Comparative Characteristics Of Rheological Properties Of Blood In Combined Face Injuries Before And After Treatment

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ABSTRACT

Fractures are one of the most common components in multiple and associated injuries. Fractures of the lower jaw are observed in 76%, of the upper jaw in 24%, a fracture of the zygomatic-orbital complex in 12%. The actual problem of modern traumatology is the violation of reparative osteogenesis with injuries combined with facial trauma. The duration of the consolidation of fractures is determined by the degree of microcirculation disorders in the fracture zone, changes in cellular and coagulation hemostasis and mineral metabolism. In this connection, increasing the effectiveness of treatment of fractures of the bones of the face on the basis of early diagnosis of vascular and hemostasiological changes is of great importance for practical health care.

KEYWORDS

Fractures of the mandible, hemostasis, platelet aggregation, blood plasma.

INTRODUCTION

Fractures of the lower jaw are the most common among all fractures of the bones of the facial skeleton and, according to different authors, range from 75 to 96.5%, and 28-36% of the total number of inpatient dental patients [1]. The high incidence of fractures of the lower jaw is explained by its extended ("borderline") position and relatively large size [2,3]. Traditionally, it is believed that the main reasons contributing to the occurrence of

complications are late treatment of victims for medical care, diagnostic errors and incorrect treatment tactics at the prehospital stage, inaccurate reduction and inadequate fixation of fragments, incorrect tactics in relation to the tooth in the fracture gap [4].

Despite the constant improvement of the methods of complex treatment of mandibular fractures and their introduction into clinical practice, the frequency of pyoinflammatory complications remains high and fluctuates, according to different authors, from 9 to 41%, which necessitates further study of this issue [5]. There is no doubt that the high risk of developing infectious and inflammatory complications in a fracture is determined by the anatomical and physiological features of both the lower jaw itself and the soft tissues surrounding it, as well as the presence of a significant amount of opportunistic microflora in the oral cavity [6].

In 67-82% of cases, fractures of the lower jaw are localized within the dentition and, therefore, are open [7]. In this regard, some foreign authors call such fractures already initially complicated due to infection of the bone wound with pathogenic microflora [8]. Malnutrition, taking antibiotics, a stressful situation with injuries of the PMO, together with the influence of other unfavorable environmental factors, lead to a decrease in general and local immunity, metabolic disorders in most patients [9]. A serious cause of the development of inflammatory complications is a violation of the blood supply to damaged tissues, aggravated by the development of post-traumatic edema [10,11]. In case of fractures of the lower jaw due to the presence of fixing structures in the oral cavity, microcirculation of periodontal tissues and the process of self-cleaning in the oral cavity are

sharply disturbed [12]. In this regard, the number of pathogenic microorganisms on the surface of the teeth and mucous membranes increases, and the likelihood of infection of the wound substrate increases [13]. This is only a small part of the known factors that disrupt the processes of reparative regeneration in fractures of the lower jaw and contribute to the development of complications [14].

The disadvantage of most of the known methods of treating mandibular fractures is the effect mainly on certain unfavorable factors: bacterial factor, microcirculation system, reparative osteogenesis, immune status, etc. Moreover, most of the drugs used for treatment are of a chemical nature and, as a result, have a toxic effect, have many side effects, destroy, in addition to pathogenic, and normal microflora, cause allergic reactions [15].

The combined use of several drugs to influence various links of pathogenesis will inevitably lead to the summation of their undesirable effects. In this regard, it is especially important to search for more advanced drugs that are devoid of these drawbacks and, at the same time, have a multicomponent effect: antibacterial, anti-inflammatory, antioxidant, immunomodulatory, anabolizing, stimulating the processes of reparative bone regeneration.

PURPOSE OF THE STUDY

Study of the state of the hemostasis system in patients with injuries associated with facial trauma in acute trauma in the immediate and postoperative period.

