TIBBIYOT
TA'LIMI & INNOVATSIYALARI

O'ZBEKISTON RESPUBLIKASI
19 20
TOSHKENT TIBBIYOT AKADEMIYASI

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- 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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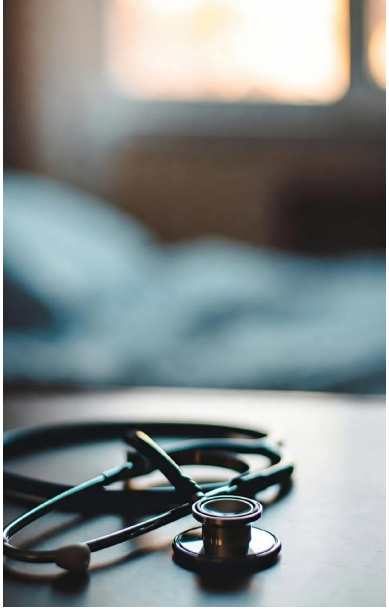
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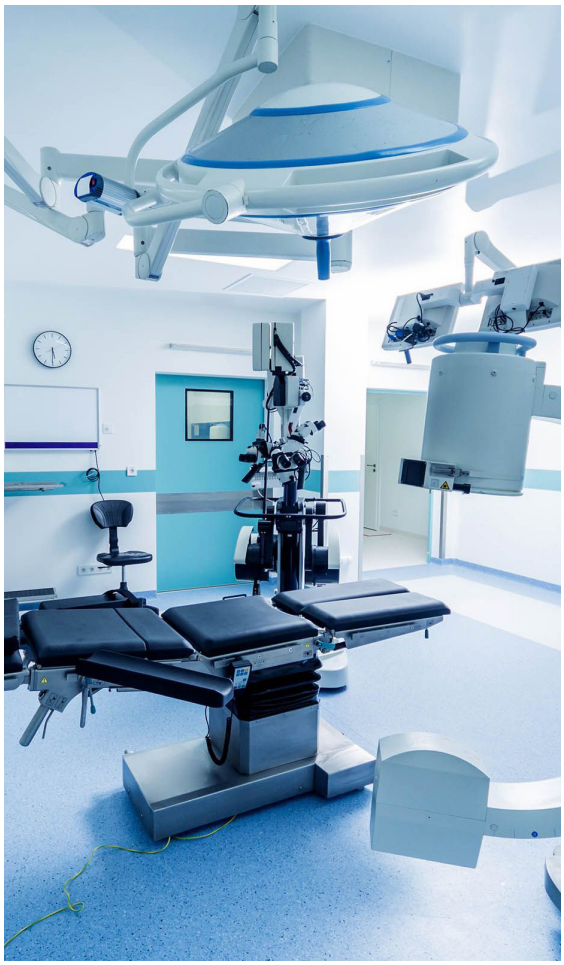
Litsenziya raqami: №745444













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MUNDARIJA

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THE COL1A1 1997C/A (RS1107946) GENE POLYMORPHISM IN OSTEOPOROSIS ASSOCIATED WITH CARDIORENAL SYNDROME

