TIBBIYOT
TA'LIMI & INNOVATSIYALARI



- 14.00.00 - Tibbiyot fanlari;
- 14.00.01 - Akusherlik va ginekologiya;
- 14.00.02 - Morfologiya;
- 14.00.03 - Endokrinologiya;
- 14.00.04 - Otorinolaringologiya;
- 14.00.05 - Ichki kasalliklar;
- 14.00.06 - Kardiologiya;
- 14.00.07 - Gigiena;
- 14.00.08 - Oftal'mologiya;
- 14.00.09 - Pediatriya;
- 14.00.10 - Yuqumli kasalliklar;
- 14.00.11 - Dermatologiya va venerologiya;
- 14.00.12 - Tibbiy reabilitologiya;
- 14.00.13 - Nevrologiya;
- 14.00.14 - Onkologiya;
- 14.00.15 - Patologik anatomiya;
- 14.00.16 - Normal va patologik fiziologiya;
- 14.00.17 - Farmakologiya va klinik farmakologiya;
- 14.00.18 - Psixiatriya va narkologiya;
- 14.00.19 - Klinik radiologiya;
- 14.00.20 - Tibbiy genetika;
- 14.00.21 - Stomatologiya;
- 14.00.22 - Travmatologiya va ortopediya;
- 14.00.23 - Hamshiralik ishini tashkil etish;
- 14.00.24 - Sud tibbiyoti;
- 14.00.27 - Xirurgiya;
- 14.00.28 - Neyroxirurgiya;
- 14.00.41 - Xalq tabobati;
- 14.00.35 - Bolalar xirurgiyasi;
- 14.00.34 - Yurak-qon tomir xirurgiyasi

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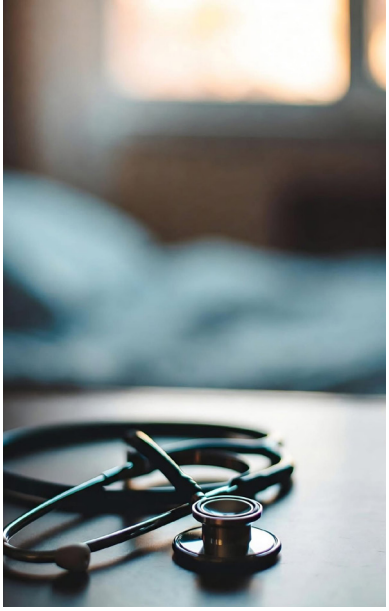
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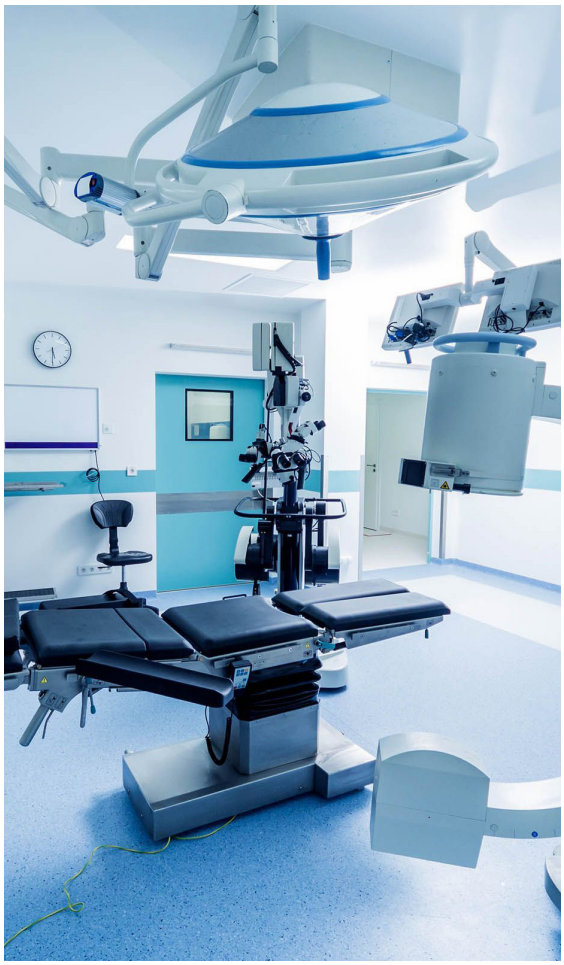
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MECHANISM-DEPENDENT KINETICS OF BONE REMODELING AND INFLAMMATORY MARKERS AFTER COMBINED MAXILLOFACIAL TRAUMA