MATERIAL AND METHODS

The study included patients who were treated in a multidisciplinary TMA clinic with damage to

associated facial injuries: 56 male patients from 18.1 to 50.2 years old, mean age - 34 years. Inclusion criteria - combined facial injuries. Exclusion criteria: severe anemia, atherosclerotic and diabetic vascular lesions, chronic inflammatory diseases in the acute stage. According to the location of the fractures, the patients were divided into 2 groups. Group 1 - 21 patients with fractures of the upper jaws, Group 2 - 35 patients with fractures of the lower jaws. The control group consisted of 20 people, practically healthy men without traumatic injuries. To study the hemostasis system, venous blood was obtained from the cubital vein with a wide needle into plastic tubes in accordance with the recommendations of the Z.S. Barkagan and A.P. Momota (2001). The blood was immediately mixed with a 3.8% sodium citrate solution in a 9: 1 ratio. To study the function of platelets, platelet-rich plasma was obtained for which the blood was centrifuged at 1000 rpm (140-160g) for 7 minutes. It was centrifuged at 3000-4000 rpm (1200-1400 g) for 15 min at room temperature (+ 18... + 25 ° C). The resulting platelet-rich and platelet-poor plasma (PRP) was used in studies during the first 2 hours. When evaluating the hemostasis system, general parameters were determined using kits and reagents from Technologia-Standard Barnaul, NPO Renam Moscow:

activated partial thromboplastin time (APTT) according to Caen et al. (1968); thrombin time according to Biggs, Macfarlane (1962); the level of soluble fibrin in plasma - orthophenanthroline test according to V.A. Elykomov, A.P. Momotu; XIIa-dependent lysis of euglobulins according to G.F. Eremin, A.G. Arkhipov; the concentration of fibrinogen in plasma according to Clauss; activity of antithrombin III (AT-III) according to Abildgaard; screening of disorders in the protein C system - according to the assessment of the normalized ratio, which was determined in the PPP before and after the introduction of the protein C activator into it (patent No. 2184976, Russia); Studies of platelet hemostasis: spontaneous aggregation of platelets in the blood according to N.I. Tarasova; determination of platelet aggregation activity on a Biola LA 230-2 aggregometer with inductors; this method used aggregation inductors manufactured by TekhnologiyaStandart (Barnaul), NPO Renam (Moscow), in the following concentrations: ADP - 2.5 µg / l (2-10 µM); adrenaline - 0.5 µg / l; collagen - 4 µg / ml (prepared according to the method of A.S. Shchitikova). Aggregation was recorded for 2 minutes with fixation of time values, then, if necessary, the degree of ADP release was assessed.

Table 1

State of vascular-platelet hemostasis

Name indicators	control n=20	Study timing.					
		After trauma		After treatment		10–11 days after treatment	
		1 group n=21	2 group n=35	1 group n=21	2 group n=35	1 group n=21	2 group n=35
n-r thrombocytes ×109/л.	247,9±4,46	235,4±8,8	* 190,5±9,2**	* 224,5±6,1	* 185,4±7,1 **	243,4±8,5	* 209,2±7,2 **
Spontaneous Aggregation platelets %	24,4±2,5	26,5±3,2	29,1±1,8	28,3±4,1	* 32,3±2,7	25,8±5,1	* 31,2±1,6
ADP-aggregation %	90,1±4,66	93,2±3,7	* 108,2±2,1	96,4±4,1	* 114,3±4,5	92,8±5,1	* 101,6±3,2
Adrenalin-aggregation %	96,9±2,40	99,3±3,1	* 110,9±3,7 **	101,2±4,3	* 114,5±5,8 **	98,5±4,3	* 109,7±3,4 **
Collagen-aggregation %	90,50±3,89	99,5±5,4	* 120,8±4,1	* 107,3±3,5	* 125,1±7,3	* 101,4±2,8	* 112,1±6,2
Thrombin-aggregation ,%	98,54±4,47	103,5±4,3	* 117,5±6,5	108,1±6,2	* 120,8±6,2	99,3±4,1	* 116,8±5,5
Activate f-ra Von Willebrand in plasma %	90,87±3,5	102,1±3,7	* 132,5±3,1 **	* 109,3±6,2	* 142,2±3,3 **	104,2±3,1	* 125,1±2,4 **

