicate prematurity, as it could be caused by a left-shift due to neutrophilic leukocytosis induced by tissue necrosis or bacterial infection (9).

The aim of this report is to present a case of a 73-year-old female patient with a urinary infection in whom we have observed a platelet satellitism which has persisted even after the disease was successfully treated. With this report we wish to contribute to the global understanding of this rare phenomenon by providing some new information and discussing the possible mechanisms.

Case subject and methods

Case subject

A 73-year-old female patient was hospitalized at the University hospital for infectious diseases Dr. Fran Mihaljevic in Zagreb, Croatia, for a period of nine days, due to recurrent urinary tract infection (diagnosis: acute pyelonephritis, *E. coli*). Medical history revealed that the patient had suffered from rheumatic fever, underwent tonsillectomy, appendectomy and cholecystectomy, and had chronic gastritis and cysts in both kidneys. She was using esomeprazole on a regular basis and benzodiazepine and ketoprofen when necessary. The patient reported allergy to the following medications: Analgin® (Pliva, Croatia) and Buscopan® (Boehringer Ingelheim, Germany).

The patient was referred to the Hospital because of frequent, repeated urinary tract infections, shaking chills and current pain in the kidney area accompanied with frequent and painful urination.

Methods

After admission of the patient to the emergency room, along with the medical history, all routine biochemical tests were made (C-reactive protein (CRP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyltransferase (GGT), bilirubin, sodium, potassium, chloride, urea and creatinine (Table 1), blood culture, urinalysis and urine culture (Table 2) and also complete blood count (CBC) and erythrocyte sedimentation rate (ESR) (Table 3). Control testing was done on 3rd and 9th day of hospital stay, as well as serum protein electrophoresis test (9th day) and blood smear examination (3rd and 9th day of hospital stay and 23 days after hospitalization).

Biochemical analysis was performed using the Beckman Coulter AU 640 automated chemistry analyzer with analytical principle of spectrophotometry and potentiometry, with Beckman-Coulter (BC) assays (Beckman Coulter, USA), BC calibrator kits and Bio-Rad Lyphochek (Bio-Rad Laboratories, USA) assayed chemistry controls. Analytical methods were: photometry (glucose, AST, ALT, ALP, GGT, bilirubin, urea, and creatinine), indirect ISE-potentiometry (sodium, potassium and chloride) and latex agglutination (CRP).

Urine analysis were done on Roche Urysis 1800 (reflectance photometry) with Combur test stripes (Roche Diagnostics, Switzerland), original calibrators and Bio-Rad Liquichek (Bio-Rad Laboratories, USA) urinalysis controls. Urine for native urine sediment testing was centrifuged for 10 minutes at 395 x g and sediment was examined under the light microscope Olympus CH2-CHS (Olympus, Germany).

Concentration of total proteins and protein electrophoresis were assayed on Hydrasis (Sebia, France) using agarose gel electrophoresis systems. The inter-assay CV reported by the manufacturer for IgG was from 2.0% to 3.4%.

Blood count was analyzed on UniCel DxH800, Beckman-Coulter (Beckman Coulter, USA), with Beckman-Coulter reagents and original controls.

Blood smears were stained manually, using the May-Grunwald-Giemsa stein (Merck, Germany) and observed in the light microscope Olympus BX51, Camera Olympus SC20, Olyumpus-CMAD3 (Olympus, Japan). Peripheral blood smears were prepared within 2 hours of blood collection.

All analyses were done in fresh samples, within the appropriate time for analysis. We don't have information about fasting status of the patient for any of the periods during the blood analysis.

Peripheral blood samples were collected by venipuncture in 3 mL and/or 6 mL Vaccuete® serum tubes with clot activator (red cap), hematology tubes (K_3EDTA tubes, lavender cap), ESR tubes (sodium citrate 3.2%, black cap), tubes with lithium heparin (green cap), coagulation tubes with 3.2% (0.109 M) and sodium citrate (light blue cap) tubes (Greiner Bio-One, Kremsmünster, Austria).

After the discovery of PS, blood was analyzed in test tubes with sodium-citrate and lithium-heparin. Platelet count obtained from blood with the addition of Na-citrate, was multiplied by 1.1 to correct for sample dilution. Citrate anticoagulation reduces the ionized calcium concentration normally found in blood (19).