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Abstract: Severity scores and imaging describe how much of the facial skeleton is broken, but say little about the biological response that governs repair. Whether this response differs with the mechanism of injury has not been examined in combined maxillofacial trauma. To compare the early post-traumatic kinetics of bone-turnover, inflammatory, and mineral-metabolism markers across four injury mechanisms and to relate them to clinical severity. Patients with combined maxillofacial injuries were divided into four groups by mechanism: road traffic accidents (RTA), falls from height, sports injury, and multifactorial trauma. Severity was scored with the Facial Injury Severity Scale (FISS) and the Comprehensive Facial Injury (CFI) score. Ten patients per group underwent serum sampling on days 1, 3, 5, 10, and 14 after injury for C-terminal telopeptide of type I collagen (CTX), tartrate-resistant acid phosphatase 5b (TRAP5b), osteocalcin, alkaline phosphatase (ALP), interleukin-6 (IL-6), C-reactive protein (CRP), parathyroid hormone (PTH), calcium, phosphorus, and vitamin D. CFI was highest after RTA (12.8±4.6) and multifactorial trauma (11.5±4.2) and lowest after sports injury (6.2±2.4; $p<0.05$). Resorption markers peaked on day 5: CTX reached 1.45±0.28 and 1.40±0.26 ng/mL in the multifactorial and RTA groups versus 0.62±0.13 ng/mL after sports injury, and TRAP5b 7.3±1.6 and 7.0±1.5 versus 3.8±0.9 U/L. Osteocalcin and ALP rose continuously to day 14, reaching 34.8±7.2 ng/mL and 205±52 U/L after multifactorial trauma compared with 21.5±4.5 ng/mL and 120±28 U/L after sports injury. IL-6 and CRP peaked on day 3 (IL-6 78±22 vs 28±8 pg/mL; CRP 75±25 vs 18±8 mg/L). Calcium and vitamin D reached their lowest values on day 5, when phosphorus and PTH were highest; these shifts were largest in the high-energy groups, whereas calcium hardly changed after sports injury. Falls occupied an intermediate position for every marker. Repair after combined facial trauma follows a common sequence of inflammation, resorption, and formation, but the amplitude of each phase depends on the injury mechanism and parallels clinical severity. A small serial marker panel may complement anatomical scores when grading severity and timing osteogenesis-supportive treatment.

Key words: maxillofacial trauma; bone turnover markers; fracture healing; interleukin-6; C-reactive protein; osteocalcin; vitamin D; injury mechanism.

Annotatsiya: Og‘irlik darajasi ko‘rsatkichlari va tasviriy tekshiruvlar yuz skeletining qanchalik darajada singanini tavsiflaydi, biroq reparatsiyani belgilovchi biologik javob haqida kam ma’lumot beradi. Ushbu javobning shikastlanish mexanizmiga qarab farq qilishi birgalikdagi yuz-jag‘ sohasi jarohatlarida yetarlicha o‘rganilmagan. Tadqiqotning maqsadi to‘rtta shikastlanish mexanizmi bo‘yicha suyak almashinuvi, yallig‘lanish va mineral almashinuvi markerlarining jarohatdan keyingi dastlabki davrdagi kinetikasini taqqoslash hamda ularni klinik og‘irlik darajasi bilan bog‘lashdan iborat. Birgalikdagi yuz-jag‘ sohasi jarohatlari bo‘lgan bemorlar shikastlanish mexanizmiga ko‘ra to‘rt guruhga ajratildi: yo‘l-transport hodisalari (RTA), balandlikdan yiqilish, sport jarohati va multifaktorial jarohat. Og‘irlik darajasi Facial Injury Severity Scale (FISS) va Comprehensive Facial Injury (CFI) ko‘rsatkichlari yordamida baholandi. Har bir guruhdan 10