Note: * - values that reliably (P <0.05) differ from the control; ** - values of reliability between 1st and 2nd groups. (P<0,05)

Studies with universal inductors of aggregation were carried out under standard conditions, in the daytime under natural light, at a room temperature of at least 18 ± 2.5 ° C, relative humidity 45 ± 5%, visually. The aggregation time of normal platelets depends on their number; the more platelets in PRP, the faster the reaction develops. Therefore, we used the plasma of patients, in which the platelet count varied from 100 × 109 / L to 400 × 109 / L. The lengthening of the aggregation time by more than 3 s in comparison with the control parameters indicated the hypoaggregation of platelets with the universal inducer, and the

shortening of the time indicated hyperaggregation. A decrease in light transmission for the introduction of an inducer (ADP, collagen, adrenaline) indicates hypoaggregation, and an increase in the percentage of hyperaggregation. The von Willebrand factor activity was determined on a Biola LA 230-2 analyzer. The quality control of the coagulation tests was carried out using RNP-plasma (reference pooled plasma of donors) or standard-plasma (standardized for the activity of the determined component) for methods of quantitative determination of the activity of plasma procoagulants. All studies were carried out in the post-traumatic, postoperative periods and on the 10th day after the operation.

RESULTS AND DISCUSSION

Changes in hemostasis were characterized by activation of the coagulation potential, decreased clotting time after injury on the upper jaw 210 ± 9.3 s 201 ± 5.1 on the lower, despite the introduction of standard doses of fraxiparine (0.3 U). The subsequent decrease in the postoperative period to 192.2 ± 4.1 s with PVK and 180.1 ± 8.1 s with PNK, with the preservation of the reduced indicators on day 10 to 175.3 ± 5.2 s with PVK and $170.2 \pm 11,2$ s at PNK. Spontaneous platelet aggregation was higher than the control value. The patients showed changes in platelet membrane activation, confirmed by increased aggregation with various inducers of ADP-aggregation - $93.2 \pm 3.7\%$ with fractures of the upper jaw (UJ) and $108.2 \pm 2.1\%$ with fractures of the mandible. Adrenaline aggregation was 99.3 ± 3.1 and $110.9 \pm 3.7\%$, respectively. After trauma, the level of collagen-induced aggregation was $120.8 \pm 4.1\%$, which is higher than in the control group. With PVK, the indicators did not differ significantly from the control and amounted to $99.5 \pm 5.4\%$.

After trauma, changes in the intracellular activity of platelets were noted, the thrombin-aggregation index for PNA was $117.5 \pm 6.5\%$, which is significantly higher than the control level p p <0.05. An increase up to 103.5 ± 4.3 is characteristic of the UJ. An increase in the activity of von Willebrand factor (VWF) in blood plasma was determined with PVK - $102.1 \pm 3.7\%$ and PNA - $132.5 \pm 3.1\%$, which is 1.11 and 1.42 times higher than the control indicator, respectively (p <0.05). In the early postoperative period, hemostasis disorders persist, there is a further tendency to a decrease in the number of platelets, which is more pronounced by 25.12% with PNA. Spontaneous platelet aggregation remained accelerated by $28.3 \pm 4.1\%$ for PVK and $32.3 \pm 2.7\%$ for PNA. An increase in the induced ADP-aggregation was determined: $96.4 \pm 4.1\%$, $114.3 \pm 4.5\%$, respectively. There was also an accelerated aggregation with the adrenaline inducer, compared with the control, 18.16% - with PNA and 4.44% - with UJ. After the operation, abnormalities in the parameters of parietal platelet activation were revealed. The level of collagen-induced platelet aggregation was $107.3 \pm 3.5\%$ for PVK and $125.1 \pm 7.3\%$ for PNA, which is significantly higher than the control standard by 18, 56% and 38.23%, respectively. After the operation, increased intracellular platelet aggregation was determined: the level of thrombin-induced aggregation was $120.8 \pm 6.2\%$ for PNA, which significantly exceeds the control group by 1.2 times (p <0.05). On the 10th day of the postoperative period, positive trends were revealed in the state of cellular hemostasis, but the main changes remained. Thus, the number of platelets in the smear - with fractures of the upper jaw practically returned to the control values and amounted to $243.4 \pm 8.5 \times 10^9$ / L. In case of fractures of the lower jaws, the number