Blood for serum testing was centrifuged for 10 minutes at 2150 x g at 4°C on Hettich ROTINA35 centrifuge (Hettich, Germany).

After the discovery of the PS we had controlled conditions (analysis 15 minutes and 2 hours after phlebotomy, capillary blood smear and blood smear from venous blood) of blood samples manipulation.

While the patient was admitted to the emergency room, blood count, ESR, CRP and other biochemical analysis were made within an hour.

Results

Table 1 presents the results of biochemical parameters during hospitalization, Table 2 shows laboratory values of urine analysis during the hospitalization, while Table 3 indicate values of hematology parameters during and after hospitalization and Table 4 serum electrophoresis, respectively.

The results show a high concentration of CRP and bilirubin on the 1st day of hospitalization (Table 1), pathological urine constituents at 1st day (Table 2) as well as leukocytosis, mild thrombocytopenia (Plt = 129 x10^9/L) and high sedimentation rate (Table 3), all probably due to bacterial infection.

Intravenous antibiotic therapy was initiated after the patient was admitted to the hospital. Symptoms of inflammation, WBC count and CRP concentration have decreased after 3 days of hospitalization, as a sign of successful antibacterial treatment. Biochemical tests - CRP, AST, ALT, ALP, GGT and bilirubin (Table 1), urinalysis (Table 2), CBC and sedimentation rate (Table 3) were within the normal range on the 9th (last) day of hospitalization and the patient was discharged from the hospital.

On the 1st and 3rd day of hospitalization blood smears were not performed by microscope, fol-

Table 1. Laboratory values of biochemical parameters during hospitalization

Parameter	1st day	3rd day	9th day*	Reference interval
CRP (mg/L)	195.9	157.0	13.6	<5
Glucose (mmol/L)	6.0	-	-	4.4-6.4
AST (U/L)	19	-	18	8-30
ALT (U/L)	16	-	23	10-36
ALP (U/L)	54	-	63	64-153
GGT (U/L)	19	-	27	9-35
Bilirubin (µmol/L)	26.7	-	6.8	3-20
Sodium (mmol/L)	140	-	-	137-146
Potassium (mmol/L)	4.5	-	-	3.9-5.1
Chloride (mmol/L)	104	-	-	97-108
Urea (mmol/L)	6.8	-	-	2.8-8.3
Creatinine (µmol/L)	94	-	-	63-107

CRP - C-reactive protein; AST - aspartate aminotransferase; ALT - alanine aminotransferase; ALP - alkaline phosphatase; GGT - gamma-glutamyltransferase.
* Patient was discharged on 9th day.

Table 2. Laboratory values of urine analysis during hospitalization.

	1st day	RI	MEUS	9th day*	RI	MEUS
Albumin	2+	0	10-15	0	0	1-2 leukocytes
Ketone	3+	0	leukocytes,	0	0	
Urobilinogen	1+	0	6-8 erithrocytes,	0	0	
Leukocytes	3+	0	many bacteria	0	0	
Erythrocytes	4+	0		0	0	
Nitrites	pos(+)	neg(-)		neg(-)	neg(-)	
Blood culture	sterile					
Urine culture	E.coli > 10^6 + K. pneumonia = 10^4					

MEUS - Microscopic examination of urine sediment; RI - reference interval.
* Patient was discharged on 9th day.

Table 3. Laboratory values of hematology parameters during and after hospitalization.

	1st day	3rd day	9th day*	23 day**	Reference interval
WBC (x 10^9/L)	12.3	6.3	7.9	8.7	3.4-9.7
RBC (x 10^{12}/L)	4.76	4.37	4.33	4.67	3.86-5.08
HB (g/L)	140	127	124	137	119-157
HCT (L/L)	0.419	0.382	0.379	0.408	0.356-0.470
MCV (fL)	88.0	87.3	87.6	87.4	83.0-97.2
MCH (pg)	29.4	29.0	28.7	29.4	27.4-33.9
MCHC (g/L)	334	332	328	336	320-345
PLT K_3EDTA (x 10^9/L)*	129	132	232	160	158-424
MPV (fL)	10.4	10.1	9.4	9.6	6.8-10.4
PLT K_3EDTA (x 10^9/L)#	-	-	-	156	158-424
MPV (fL)	-	-	-	9.9	6.8-10.4
PLT Na-citrate (x 10^9/L)*	-	-	-	136	158-424
MPV (fL)	-	-	-	8.7	6.8-10.4
PLT Na-citrate (x 10^9/L)#	-	-	-	130	158-424
MPV (fL)	-	-	-	8.7	6.8-10.4
PLT Li-heparin (x 10^9/L)*	-	-	-	133	158-424
MPV (fL)	-	-	-	9.3	6.8-10.4
PLT Li-heparin (x 10^9/L)#	-	-	-	108	158-424
MPV (fL)	-	-	-	9.4	6.8-10.4
ESR mm/3.6 ks	58	-	52	-	5-28