nafar bemorda jarohatdan keyingi 1-, 3-, 5-, 10- va 14-kunlarda zardob namunalari olinib, I tip kollagenning C-terminal telopeptidi (CTX), tartratga chidamli kislota fosfataza 5b (TRAP5b), osteokalsin, ishqoriy fosfataza (ALP), interleykin-6 (IL-6), C-reaktiv oqsil (CRP), paratireoid gormon (PTH), kalsiy, fosfor va D vitamini tekshirildi. CFI RTA guruhida (12.8±4.6) va multifaktorial jarohat guruhida (11.5±4.2) eng yuqori, sport jarohati guruhida esa (6.2±2.4; p<0.05) eng past bo‘ldi. Rezorbsiya markerlari 5-kuni eng yuqori qiymatga erishdi: CTX multifaktorial va RTA guruhlarida mos ravishda 1.45±0.28 va 1.40±0.26 ng/mL ga, sport jarohatidan keyin esa 0.62±0.13 ng/mL ga yetdi; TRAP5b esa mos ravishda 7.3±1.6 va 7.0±1.5 ga, sport jarohati guruhida 3.8±0.9 U/L ga yetdi. Osteokalsin va ALP 14-kungacha uzluksiz oshib, multifaktorial jarohatdan keyin mos ravishda 34.8±7.2 ng/mL va 205±52 U/L ga, sport jarohatidan keyin esa 21.5±4.5 ng/mL va 120±28 U/L ga yetdi. IL-6 va CRP 3-kuni eng yuqori qiymatga erishdi (IL-6 78±22 va 28±8 pg/mL; CRP 75±25 va 18±8 mg/L). Kalsiy va D vitamini 5-kuni eng past qiymatga tushdi, ayni vaqtda fosfor va PTH eng yuqori bo‘ldi; ushbu o‘zgarishlar yuqori energiyali guruhlarda eng katta bo‘ldi, sport jarohatidan keyin esa kalsiy deyarli o‘zgarmadi. Yiqilish guruhi barcha markerlar bo‘yicha oraliq holatni egalladi. Yuz-jag‘ sohasi qo‘shma jarohatidan keyingi reparatsiya yallig‘lanish, rezorbsiya va shakllanishning umumiy ketma-ketligi bo‘yicha kechadi, biroq har bir bosqichning amplitudasi shikastlanish mexanizmiga bog‘liq bo‘lib, klinik og‘irlik darajasiga mos keladi. Kichik hajmdagi ketma-ket markerlar paneli og‘irlik darajasini baholash va osteogenezni qo‘llab-quvvatlovchi davolash vaqtini belgilashda anatomik ko‘rsatkichlarni to‘ldirishi mumkin.

Kalit so‘zlar: yuz-jag‘ sohasi qo‘shma jarohati; suyak almashinuvi markerlari; sinish bitishi; interleykin-6; C-reaktiv oqsil; osteokalsin; D vitamini; shikastlanish mexanizmi.

Аннотация: Показатели тяжести и методы визуализации описывают степень повреждения лицевого скелета, однако мало характеризуют биологический ответ, определяющий процессы репарации. Вопрос о различиях этого ответа в зависимости от механизма травмы при сочетанной челюстно-лицевой травме изучен недостаточно. Целью исследования было сравнить кинетику маркеров костного обмена, воспаления и минерального метаболизма в раннем посттравматическом периоде при четырех механизмах травмы и сопоставить их с клинической тяжестью повреждения. Пациенты с сочетанной челюстно-лицевой травмой были разделены на четыре группы в зависимости от механизма повреждения: дорожно-транспортные происшествия (RTA), падение с высоты, спортивная травма и мультифакторная травма. Тяжесть повреждения оценивали с использованием Facial Injury Severity Scale (FISS) и показателя Comprehensive Facial Injury (CFI). У 10 пациентов из каждой группы на 1-, 3-, 5-, 10- и 14-е сутки после травмы проводили забор сыворотки крови для определения С-концевого телопептида коллагена I типа (CTX), тартрат-резистентной кислой фосфатазы 5b (TRAP5b), остеокальцина, щелочной фосфатазы (ALP), интерлейкина-6 (IL-6), С-реактивного белка (CRP), паратиреоидного гормона (PTH), кальция, фосфора и витамина D. CFI был наиболее высоким после RTA (12.8±4.6) и мультифакторной травмы (11.5±4.2) и наиболее низким после спортивной травмы (6.2±2.4; p<0.05). Маркеры резорбции достигали пиковых значений на 5-е сутки: уровень CTX составлял 1.45±0.28 и 1.40±0.26 ng/mL в группах мультифакторной травмы и RTA соответственно против 0.62±0.13 ng/mL после спортивной травмы, а TRAP5b — 7.3±1.6 и 7.0±1.5 против 3.8±0.9 U/L. Уровни остеокальцина и ALP непрерывно повышались до 14-х суток, достигая после мультифакторной травмы 34.8±7.2 ng/mL и 205±52 U/L соответственно, по сравнению с 21.5±4.5 ng/mL и 120±28 U/L после спортивной травмы. IL-6 и CRP достигали пиковых значений на 3-и сутки (IL-6 78±22 против 28±8 pg/mL; CRP 75±25 против 18±8 mg/L). Наиболее низкие значения кальция и витамина D отмечались на 5-е сутки, когда уровни фосфора и PTH были наиболее высокими; эти изменения были наиболее выражены в группах с высокоэнергетической травмой, тогда как после спортивной травмы уровень кальция практически не изменялся. Группа пациентов после падения занимала промежуточное положение по всем маркерам. Репарация после сочетанной челюстно-лицевой травмы протекает по общей последовательности воспаления, резорбции и формирования, однако амплитуда каждой фазы зависит от механизма травмы и соответствует клинической тяжести повреждения. Небольшая панель последовательного определения маркеров может дополнять анатомическую оценку тяжести при определении степени повреждения и сроков проведения остеогенез-поддерживающей терапии.