of platelets, as before, differed from the control and amounted to 209.2 ± 7.2 x 109 / L. Spontaneous platelet aggregation with PVK was equal to - 25.8 ± 5.1%, and with PVK 31.2 ± 1.6%, which is higher than the control standard by 5.74% and 27.87%, respectively (p <0.05) ... Induced platelet aggregation was determined with various inducers: ADP-aggregation was 92.8 ± 5.1% with PVK and 101.6 ± 3.2% with PNA. The level of aggregation with adrenaline was also higher than the control value in PVK and PNA and amounted to 98.5 ± 4.3% and 109.7 ± 3.4%, respectively, which significantly differed from both the control and the indicators of group 1. By the 10th day after surgical

treatment, disturbances in parietal hemostasis persisted, the level of collagen-induced aggregation was accelerated and amounted to 112.1 ± 6.2% in PNA and 101.4 ± 2.8% in PVK, which significantly differed from the control values. The index of thrombin-induced aggregation in PNA was 116.8 ± 5.5%, which remained significantly higher than the control (p <0.05). With UJ, the values approached those of the control group and amounted to 99.3 ± 4.1%. The activity of von Willebrand factor in blood plasma was 1.34 times higher than the control value, (p<0,05).

Table 2

Indicators of plasma coagulation hemostasis

Indicators of plasma coagulation hemostasis	Control group n=20	Study timing					
		After trauma		After treatment		10–11 days after treatment	
		1 group n=21	2 group n=35	1 group n=21	2 group n=35	1 group n=21	2 group n=35
APTV, s	37,3±0,9	34,5±1,9	32,2±1,5	33,1±2,7	* 30,1±2,4	* 33,7±0,7	* 31,1±0,6
Concentration fibrinogen, g/l	3,2±1,72	4,9±1,3	* 6,9±1,2	5,7±5,1	* 8,5±4,7	4,1±3,2	* 7,9±0,52
Tv, s	15,2±0,15	16,4±3,4	17,2±1,2	17,3±2,6	* 20,2±1,2	15,9±4,1	* 18,85±1,2

Note: * - values that reliably (P <0.05) differ from the control; ** - values of reliability between 1st and 2nd groups. (P<0,05)

The study of the parameters of plasma-coagulation hemostasis in patients with concomitant facial trauma revealed a syndrome of transient hypercoagulation (Table 2). After injury, significant differences from the control were revealed in terms of APTT coagulation indices - 34.1 ± 0.9 s with PVK and 32.2 ± 1.5 s with PNK. The concentration of

fibrinogen in blood plasma significantly increased with a PNA of 6.9 ± 1.2 g / l (p <0.05). The thrombin time was lengthened and amounted to 16.4 ± 3.4 s for PVK and 17.2 ± 1.2 s for mandibular fractures. After the operation, changes in the main coagulation samples were determined. Differences from the control values were obtained in terms of APTT - 30.1 ±

2.4 s with PNA, in terms of fibrinogen level 8.5 ± 4.7 g / l, thrombin time 20.2 ± 1.2 s (p <0.05). On the 10th day, the stabilization of APTT indices from 31.1 ± 0.6 s with PNK was noted. The content of fibrinogen in blood plasma remained elevated, 4.1 ± 3.2 g / l with PVK and approached the control values. But with PNA it significantly differed from the control 7.9 ± 0.52 g / l, while the thrombin time was lengthened and amounted to 18.85 ± 1.2 s. For a more complete study of hemostasiological changes in combined facial injuries, the indicators of fibrinolysis were studied (Table 3).