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Abstract: Background. Bone strength depends on the organic matrix as well as on mineral content, and type I collagen is its principal protein. The COL1A1 gene encodes the alpha-1 chain of this collagen, and promoter variants alter transcription and the ratio of alpha-1 to alpha-2 chains, thereby affecting bone quality independently of bone mineral density. Aim. In chronic kidney disease (CKD), the bone matrix is already compromised by mineral dysregulation and secondary hyperparathyroidism, while concomitant chronic heart failure (CHF) adds neurohumoral, inflammatory and nutritional burdens. Whether a matrix-related genetic variant becomes phenotypically apparent in this setting has not yet been examined. Materials and Methods. The study enrolled 196 subjects divided into three groups: patients with CHF, CKD and osteoporosis (n = 52); patients with CKD and osteoporosis without clinically significant CHF (n = 48); and healthy volunteers comprising the control group (n = 96). The study assessed calcium-phosphorus metabolism, intact parathyroid hormone, 25-hydroxyvitamin D, serum creatinine, estimated glomerular filtration rate and N-terminal pro-B-type natriuretic peptide. The COL1A1 1997C/A polymorphism (rs1107946) was genotyped by polymerase chain reaction with restriction fragment length polymorphism analysis. Odds ratios and their 95% confidence intervals were calculated using the Woolf method, and statistical significance was assessed using the two-sided Fisher exact test. Results. Genotype distributions conformed to Hardy–Weinberg equilibrium in all groups. The minor A allele was significantly more frequent in both patient groups than in the control group, and the association demonstrated a graded pattern: it was stronger in the group with CHF than in the group without CHF, with a statistically significant difference between the two patient groups. Moreover, heterozygous C/A genotype carriage was overrepresented to almost the same extent in both patient groups, whereas A/A homozygosity was overrepresented only in the group with CHF. In the absence of CHF, the frequency of the A/A genotype did not differ significantly from that in the control group. Conclusion. The A allele of the COL1A1 1997C/A polymorphism acts as a risk marker for osteoporosis in CKD, irrespective of cardiac status, whereas its homozygous form is specifically associated with the cardiorenal phenotype. Consequently, genotype provides an additional, non-fluctuating layer of information alongside mineral indices already used to predict fracture risk in CKD. These findings warrant replication in larger regional cohorts.

Key words: chronic kidney disease; osteoporosis; COL1A1 gene; rs1107946; chronic heart failure; cardiorenal syndrome.

Annotatsiya: Tadqiqotning dolzarbligi. Suyak mustahkamligi mineral moddalar miqdori bilan bir qatorda organik matritsaga ham bog‘liq bo‘lib, uning asosiy oqsili I turdagi kollagen hisoblanadi. COL1A1 geni ushbu kollagenning alfa-1 zanjirini kodlaydi. Gen promotoridagi variantlar transkripsiya jarayonini hamda alfa-1 va alfa-2 zanjirlari nisbatini o‘zgartirib, suyak mineral zichligidan mustaqil ravishda uning sifatiga ta’sir ko‘rsatadi. Tadqiqot maqsadi. Surunkali buyrak kasalligida (SBK) mineral almashinuvining buzilishi va ikkilamchi giperparatireoz tufayli suyak matritsasi allaqachon zararlangan bo‘ladi. Hamroh surunkali yurak yetishmovchiligi (SYY) esa qo‘shimcha neyrogumoral, yallig‘lanish va nutritiv yuklamalarni keltirib chiqaradi. Bunday sharoitda suyak matritsasi bilan bog‘liq genetik variantning fenotipik namoyon bo‘lishi hozirgacha o‘rganilmagan. Materiallar va usullar. Tadqiqotga uch guruhga ajratilgan 196 nafar ishtirokchi jalb qilindi: SYY, SBK va