Ключевые слова: челюстно-лицевая травма; маркеры костного обмена; консолидация переломов; интерлейкин-6; С-реактивный белок; остеокальцин; витамин D; механизм травмы.

INTRODUCTION

Road traffic crashes, falls, sports injuries, and assaults cause most facial fractures, and road traffic injury alone kills about 1.2 million people every year [1,2]. Large clinical series have shown that the cause of injury shapes both the anatomical pattern and the severity of facial fractures [3–6], and that high-energy impacts to the face are frequently accompanied by brain injury and damage to other body regions [7,8]. Assessment of these patients relies on clinical examination, computed tomography, and anatomical severity scores [9]. In earlier reports on the same patient population, we described the epidemiology, fracture distribution, and imaging

trends of combined maxillofacial trauma [10], as well as the regional density, porosity, and finite element stress behaviour of the facial bones [11]. Related work has refined finite element modelling of the craniofacial skeleton [12–15] and automated fracture detection with artificial intelligence [16–19], while studies in Uzbekistan have addressed three-dimensional planning, surgical management, and rehabilitation of facial fractures [20–23]. All of these approaches, however, measure structural damage rather than the body's response to it.

LITERATURE REVIEW

Bone repair passes through overlapping inflammatory, resorptive, and formative phases, and each phase has measurable circulating correlates. IL-6 and CRP reflect the acute-phase reaction; CTX and TRAP5b reflect osteoclastic resorption; osteocalcin and ALP reflect osteoblastic activity; and calcium, phosphorus, PTH, and vitamin D describe the mineral supply on which new bone depends. How these markers behave after facial fractures, and whether their course differs between injury mechanisms, has received little attention. The aim of this study was therefore to compare the kinetics of bone-turnover, inflammatory, and mineral-metabolism markers during the first two weeks after combined maxillofacial trauma of different mechanisms, and to relate the magnitude of these changes to clinical injury severity.

RESEARCH METHODOLOGY

This laboratory study was nested in a retrospective–prospective cohort of patients with combined maxillofacial injuries treated in 2015–2025 at the Republican Research Center of Emergency Medicine and the clinical bases of Tashkent State Medical University, whose clinical and biomechanical characteristics have been reported previously [10,11]. Patients were allocated by injury mechanism to group 1 (RTA, n = 186), group 2 (falls from height, n = 139), group 3 (sports injury, n = 124), or group 4 (multifactorial trauma, n = 117). From each group, 10 patients were enrolled in serial laboratory monitoring. Injury severity was scored on admission with the Facial Injury Severity Scale (FISS) and the Comprehensive Facial Injury (CFI) score; a CFI above 12 was regarded as severe injury.

Venous blood was sampled on days 1, 3, 5, 10, and 14 after injury. Bone resorption was assessed by CTX (ng/mL) and TRAP5b (U/L); bone formation by osteocalcin (ng/mL) and ALP (U/L); the inflammatory response by IL-6 (pg/mL) and CRP (mg/L); and mineral metabolism by total calcium and phosphorus (mmol/L), PTH (pg/mL), and vitamin D (ng/mL). Continuous variables are presented as mean ± standard deviation. Groups were compared with parametric or non-parametric tests according to data distribution, and $p < 0.05$ was considered statistically significant.