Table 3

Fibrinolysis indicators

Indicators	control n=20	Study timing.					
		After trauma		After treatment		10–11 days after treatment	
		1 group n=21	2 group n=35	1 group n=21	2 group n=35	1 group n=21	2 group n=35
XIIa-dependent fibrinolysis, min	8,3±2,1	10,5±1,3	* 15,2±2,9	* 16,2±4,7	* 25,5±2,1 **	11,5±2,6	* 16,2±1,7
Concentration RFMK, mg per 100 ml	8,3±1,6	* 16,3±0,4	* 17,0±0,7	* 19,1±1,1	* 23,8±1,4 **	* 17,1±0,9	* 21,4±1,4 **
Activity protein C,%	100,8±4,6	97,2±2,4	* 88,3±4,8 **	* 91,4±3,1	* 82,4±4,3 **	95,4±3,7	96,2±3,7
Activity antithroma bin-III	90,5±4,7	86,3±2,5	* 82,1±1,7	* 81,5±4,2	* 77,4±4,6	89,3±2,5	87,2+4,5

Note: * - values that reliably (P <0.05) differ from the control; ** - values of reliability between 1st and 2nd groups. (P<0,05)

In the post-traumatic period, the values for group XIIa of dependent fibrinolysis were 15.2 ± 2.9 min for PNA, which is significantly higher than the control indicator; with PVK, the increase was up to 10.5 ± 1.3 min. An increased level of RFMK concentration of 16.9 ± 0.4 mg per 100 ml with PVK and 17.0 ± 0.7 min per 100 ml with PNA was revealed, both indicators significantly differed by 100 ml with PNA, both indicators significantly differed from the control values. The level of protein C in PNA decreased to 88.3 ± 4.8%. Also, the level of anticoagulants - antithrombin III - 86.3 ± 2.5%, which was insignificantly reduced in PVK, and a significant decrease in control with PNA 82.1 ± 1.7%. After the operation, further lengthening of fibrinolysis was determined. The level of XIIa-dependent fibrinolysis was 25.5 ± 2.1 min with PNA. A persistent elevated level of RFMK

concentration of 23.8 ± 1.4 mg per 100 ml was revealed, more pronounced in PNA. Decreased activity of procoagulant factors. The activity of antithrombin-III was 77.4 ± 4.6% with PNA. A decrease in the level of protein C was detected at PNA 82.4 ± 4.3%, which distinguished these indicators from control by 16%. On day 10, the indicators of XIIa-dependent fibrinolysis were 16.2 ± 1.7 min with PNA, while maintaining a significant difference from the control values. There was also an increased level of RFMK concentration, which was more pronounced with PNA, and amounted to 21.4 ± 1.4 mg per 100 ml. Also, an insignificantly reduced level of anticoagulants - antithrombin III - 89.3 ± 2.5% with PVK and 87.2 + 4.5% - with PNA was determined. The level of protein C was reduced, but did not differ significantly from the control. In patients, inhibition of the fibrinolysis system (significant lengthening of XIIa-dependent fibrinolysis) was revealed, which was accompanied by an increase in the content of RFMK fibrin degradation products in plasma and against the background of a decrease in the activity of anticoagulant factors and protein C, which is associated with depletion of their reserve potential. These changes against the background of enhanced parietal and intravascular coagulation activation under conditions of endothelial dysfunction can determine a more pronounced degree of thrombinemia and the development of microthrombotic changes.

CONCLUSION

The state of systemic hemostasis in a patient with combined facial injuries is characterized by early pre-endothelial and endothelial platelet activation (adrenaline and collagen hyperaggregation). Strengthening the prothrombogenic potential of the vascular wall (von Willebrand factor), depletion of the anticoagulant reserve (protein C), along with changes in plasma coagulation hemostasis and fibrinolysis. Changes in the indicators of vascular platelet, plasma coagulation hemostasis and fibrinolysis depend on the location of the injury. The most pronounced disorders in all mechanisms of platelet activation (endothelial, membrane, intracellular), the system of plasma-coagulation hemostasis and fibrinolysis are observed in PNA, especially in the postoperative period. The persisting changes in systemic hemostasis on the 10th day of observation, mainly with PNA, necessitate a longer use of antiplatelet and antithrombin drugs.

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