osteoporoz mavjud bemorlar (n = 52); klinik jihatdan ahamiyatli SYY kuzatilmagan, SBK va osteoporoz mavjud bemorlar (n = 48); hamda nazorat guruhini tashkil etgan sog‘lom ko‘ngillilar (n = 96). Kalsiy-fosfor almashinuvi ko‘rsatkichlari, intakt paratgormon, 25-gidroksivitamin D, qon zardobidagi kreatinin, hisoblangan glomerulyar filtratsiya tezligi va B turdagi natriyuretik peptidning N-terminal propeptidi baholandi. COL1A1 genining 1997C/A polimorfizmi (rs1107946) polimeraza zanjirli reaksiyasi va restriksion fragmentlar uzunligi polimorfizmi tahlili yordamida genotiplandi. Shanslar nisbati va uning 95 % ishonch oralig‘i Vulf usuli bilan hisoblandi, statistik ahamiyatlilik esa Fisherning ikki tomonlama aniq testi yordamida baholandi. Natijalar. Barcha guruhlarda genotiplar taqsimoti Hardi–Vaynberg muvozanatiga mos keldi. Minor A alleli bemorlarning har ikki guruhida nazorat guruhiga nisbatan statistik jihatdan ahamiyatli darajada ko‘proq uchradi. Assotsiatsiya darajali xususiyatga ega bo‘lib, SYY mavjud guruhda SYY kuzatilmagan guruhga qaraganda kuchliroq namoyon bo‘ldi. Bemorlarning ushbu ikki guruhi o‘rtasidagi farq ham statistik jihatdan ahamiyatli edi. Bundan tashqari, C/A geterozigota genotipining ortiqcha uchrash darajasi bemorlarning har ikki guruhida deyarli bir xil bo‘ldi. A/A gomozigota genotipining ortiqcha uchrashi esa faqat SYY mavjud guruhda kuzatildi. SYY kuzatilmagan guruhda A/A genotipining chastotasi nazorat guruhidagidan statistik jihatdan ahamiyatli farq qilmadi. Xulosa. COL1A1 genining 1997C/A polimorfizmidagi A alleli, yurak holatidan qat’i nazar, SBKda osteoporoz xavfining markeri sifatida namoyon bo‘ladi, uning gomozigota shakli esa aynan kardiorenal fenotip bilan bog‘liq. Shu sababli genotip SBKda sinish xavfini bashorat qilishda allaqachon qo‘llaniladigan mineral almashinuvi ko‘rsatkichlarini vaqt davomida o‘zgarmaydigan qo‘shimcha axborot bilan to‘ldiradi. Olingan natijalarni yirikroq hududiy kogortalarda takroriy tekshirish zarur.

Kalit so‘zlar: surunkali buyrak kasalligi; osteoporoz; COL1A1 geni; rs1107946; surunkali yurak yetishmovchiligi; kardiorenal sindrom.

Аннотация: Актуальность. Прочность кости зависит как от содержания минеральных веществ, так и от органического матрикса, основным белком которого является коллаген I типа. Ген COL1A1 кодирует альфа-1-цепь этого коллагена, а варианты его промотора изменяют транскрипцию и соотношение альфа-1- и альфа-2-цепей, влияя на качество кости независимо от её минеральной плотности. Цель исследования. При хронической болезни почек (ХБП) костный матрикс уже повреждён вследствие нарушений минерального обмена и вторичного гиперпаратиреоза, а сопутствующая хроническая сердечная недостаточность (ХСН) создаёт дополнительную нейрогуморальную, воспалительную и нутритивную нагрузку. Вопрос о том, проявляется ли в этих условиях фенотипический эффект генетического варианта, связанного с костным матриксом, ранее не изучался. Материалы и методы. В исследование включены 196 участников, распределённых на три группы: пациенты с ХСН, ХБП и остеопорозом (n = 52); пациенты с ХБП и остеопорозом без клинически значимой ХСН (n = 48); и здоровые добровольцы, составившие контрольную группу (n = 96). Оценивали показатели кальций-фосфорного обмена, интактный паратгормон, 25-гидроксивитамин D, креатинин сыворотки крови, расчётную скорость клубочковой фильтрации и N-концевой фрагмент предшественника натрийуретического пептида B-типа. Генотипирование полиморфизма 1997C/A гена COL1A1 (rs1107946) проводили методом полимеразной цепной реакции с анализом полиморфизма длин рестрикционных фрагментов. Отношения шансов и их 95 % доверительные интервалы рассчитывали методом Вулфа, статистическую значимость оценивали с помощью двустороннего точного критерия Фишера. Результаты. Распределение генотипов во всех группах соответствовало равновесию Харди–Вайнберга. Минорный аллель A встречался статистически значимо чаще в обеих группах пациентов по сравнению с контрольной группой. Выраженность ассоциации имела градиентный характер: она была выше в группе с ХСН, чем в группе без неё; различия между двумя группами пациентов также были статистически значимыми. При этом повышенная частота гетерозиготного генотипа C/A была практически одинаковой в обеих группах пациентов, тогда как повышенная частота гомозиготного генотипа A/A отмечалась только в группе с ХСН. В группе без ХСН частота генотипа A/A статистически значимо не отличалась от показателя контрольной группы. Заключение. Аллель A полиморфизма 1997C/A гена COL1A1 выступает маркером риска остеопороза при ХБП независимо от состояния сердца, тогда как его гомозиготная форма ассоциирована именно с кардиоренальным фенотипом. Таким образом, генотип дополняет показатели минерального обмена, уже используемые для прогнозирования риска переломов при ХБП, генетической характеристикой, не подверженной временным колебаниям. Полученные результаты требуют подтверждения в более крупных региональных когортах.