WBC - white blood cells; RBC - red blood cells; HB - hemoglobin; HCT - hematocrit; MCV - mean corpuscular volume; MCH - mean corpuscular hemoglobin; MCHC - mean corpuscular hemoglobin concentration; PLT - platelet; MPV - mean platelet volume; ESR - erythrocyte sedimentation rate test.
* blood analysis 15 minutes after venous blood sampling
# blood analysis 2 hours after venous blood sampling
- blood analysis wasn't done
* Patient was discharged on 9th day.
** Control visit

Table 4. Laboratory values of serum proteins during hospitalization.

Protein fractions	9th day Rel %	Reference interval %
Albumin	52.5	59.8-72.4
α_1-globulins	4.1	1.0-3.2
α_2-globulins	17.2	7.4-12.6
β-globulins	10.5	7.5-12.9
γ-globulins	15.7	8.0-15.8
weak peak seen in gamma fraction		
total proteins g/L	72	66-81

lowing the routine policy in our laboratory and international recommendations (20). Longitudinal follow-up of our patient on the 9th day revealed that patient didn't have any microscopic differential blood count but significant increase in platelet count and a microscopic, differential blood count was made as per internal rules of the laboratory (Table 3). Platelet satellitism was discovered after the analysis of blood slides, after which we searched for blood smears from 1st and 3rd day, but only 3rd day smear was found (Figure 1).

Since the patient had been discharged from the hospital on the 9th day, the same day that PS was found, there was no possibility of taking a capillary blood sample nor for using other anticoagulant for determining complete blood count, but serum protein analysis (protein electrophoresis) was requested. Serum protein electrophoresis (Table 4) showed hipoalbuminemia, increased alpha$_1$- and alpha$_2$-globulins component which could be the consequence of acute-phase reaction. Due to the weak peak in gamma fraction and high ESR, the patient is under further, additional observation. Three weeks after discharge the patient came to the hospital for a follow-up visit (Table 3), during which satellitism was repeatedly observed after a blood smear examination (Figure 2).

Figure 1 shows smears made of blood sample with K_3EDTA as anticoagulant, 3rd and 9th day of hospitalization and 23 days after hospitalization and is obviously that all presents PS. Figure 2 and Figure 3 showed that only blood smears with EDTA as an anticoagulant, had PS while blood smears with citrate have no satellitism.