ANALYSIS AND RESULTS

Severity differed markedly between mechanisms (Table 1). CFI was highest after RTA and multifactorial trauma and lowest after sports injury ($p < 0.05$); severe injury (CFI > 12) was found in 41.5% of RTA and 46.1% of multifactorial cases but in fewer than 10% of sports injuries. FISS followed the same order, consistent with our earlier analysis of the full cohort [10].

Table 1. Injury severity by mechanism (mean ± SD). A dash indicates that the value was not reported

Group	Mechanism	FISS	CFI	CFI > 12, %
1	Road traffic accident	5.21 ± 2.34	12.8 ± 4.6	41.5
2	Fall from height	3.18 ± 1.87	8.6 ± 3.5	—
3	Sports injury	2.41 ± 1.32	6.2 ± 2.4	< 10
4	Multifactorial trauma	4.36 ± 2.15	11.5 ± 4.2	46.1

CTX rose in every group, peaked on day 5, and then declined (Table 2). The peak was 2.3 times higher after multifactorial trauma (1.45 ± 0.28 ng/mL) than after sports injury (0.62 ± 0.13 ng/mL), and almost as high after RTA (1.40 ± 0.26 ng/mL). TRAP5b showed the same timing, with peak values of 7.3 ± 1.6 U/L (multifactorial), 7.0 ± 1.5 U/L (RTA), 6.0 U/L (falls), and 3.8 ± 0.9 U/L (sports), and a gradual fall afterwards, pointing to an early burst of osteoclastic activity whose size depended on the injury. Osteocalcin and ALP increased steadily throughout follow-up without a plateau (Table 2). On day 14, osteocalcin was 34.8 ± 7.2 ng/mL after multifactorial trauma and 32.5 ± 6.8 ng/mL after RTA, compared with 21.5 ± 4.5 ng/mL after sports injury; ALP was 205 ± 52, 190 ± 50, and 120 ± 28 U/L, respectively. Formation markers thus continued to rise after resorption markers had begun to fall, a shift that became evident between days 5 and 10.

Table 2. Group mean values of bone-turnover markers after injury (n = 10 per group

Marker / group	Day 1	Day 3	Day 5	Day 10	Day 14
CTX, ng/mL — RTA	0.82	1.30	1.40	1.05	0.85
CTX — falls	0.65	1.05	1.15	0.90	0.70
CTX — sports	0.42	0.58	0.62	0.50	0.45
CTX — multifactorial	0.78	1.35	1.45	1.10	0.90
Osteocalcin, ng/mL — RTA	18.5	22.0	24.5	28.6	32.5
Osteocalcin — falls	16.2	19.5	21.5	25.0	28.8
Osteocalcin — sports	14.0	15.5	16.8	19.2	21.5
Osteocalcin — multifactorial	17.8	22.5	25.5	30.2	34.8
ALP, U/L — RTA	145	155	165	180	190
ALP — falls	130	140	150	165	175
ALP — sports	95	100	105	115	120
ALP — multifactorial	155	165	175	190	205

IL-6 peaked on day 3 in all groups, reaching 78 ± 22 pg/mL after multifactorial trauma, 75 ± 20 pg/mL after RTA, 60 pg/mL after falls, and 28 ± 8 pg/mL after sports injury, and returned to 8–20 pg/mL by day 14. CRP followed a similar curve (Table 3), with day-3 values of 75 ± 25 and 70 ± 22 mg/L in the high-energy groups versus 18 ± 8 mg/L after sports injury, a 4.2-fold difference between the extreme groups. Serum calcium fell slightly until day 5 and recovered by days 10–14 in groups 1, 2, and 4, whereas it remained almost constant after sports injury (Table 3). Phosphorus and PTH moved in the opposite direction, peaking on day 5 at 1.60 ± 0.28 mmol/L and 85 ± 22 pg/mL after multifactorial trauma and 1.52 ± 0.25 mmol/L and 80 ± 20 pg/mL after RTA, against 48 ± 12 pg/mL PTH after sports injury. Vitamin D reached its lowest level on day 5 and then recovered; it was lowest after multifactorial trauma (18 ± 4 ng/mL) and RTA (19 ± 5 ng/mL) and highest after sports injury, where it reached 32 ± 8 ng/mL by day 14.