Ключевые слова: хроническая болезнь почек; остеопороз; ген COL1A1; rs1107946; хроническая сердечная недостаточность; кардиоренальный синдром.

INTRODUCTION

Osteoporosis in patients with chronic kidney disease (CKD) presents a complex clinical problem because skeletal fragility may reflect not only reduced bone mineral density but also abnormalities in bone turnover, mineralization, and matrix properties. CKD–mineral and bone disorder encompasses disturbances in calcium, phosphate, parathyroid hormone, and vitamin D metabolism, together with skeletal and extraskeletal

manifestations. Consequently, the assessment of skeletal health in patients with CKD requires consideration of biochemical abnormalities alongside bone mineral density and the broader clinical context. The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines recommend bone mineral density testing in patients with CKD and evidence of mineral and bone disorder or risk factors for osteoporosis when the results are expected to influence treatment decisions. The coexistence of chronic heart failure (CHF) adds another dimension to skeletal vulnerability. A community-based investigation demonstrated an association between heart failure and osteoporotic fractures, supporting the importance of assessing bone health in this population. However, shared characteristics, including advanced age, comorbidities, and functional impairment, complicate the interpretation of this relationship. In patients with concurrent cardiac and renal disease, investigating inherited determinants of skeletal integrity may help characterize individual susceptibility beyond routinely measured biochemical indices.

The COL1A1 gene represents a biologically plausible candidate because it encodes the alpha-1 chain of type I collagen, a principal structural component of the organic bone matrix. Regulatory variation within this gene may influence collagen expression and thereby contribute to differences in skeletal properties. The promoter polymorphism rs1107946, commonly designated −1997G/T and represented as C/A on the complementary strand, has been investigated in relation to bone mineral density. Its initial characterization demonstrated an association with bone mass, while subsequent functional studies examined the transcriptional activity of different promoter alleles and haplotypes. These findings provide a rationale for investigating this variant in clinically defined populations with skeletal disease. Nevertheless, biological plausibility alone does not establish that rs1107946 independently determines osteoporosis or fracture risk in patients with cardiorenal disease. In particular, the functional consequences of other COL1A1 variants should not automatically be extrapolated to this promoter polymorphism. Therefore, the present investigation addresses a specific question: whether the distribution of rs1107946 alleles and genotypes differs between patients with CKD-associated osteoporosis, with and without concomitant CHF, and healthy controls.

LITERATURE REVIEW

Bone disease in patients with chronic kidney disease (CKD) is no longer regarded as merely a secondary complication. Phosphate retention, declining calcitriol synthesis, increasing fibroblast growth factor 23 levels and decreasing Klotho expression occur long before the end stage of CKD and collectively contribute to secondary hyperparathyroidism. These processes drive systemic disturbances in mineral metabolism, bone turnover and extraskeletal calcification, collectively referred to as CKD–mineral and bone disorder (CKD-MBD) [11, 16, 17]. A recent international consensus proposes CKD-associated osteoporosis as a distinct clinical entity and emphasises that bone strength depends on bone quality as much as on bone quantity [5, 8]. Consistent with this concept, disturbances in mineral metabolism markers, particularly parathyroid hormone and phosphate, independently predict fracture risk in patients with CKD [2, 12]. Chronic heart failure (CHF) superimposes an additional set of pathophysiological mechanisms on this background. Activation of the renin–angiotensin–aldosterone system increases urinary calcium and magnesium losses and contributes to the persistence of secondary hyperparathyroidism. Low-grade inflammation shifts the RANK/RANKL/osteoprotegerin balance towards increased osteoclastic resorption. Limited sun exposure, impaired hepatic 25-hydroxylation and intestinal oedema further exacerbate vitamin D deficiency. Loop diuretic use, immobilisation, sarcopenia and low body weight contribute to an unfavourable skeletal environment [7, 15, 21]. Bone disease is common in patients with heart failure with reduced ejection fraction and has independent prognostic significance [20, 22]. When renal and cardiac dysfunction coexist, this combination is classified within the chronic forms of cardiorenal syndrome [19].