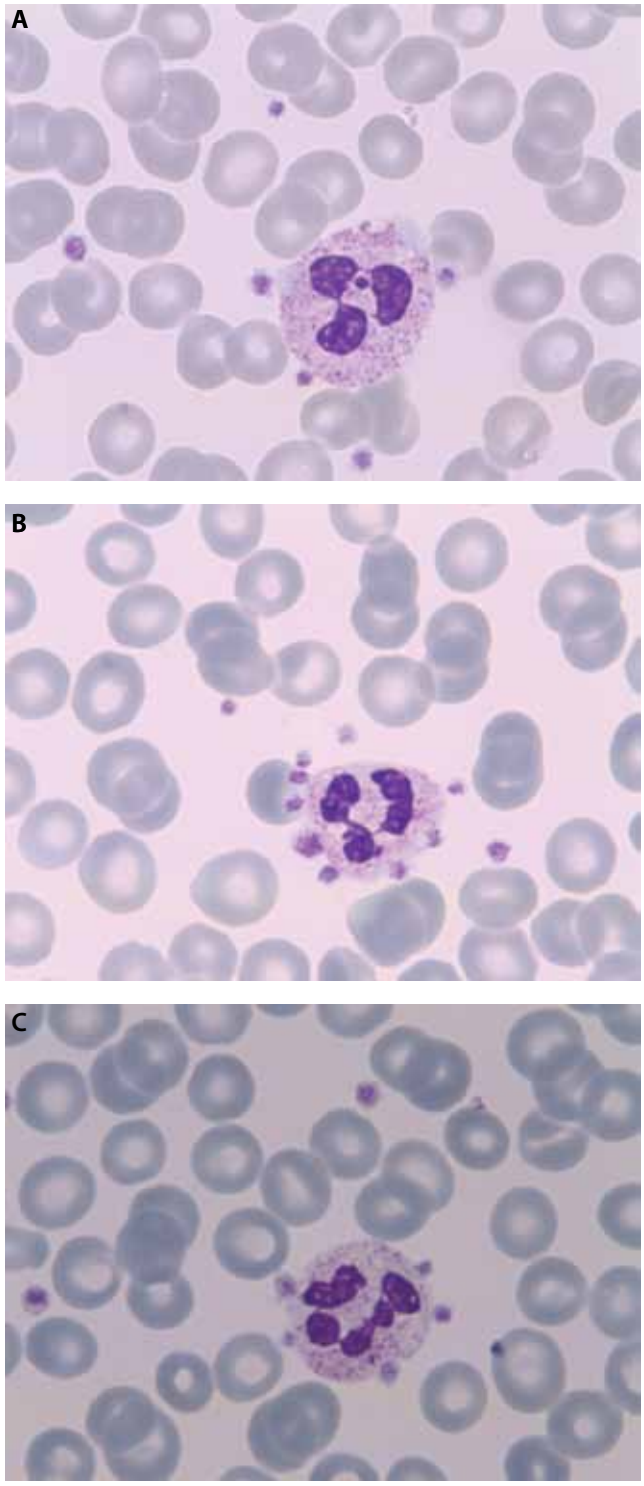

Figure 1. Blood smear (K_3EDTA) A: 3rd day, B: 9th day of hospitalization and C: 23 days after hospitalization.

