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BIOCHEMICAL INDICATORS OF INFLAMMATION AND DYSBIOSIS IN THE ORAL CAVITY OF PATIENTS WITH MANDIBULAR FRACTURES ON THE BACKGROUND OF THE HEPATO-BILIARY PATHOLOGY

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Abstract

<u>Purpose</u>: To determine the effect of hepatobiliary pathology (HBP) on the development of dysbiosis and inflammation in the oral cavity of patients with fractures of the lower jaw (mandible).

<u>Methods</u>: 157 patients with HPV were surveyed, 57 of them were diagnosed with HBP. The state of the oral cavity was assessed by biochemical parameters of saliva: elastase activity and MDA content (inflammation markers), urease activity (bacteriological contamination marker), lysozyme (nonspecific immunity index), catalase activity (antioxidant enzyme). Mixed oral fluid was collected on the 1st day and on the 14th day after standard treatment according to the protocol. <u>Results:</u> In patients with fractures of the jaw increases the activity of elastase and urease, reduced activity of lysozyme and catalase, especially in patients with HBP. The treatment reduces the activity of elastase and urease and increases the activity of catalase. In patients with HBP standard treatment does not reduce the activity of elastase and urease and does not increase the activity of catalase and lysozyme.

<u>The conclusion</u>: HBP complicates inflammation and dysbiosis in the oral cavity in patients with fractures of the jaw, which requires the use of hepatoprotectors.

Key words: jaw fractures, hepatobiliary pathology, inflammation, dysbiosis

INTRODUCTION

Hepatobiliary system plays an extremely important role in the physiology and pathology of the tissues of the oral cavity [1, 2]. In the mechanism of the pathogenesis of many dental diseases, the violation of the antimicrobial function of the liver [3] plays an essential role, which has been embodied in hepatotoxic biliary syndrome [4].

The participation of the liver in the mineral metabolism, in particular, in the processes of bone mineralization [5, 6], may be an important cause of the development of various diseases of the maxillofacial system [7, 9].

The purpose of this work was to determine the peculiarities of the development of dysbiosis and inflammation in the oral cavity of patients with fracture of the mandible on the background of HBP.

MATERIALS AND RESEARCH METHODS

Under our supervision, there were 156 patients with fractures of the mandible that received fractures within the dentition: angular, jaw, mental, middle. In 57 patients with fractures of the mandible, hepatotoxicity was detected (Table 1).

Pathology	Absolute number	%
Diarrhea of the gall bladder and biliary tract	26	45,6
Cholecystitis	10	17,5
Chronic hepatitis	9	15,9
Cholangit	8	14,0
Cholelithiasis	4	7,0
Total	57	100

Table 1. Hepatobiliary pathology of patients with fractures of the mandible

The examination of patients with HBP was made at the sity clinical hospital of firstaid (Vinnytsia). In patients with fractures of the mandible and in patients with fractures against the background of hepatotoxicity, the condition of the oral cavity was assessed on the first day and on the 14th day of standard treatment. All patients were given double-jaw splinting with Tigerstedts splints with hinged loops and tooth removal from the fracture line. Conservative treatment consisted of Lincomycin 30% in 2 ml 3 times a day, Loratadine 0.01 in 1 tablet per day, Hexavit in 1 dose of 3 times a day, Analgin in 1 tablet 2-3 times a day for 5 days, as well as cold to the damaged area for the first 3 days in one hour 6-8 times a day.

In all patients, oral fluids were collected on the first and on the 14th day of treatment. The level of markers of inflammation [10] was determined in the oral fluid: the activity of the proteolytic enzyme elastase [11] and the content of malondialdehyde (MDA) [12]. The level of bacterial insemination was evaluated by the activity of the bacterial enzyme urease [13], the state of non-specific immunity was determined by the activity of lysozyme [14] and the ratio of relative activity of urease and lysozyme was calculated by the degree of dysbiosis by AP Levytsky [15]. Antioxidant activity was assessed by the level of catalase [10] and by the antioxidant-prooxidant index API (ratio of catalase and MDA) [10].

The results were subjected to standard stats processing [16].

RESULTS AND DISCUSSION

According to our data, in 36.5% of patients with fractures of the mandible there was a hepato-biliary pathology of different genesis. These data may indicate that hepato-biliary pathology can be a beneficial backdrop for the development of bone pathology. Table 2 presents the results of determining the level of biochemical markers of inflammation in the oral fluid of patients with fractures of the mandible. It is seen that in patients with fractures twice as high elastase activity and it remains significantly higher even after 2 weeks of standard treatment.

In patients with fractures in the background of HBP, the activity of elastase is higher than the control rate by 2.4 times and remains high even after treatment. Regarding the second biochemical marker of inflammation, MDA, it does not significantly increase in patients with fractures (by 20.7%) and in patients with fractures in the background of DBP (by 24.4%).

Table 3 shows the results of the determination of the antioxidant enzyme catalase activity in the oral liquid and the API index. From these data it is clear that fractures of the mandible reduce the activity of catalase by 25% and the index of API by 38.6%, however, in patients with fractures against the background of HBP, the activity of catalase and the API index is halved. Treatment increases the level of catalase and the API.