Bone strength, however, is not determined by mineral content alone. Type I collagen is the principal protein of the organic bone matrix, and the COL1A1 gene encodes its alpha-1 chain. Regulatory variants of this gene may alter transcription and the ratio of alpha-1 to alpha-2 chains, potentially resulting in a weaker organic matrix at a given bone mineral density. Thus, their effects may be mediated through bone quality rather than bone quantity [13]. The 1997C/A substitution (rs1107946; reported on the opposite DNA strand as −1997G/T) is located in the 5′ flanking region of the gene. However, evidence regarding its contribution in general and postmenopausal populations remains inconsistent. Two meta-analyses reported, at most, marginal associations with bone mineral density and no significant association with fracture risk [6, 24, 25]. Furthermore, a recent European study found no association under any of the genetic models examined [1, 9]. These negative findings were obtained in populations without the specific combination of skeletal matrix disturbances associated with CKD and cardiorenal disease. In CKD, the bone matrix is already compromised by mineral dysregulation, while the coexistence of cardiac dysfunction introduces additional neurohumoral, inflammatory and nutritional burdens. Consequently, a genetic variant affecting matrix quality might become phenotypically apparent under

these conditions, even if its effects are not detectable in other populations. This possibility has not yet been investigated. Furthermore, reference allele frequencies for Central Asian populations remain scarce, making the direct extrapolation of published estimates to this region potentially unreliable [23]. Design and ethics. A single-centre, cross-sectional comparative study was conducted over a two-year period at the clinical site of the Tashkent Specialised Somatic Hospital. The study protocol was approved by the local ethics committee and complied with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before any study-related procedures were performed.

Participants and groups. A total of 196 subjects were examined and allocated to three groups. Group 1 comprised 52 patients with chronic kidney disease, concomitant chronic heart failure and confirmed osteoporosis. Group 2 comprised 48 patients with chronic kidney disease and osteoporosis without clinically significant heart failure. The control group comprised 96 healthy volunteers with a comparable age and sex distribution and no evidence of renal or cardiac disease or osteoporosis. Diagnostic criteria. Chronic kidney disease was diagnosed according to KDIGO criteria, based on a glomerular filtration rate below 60 mL/min/1.73 m² persisting for at least three months and/or persistent albuminuria [11]. Chronic heart failure was diagnosed based on the presence of characteristic symptoms and signs, structural or functional cardiac abnormalities detected by echocardiography, and elevated levels of N-terminal pro-B-type natriuretic peptide [14]. Osteoporosis was diagnosed according to accepted international criteria, based on a T-score of −2.5 standard deviations or below at the lumbar spine and/or femoral neck. In patients younger than 50 years, a Z-score of −2.0 or below was used for the assessment of bone mineral density [10]. Exclusion criteria. Patients receiving renal replacement therapy or with a history of kidney transplantation were excluded. Additional exclusion criteria included primary hyperparathyroidism, thyrotoxicosis, systemic glucocorticoid therapy lasting more than three months, malignancy, malabsorption, chronic liver disease, inflammatory rheumatic disease, pregnancy, previous antiresorptive treatment, and an episode of acute cardiac decompensation within four weeks before enrolment.