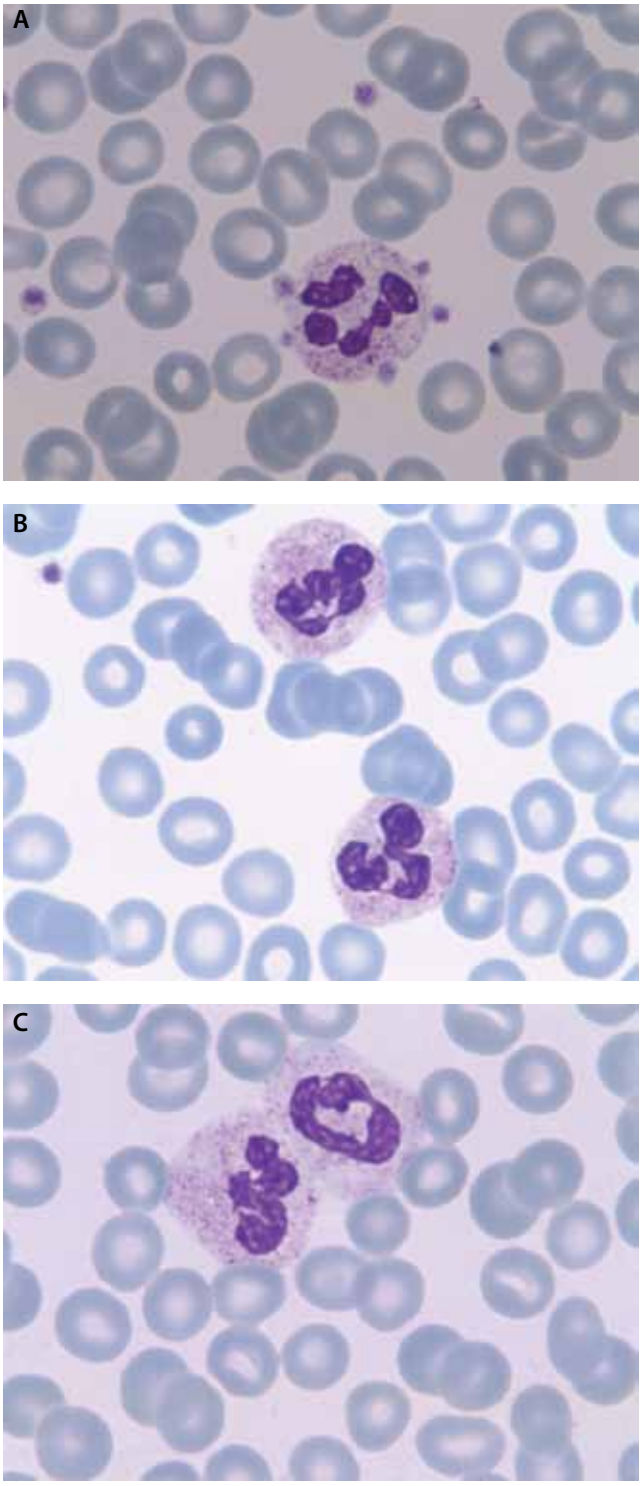

Figure 2. Blood smear 23 days after hospitalization - 15 min after blood sampling (A: K_3EDTA, B: Na-citrate, C: Li-heparin).

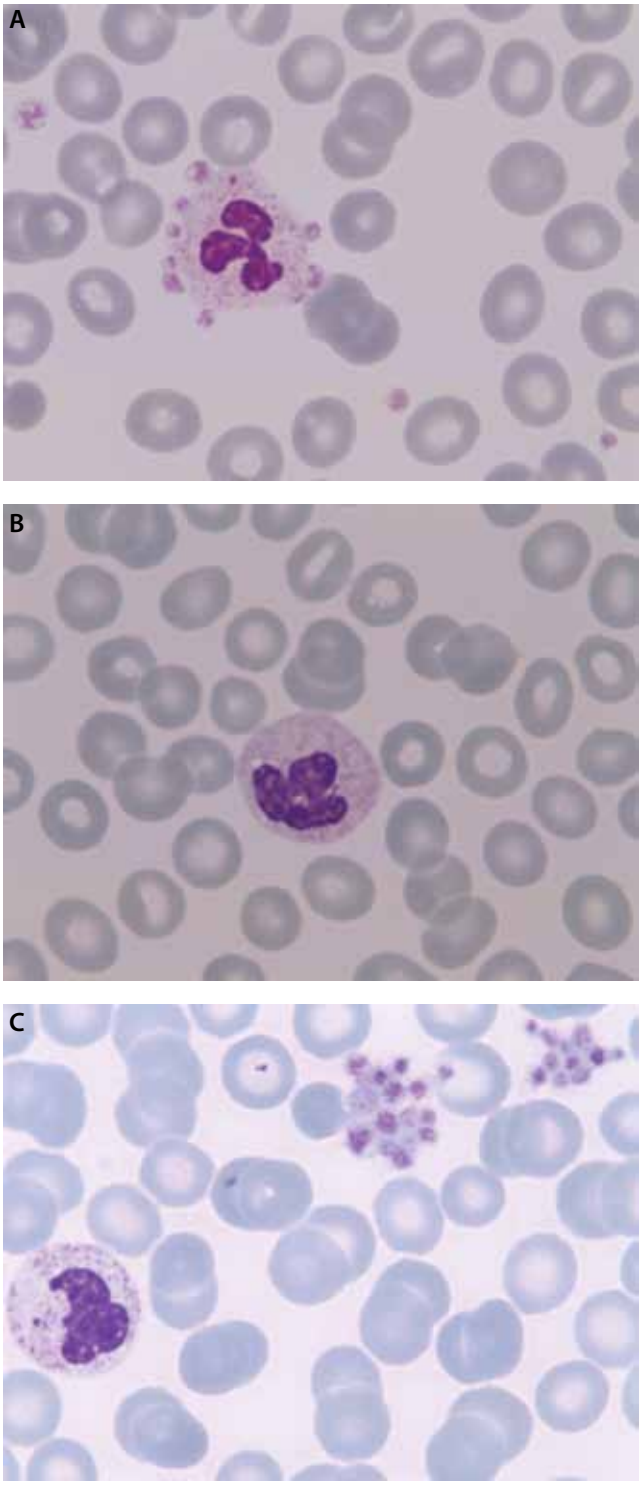

Figure 3. Blood smear 23 days after hospitalization - 120 min after blood sampling (A: K_3EDTA, B: Na-citrate, C: Li-heparin).

However, 23th days after hospitalization no significant differences were observed in the platelet count, in samples with different anticoagulant, during the period of time from sampling to the analysis (15 and 120 minutes). Exception was a blood sample with Li-heparin, where platelet

count was significantly lower 120 minutes after blood sampling (Table 3).