 Table 2. Level of markers of inflammation in the oral fluid of patients with fractures of the mandible on the background of hepato-biliary pathology (HBP)

№	Indicators	n	Elastase, mc- cat / 1	MDA, mmol/l
1	Control	10	$0,38 \pm 0,05$	$0,\!45 \pm 0,\!04$
2	Fractures of the lower jaw	13	$0,75 \pm 0,09$	$0,\!57 \pm 0,\!07$
	1st day		p<0,01	p >0,05
3	Fractures of the mandible on the	14	$0,92 \pm 0,10$	$0,56 \pm 0,06$
	background of the HBP, day 1		p<0,01; p1>0,1	p>0,05; p ₁ >0,9
4	Fractures of the lower jaw	6	$0{,}59\pm0{,}08$	$0,\!46 \pm 0,\!06$
	14th day		p<0,05; p ₁ >0,05	p>0,7; p ₁ >0,7
5	Fractures of the mandible on the	7	$0,84 \pm 0,09$	$0{,}52\pm0{,}06$
	background of HBP, day 14		p<0,01; p ₂ >0,3	p>0,3; p ₂ >0,3
			p ₃ <0,05	p ₃ >0,3

Notes: p - in comparison with gr. 1; p1 - in comparison with gr. 2; p2 - in comparison with gr. 3; p3 - in comparison with gr. 4

 Table 3. Activity of catalase and index of API in the oral fluid of patients with fractures of the mandible on the background hepato-biliary pathology (HBP)

N⁰	Indicators	n	Catalase, mkat / l	API
1	Control	10	$0,\!40 \pm 0,\!03$	$8,8 \pm 1,0$
2	Fractures of the lower jaw	13	$0,\!30\pm0,\!04$	$5,4 \pm 0,4$
	1st day		p<0,05	p<0,05
3	Fractures of the mandible on the	14	$0,22\pm0,02$	$4,2 \pm 0,4$
	background of the HBP, day 1		p<0,05; p ₁ <0,05	$p < 0,01; p_1 < 0,05$
4	Fractures of the lower jaw	6	$0,34 \pm 0,04$	$6,7 \pm 0,7$
	14th day		p>0,1; p1>0,3	p>0,05; p ₁ >0,05
5	Fractures of the mandible on the	7	$0,\!28\pm0,\!03$	$5,3 \pm 0,6$
	background of HBP, day 14		p<0,05; p ₂ >0,05	p<0,05; p ₂ >0,05
			p ₃ >0,05	p ₃ >0,05

Notes: p, p1, p2, p3 - see. tabl. 2

Table 4 presents the results of determination in the oral fluid of the activity of urease, lysozyme and degree of dysbiosis. Apparently, the fracture increases the activity of urease by 31.5%, but a fracture on the background of HBP increases the urease activity twice.

After treatment of patients with fractures of the mandible, the urease activity is normalized, whereas in patients with fractures on the background of HBP, the urease activity remains significantly high.

The activity of lysozyme significantly (more than 2-fold) decreases in patients, with a fracture in the background of HBP and remains very low, despite on the treatment.

 Table 4. The activity of urease, lysozyme and degree of dysbiosis in the oral fluid of patients

 with fractures of the mandible on the background hepato-biliary pathology

N⁰	Indiastors		Urease,	Lysozyme	Degree
Π/Π	Indicators	n	nkat / l	unit / l	dysbiosis
1	Control	10	54 ± 6	71 ± 6	$1,00 \pm 0,13$
2	Fractures of the lower jaw	13	71 ± 8	75 ± 8	$1,34 \pm 0,21$
	1st day		p>0,05	p>0,5	p>0,05
3	Fractures of the mandible on	14	108 ± 11	34 ± 6	$4,33 \pm 0,38$
	the background of the HBP,		p<0,01;	p<0,05;	p<0,01; p ₁ <0,01
	day 1		$p_1 < 0.05$	p ₁ <0,01	
4	Fractures of the lower jaw	6	59 ± 8	47 ± 6	$1,76 \pm 0,25$
	14th day		p>0,3;	p<0,05;	p<0,05; p1>0,05
			p1>0,3	p1<0,05	
5	Fractures of the mandible on	7	87 ± 9	42 ± 8	$2,82 \pm 0,45$
	the background of the HBP,		p<0,05;	p<0,05;	p<0,05; p ₂ <0,05
	day 14		p ₂ >0,05	p ₂ >0,3	p ₃ <0,05
			p3<0,05	p ₃ >0,3	

Notes: p, p1, p2, p3 - see. tabl. 2

Calculated according to urease and lysozyme index, the degree of oral dysbiosis increases in patients with fractures in the background of HBP in 4,3 times and remains high (2,8 times more) even after treatment.

Thus, the data obtained by us indicate a significant negative impact of the HBP on the oral cavity: the degree of dysbiosis and the intensity of inflammation are increased; however, the level of antioxidant defense is significantly reduced. On this basis, it is considered expedient to use hepatoprotectors, especially with antidisbiosis properties, for the prevention and treatment of fractures of the mandible.

CONCLUSIONS

1. Hepato-biliary pathology causes the development of the oral cavity of dysbiosis and inflammation, which greatly increase in patients with fractures of the mandible.

2. Standard treatment is not sufficiently effective for the treatment of patients with fractures in the background of the HBP.

3. It may be advisable to use additional hepatoprotectors for the prevention and treatment of jaw fractures.

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