Clinical and laboratory assessment. Clinical evaluation included assessment of the New York Heart Association functional class, the six-minute walk test, echocardiography with measurement of left ventricular ejection fraction using the Simpson method, anthropometric measurements with calculation of body mass index, and a structured medical history focusing on previous low-energy fractures and falls. Venous blood samples were collected in the morning after an overnight fast to determine total and albumin-corrected calcium, inorganic phosphate, total alkaline phosphatase, intact parathyroid hormone, 25-hydroxyvitamin D, serum creatinine, N-terminal pro-B-type natriuretic peptide, haemoglobin and C-reactive protein levels. The estimated glomerular filtration rate was calculated using the CKD-EPI equation [4]. Genotyping. Genomic DNA was extracted from EDTA-anticoagulated peripheral blood leukocytes using a silica-column adsorption method. The COL1A1 1997C/A polymorphism (rs1107946) was genotyped by polymerase chain reaction followed by restriction fragment length polymorphism analysis (PCR-RFLP). The resulting products were separated by electrophoresis on a 2–3% agarose gel and visualised by ethidium bromide staining. Ten per cent of randomly selected samples were re-genotyped blindly as an internal quality-control measure.

RESEARCH METHODOLOGY

CKD was diagnosed and classified according to [diagnostic criteria and guideline version], with documentation of the duration of kidney abnormalities, estimated glomerular filtration rate (eGFR), CKD stage, and dialysis status, where applicable. CHF was established using clinical and instrumental diagnostic criteria. The definition of “without clinically significant CHF” was specified. Osteoporosis was diagnosed using the actual diagnostic method and criteria. Where dual-energy X-ray absorptiometry was performed, the skeletal sites examined, device manufacturer, reference population, and applicable T-score or Z-score criteria were specified. The operational definition of the cardiorenal phenotype was the coexistence of CHF and CKD in Group 1; classification into a particular cardiorenal syndrome subtype requires additional clinical documentation. The biochemical evaluation included serum calcium and phosphorus, intact parathyroid hormone, 25-hydroxyvitamin D, serum creatinine, and N-terminal pro-B-type natriuretic peptide. Renal function was assessed by calculating eGFR from serum creatinine using the specified equation and version. Blood collection conditions, sample processing and storage procedures, assay platforms, measurement units, and laboratory reference intervals were specified. The calcium measurement was identified as total, albumin-adjusted, or ionized calcium, according to the procedure actually used. The COL1A1 1997C/A polymorphism (rs1107946) was genotyped by polymerase chain reaction followed by restriction fragment length polymorphism analysis (PCR-RFLP). DNA was obtained from biological material using the specified extraction method or kit.

The target region was amplified using the forward and reverse primer sequences [insert primer sequences] under the specified PCR conditions. Amplification products were digested with the specified restriction enzyme, and restriction fragments were separated using the specified electrophoresis conditions. Genotypes were

assigned as C/C, C/A, or A/A according to the expected fragment patterns [insert fragment sizes]. Allele nomenclature was checked against the assay's strand orientation. Genotyping quality-control procedures, including repeat testing, controls, and the genotype call rate, were reported only where documented. Statistical analysis. Analysis was performed using IBM SPSS Statistics. Quantitative variables are presented as arithmetic means and standard deviations and were compared between groups using Welch's t-test for unequal variances; qualitative variables were compared using the two-sided Fisher's exact test. Conformity of genotype distributions to Hardy–Weinberg equilibrium was tested using the chi-square test with one degree of freedom, and the distribution of the three genotypes between groups was assessed using the Pearson chi-square test with two degrees of freedom. Pairwise comparisons of alleles and genotypes in 2×2 tables were performed using the two-sided Fisher's exact test, applied to all such comparisons regardless of expected frequencies. The strength of association is expressed as the odds ratio with a 95% confidence interval calculated using the Woolf method. Each genotype was compared with the two remaining genotypes combined, so that the C/C and A/A contrasts correspond to the dominant and recessive models for allele A, respectively. A two-sided p-value below 0.05 was considered statistically significant.