Table 3. Group mean values of inflammatory and mineral-metabolism markers after injury (n = 10 per group)

Marker / group	Day 1	Day 3	Day 5	Day 10	Day 14
CRP, mg/L — RTA	52	70	65	35	18
CRP — falls	38	55	50	28	15
CRP — sports	12	18	16	10	6
CRP — multifactorial	58	75	70	40	20
PTH, pg/mL — RTA	68	75	80	65	58
PTH — falls	60	66	70	58	52
PTH — sports	42	45	48	40	38
PTH — multifactorial	72	80	85	70	62
Calcium, mmol/L — RTA	2.16	2.12	2.10	2.18	2.22
Calcium — falls	2.20	2.18	2.15	2.22	2.25
Calcium — sports	2.28	2.27	2.26	2.30	2.32
Calcium — multifactorial	2.14	2.10	2.08	2.16	2.20
Phosphorus, mmol/L — RTA	1.42	1.48	1.52	1.40	1.32
Phosphorus — falls	1.35	1.40	1.45	1.36	1.28
Phosphorus — sports	1.25	1.28	1.30	1.24	1.20
Phosphorus — multifactorial	1.48	1.55	1.60	1.45	1.35
Vitamin D, ng/mL — RTA	21	20	19	22	24
Vitamin D — falls	24	23	22	25	27
Vitamin D — sports	29	28	28	30	32
Vitamin D — multifactorial	20	19	18	21	23

For every marker, the two high-energy groups (RTA and multifactorial trauma) showed the largest deviations, falls were intermediate, and sports injury showed the smallest; for calcium and vitamin D, the order was reversed. This ranking matched the ranking of the groups by CFI and FISS. The differences between the extreme groups were larger for inflammatory markers (CRP 4.2-fold, IL-6 2.8-fold) than for resorption markers (CTX 2.3-fold, TRAP5b 1.9-fold) and formation markers (ALP 1.7-fold, osteocalcin 1.6-fold). This study shows that the biological response to combined facial trauma has a stable time structure but a variable size. In all four

groups, inflammation peaked first (day 3), osteoclastic resorption next (days 3–5), and osteoblastic formation increased continuously thereafter. What differed between mechanisms was the amplitude of each phase, which was greatest after RTA and multifactorial trauma, the mechanisms that also produced the highest FISS and CFI values. The larger response after high-energy injury is plausible. These mechanisms more often cause multiple fractures across several facial regions and associated injuries elsewhere [3,7,8], so a larger volume of bone and soft tissue must be cleared and rebuilt. Our previous biomechanical analysis showed that impact energy and the regional density and porosity of facial bone determine where and how extensively the skeleton fails [11]; the present data add the biological side of that relationship.

The steep gradient of inflammatory markers between groups suggests that IL-6 and CRP, which are inexpensive and widely available, reflect the overall trauma load best and may help to identify patients who need closer monitoring. Resorption markers peaked on day 5, and formation markers rose mainly after this point; this timing may be relevant when osteogenesis-supportive treatment is planned. A protocol of comprehensive osteogenesis stimulation, together with diagnostic and rehabilitation algorithms for maxillofacial injuries, was developed on the basis of this work and approved for clinical use [24–26]; its introduction shortened the mean hospital stay from 14 to 10 days and reduced treatment costs per patient by 28.6%. The lower vitamin D levels after high-energy trauma deserve attention. Vitamin D is required for calcium absorption and mineralisation, and its fall at the time of maximum resorption, together with the transient drop in calcium and rise in PTH, suggests increased demand for mineral during early repair. Whether pre-existing deficiency or post-traumatic consumption drives this pattern cannot be decided from our data, but checking vitamin D in patients with severe facial trauma seems reasonable. The study has limitations. Laboratory monitoring was performed in 10 patients per group, pre-injury values were unavailable, there was no uninjured control group, and follow-up ended on day 14, before remodelling is complete. The association with severity was examined at the group level; individual-level correlations and the predictive value of the markers for complications should be tested in larger prospective studies.

CONCLUSION

After combined maxillofacial trauma, circulating markers follow a consistent sequence: the inflammatory response peaks on day 3, bone resorption on days 3–5, and bone formation increases steadily to day 14. The amplitude of these changes depends on the injury mechanism and is greatest after road traffic accidents and multifactorial trauma, intermediate after falls, and smallest after sports injury, in line with clinical severity scores. A panel of IL-6, CRP, CTX, osteocalcin, and vitamin D measured in the first two weeks may complement anatomical severity scoring and support the timing of osteogenesis-supportive treatment. Funding. This research received no external funding. Conflict of Interest. The author declares no conflict of interest. Ethics Statement. The study was conducted in accordance with the Declaration of Helsinki. Data Availability. The data supporting the findings of this study are available from the author upon reasonable request.

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