There is a possibility that the platelet count decreased because of *in vitro* thrombocytopenia and clusters of platelets induced by heparin (Figure 3). Heparin is a widely used anticoagulant. Because of its negative charge, it forms complexes with positively charged platelet factor 4 (PF4) on the platelet surface. This can induce anti-PF4/heparin IgG antibodies, resulting immune complexes that activates platelets.

Discussion

Pseudothrombocytopenia in general is a widespread problem, present in a number of diseases, and unfortunately sometimes remains undiscovered. Therefore, it is particularly necessary to carefully identify any false thrombocytopenia and exclude all possible *in vitro* interference (21). According to our experience and literature, mischaracterization of the platelet count can lead to serious problems in patient's condition, involving additional, unnecessary tests, non-invasive and invasive procedures such as bone marrow aspiration (22), surgery and blood transfusions (23), which can lead to serious life-long consequences for the patient, or even fatal outcome. Pseudothrombocytopenia is a well-known and solvable problem, unlike a rare phenomenon of platelet satellitism which is still fairly unknown among laboratory staff and therefore often not even recognized as a crucial finding. Cases of platelet satellitism due to urinary tract infection have been already reported in the literature (2,15) and observed in patients with vasculitis, lupus (12) and mantle cell lymphoma (13), marginal zone B-cell lymphoma (9,17), hepatocelular carcinoma (24), chronic lymphocytic leukemia (25), thrombocytopenic purpura (26), squamous cell carcinoma (22), cryofibrinogenemia (27), chronic alcoholism (28) as well as in healthy individuals (5-7). Cryofibrinogenemia may be primary (essential) or secondary to other underlying disorders, such as carcinoma, infection, vasculitis, collagen disease, or associated with cryoglobulinemia (29). Platelet satellitism is not normally associated with any platelet dysfunction (30). However, due to its low prevalence among the general population, insufficiently clarified mechanism and unknown causes, this phenomenon requires a written presentation in each new case.

In the literature we found information about the presence of the PS in a patients' blood during an infectious disease but with no further investigation, sometime after disease treatment, as we have done. The 48-year-old woman studied by Payne revealed recurrent, acute, urinary infection, and pharyngitis and had two predisposing conditions that could contribute to her PS, family history of cardiovascular disturbances and extremely long menstrual periods, which could consume a large number of platelets. Payne didn't say whether the patient has been monitored until discharge from hospital, when PS and is it disappeared (2). Kopčinović and Pavić reported a case of 91-year-old woman with left femoral fracture, who developed myocardial infarction, received transfusion therapy and developed urinary infection. The patient showed PS in blood smear which disappeared two days later and it was no longer present in the blood smear for the rest of hospitalization (15).

PS was also observed in healthy individuals. Jameel *et al*. was explaining a case of 55-year-old male having apparently good health, Morelli *et al*. observed 35-year-old women who have had a repeated mild thromocytopenia during routine check-up, but without any further explanation of this case as well as Hernández-Chavarría and Vega with a 21-year-old man (5-7). However, since health status was not appropriately explained in neither of the above mentioned cases, it is rather questionable whether the patients have indeed been healthy or have suffered from some unknown underlying disease.

In case of our patient the reasons for PS have not been completely understood. This case presents phenomenon in patient with an infectious disease and it still remained 17 days after curing the disease in samples with EDTA along with the in vitro thrombocytopenia and presence of platelet clusters induced by heparin.

The question arises whether our case is the phenomenon of a healthy individual, impact of infec-

tion or is there some third explanation? Unfortunately we can only speculate about the real causes behind this phenomenon, exposing the facts and conditions under which it was found, with certain limitation of the study. Given the fact that our patient had a chronic disease, high concentration of gamma–globulin and weak peak seen in gamma fraction, we were not able to speculate about the possible true cause-effect relationship responsible for PS.

Each new case has to be reported in order to raise awareness of laboratory staff of the existence of PS and thus contribute to the resolution of the causes of this phenomenon and also importance of pseudothrombocytopenia detection, in general. Possible explanation for our case could involve immunological mechanism that includes formation of antibodies and subsequent binding of platelets to target neutrophil membranes and vice versa. The trigger is not known especially because PS didn't disappear even after the patient condition improved.

To our knowledge this is the second case of platelet satellitism ever described in Croatia. Although in the absence of lack of ability to generalize, any new information can improve global understanding of possible mechanisms and nature of its appearance, in people suffering from various diseases as well as in healthy individuals.

Potential conflict of interest

None declared.

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