ANALYSIS AND RESULTS

Clinical and laboratory characteristics. The examined groups are characterised in Table 1. The two patient groups did not differ from each other in age, sex, or body mass index ($p > 0.05$ for each), so that the presence of heart failure was the systematic difference between them. Group 1 corresponded to New York Heart Association functional class II–III with mildly reduced left ventricular ejection fraction, whereas Group 2 had no clinically significant heart failure. Relative to the control group, both patient groups were somewhat older, while body mass index and sex distribution were comparable across all three groups.

Table 1. Clinical and laboratory characteristics of the examined groups

Parameter	Group 1 CHF+CKD+OP n = 52	Group 2 CKD+OP n = 48	Control n = 96	p		
				1 vs 2	1 vs C	2 vs C
Age, years	60 ± 9	58 ± 8	55 ± 7	0.242	< 0.001	0.030
Female sex, n (%)	36 (69.2)	32 (66.7)	62 (64.6)*	0.832	0.591	0.854
Body mass index, kg/m²	26.1 ± 4.2	26.8 ± 4.0	27.2 ± 3.8	0.395	0.119	0.567
NYHA functional class	II–III	—	—	—	—	—
Left ventricular ejection fraction, %	46 ± 8	—	—	—	—	—
Total calcium, mmol/L	2.12 ± 0.14	2.20 ± 0.12	2.32 ± 0.09	0.003	< 0.001	< 0.001
Inorganic phosphate, mmol/L	1.42 ± 0.28	1.25 ± 0.24	1.08 ± 0.18	0.002	< 0.001	< 0.001
Alkaline phosphatase, U/L	118 ± 36	103 ± 30	82 ± 22	0.025	< 0.001	< 0.001
Intact parathyroid hormone, pg/mL	145 ± 68	112 ± 52	48 ± 18	0.007	< 0.001	< 0.001
25-hydroxyvitamin D, ng/mL	15 ± 7	19 ± 8	27 ± 9	0.009	< 0.001	< 0.001
Creatinine, μmol/L	190 ± 65	165 ± 55	82 ± 16	0.040	< 0.001	< 0.001
eGFR (CKD-EPI), mL/min/1.73 m²	35 ± 12	42 ± 11	88 ± 12	0.003	< 0.001	< 0.001
NT-proBNP, pg/mL	2 800 ± 1 900	650 ± 400	90 ± 45	< 0.001	< 0.001	< 0.001

*Note: CHF — chronic heart failure; CKD — chronic kidney disease; OP — osteoporosis; NYHA — New York Heart Association; eGFR — estimated glomerular filtration rate; NT-proBNP — N-terminal pro-B-type natriuretic peptide; C — control group. Quantitative variables were compared by Welch's t-test and qualitative variables by the two-sided Fisher exact test. NYHA class and ejection fraction apply to Group 1 only. * the sex distribution of the control group requires verification against the primary data.*

Mineral indices. Disturbance of calcium–phosphorus metabolism increased from Group 2 to Group 1: total calcium and 25-hydroxyvitamin D fell while inorganic phosphate, alkaline phosphatase and intact parathyroid hormone rose. The addition of heart failure was therefore accompanied not by a qualitatively different mineral pattern but by a more pronounced form of the same one. Estimated glomerular filtration rate was lower in Group 1, and NT-proBNP differed between the groups by more than an order of magnitude, reflecting the

criterion by which they were separated. Genotype distribution. Genotype distributions of the COL1A1 1997C/A polymorphism conformed to Hardy–Weinberg equilibrium in every group: $\chi^2 = 1.64$ ($p = 0.201$) in Group 1, $\chi^2 = 1.54$ ($p = 0.214$) in Group 2 and $\chi^2 = 0.34$ ($p = 0.557$) in controls, which indicates representative sampling and the absence of gross selection or genotyping error. Allele and genotype frequencies are given in Table 2.

Table 2. Allele and genotype distribution of the COL1A1 1997C/A (rs1107946) polymorphism, n (%)

Group	Minor allele A	C/C	C/A	A/A
Group 1, n = 52	45 (43.3)	19 (36.5)	21 (40.4)	12 (23.1)
Group 2, n = 48	22 (22.9)	27 (56.2)	20 (41.7)	1 (2.1)
Control, n = 96	15 (7.8)	82 (85.4)	13 (13.5)	1 (1.0)

Note: allele frequencies are calculated per 2n chromosomes and genotype frequencies per n subjects. The major allele is C and the minor allele is A; the number of major alleles equals 2n minus the number of minor alleles and is therefore not tabulated. The pooled patient group (n = 100) referred to in Table 3 is the sum of Groups 1 and 2.

Association with the studied phenotypes. The minor allele was enriched in both patient groups, and the distribution of the three genotypes differed significantly from controls for the pooled patients ($\chi^2 = 34.86$; df = 2; $p < 0.001$), for Group 1 ($\chi^2 = 41.03$; df = 2; $p < 0.001$) and for Group 2 ($\chi^2 = 14.89$; df = 2; $p < 0.001$). The corresponding effect estimates are given in Table 3.

Table 3. Association of the COL1A1 1997C/A (rs1107946) polymorphism with the studied phenotypes (reference group — controls)

Allele / genotype	All patients vs control		Group 1 vs control		Group 2 vs control	
	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
Allele A	5.94 (3.25–10.87)	< 0.001	9.00 (4.68–17.32)	< 0.001	3.51 (1.72–7.14)	< 0.001
C/C vs C/A+A/A	0.15 (0.07–0.29)	< 0.001	0.10 (0.04–0.22)	< 0.001	0.22 (0.10–0.49)	< 0.001
C/A vs C/C+A/A	4.44 (2.19–9.00)	< 0.001	4.33 (1.93–9.68)	< 0.001	4.56 (2.01–10.35)	< 0.001
A/A vs C/C+C/A	14.20 (1.82–110.79)	0.001	28.50 (3.58–226.58)	< 0.001	2.02 (0.12–33.03)	1.000

Note: OR — odds ratio; CI — confidence interval. Odds ratios were calculated by the Woolf method and p values by the two-sided Fisher exact test. Each genotype is compared with the two remaining genotypes combined.

Direct comparison of the patient groups showed that the association was graded rather than uniform. Allele A was significantly more frequent in Group 1 than in Group 2 (OR 2.57; 95% CI 1.39–4.74; $p = 0.003$), and the distribution of the three genotypes differed between them ($\chi^2 = 10.58$; df = 2; $p = 0.005$). The difference was attributable entirely to the homozygotes: A/A was far more frequent in Group 1 (OR 14.10; 95% CI 1.76–113.22; $p = 0.002$), whereas heterozygous C/A carriage was almost identical in the two groups (OR 0.95; 95% CI 0.43–2.11; $p = 1.000$). Since osteoporosis was confirmed in both groups and renal impairment was comparable, the only systematic difference between them was the presence of heart failure. Two findings emerge. First, the minor A allele of COL1A1 1997C/A was associated with osteoporosis in chronic kidney disease, irrespective of cardiac status: both patient groups differed significantly from controls, and heterozygous carriage was over-represented to almost exactly the same degree in each. Second, the association was nevertheless graded, and the gradient was produced by homozygotes alone — A/A was strongly associated with the cardiorenal phenotype and was indistinguishable from controls in its absence. Allele A therefore behaves as a risk marker for osteoporosis in CKD generally, while its homozygous form marks the subgroup in which heart failure is also present.

This pattern is consistent with the known biology of the locus. Regulatory variants in the 5′ flank of COL1A1 alter transcription and the ratio of alpha-1 to alpha-2 collagen chains, and bone from carriers shows lower yield strength at comparable apparent density, which places the effect on matrix quality rather than on mineral content [13, 18]. In chronic kidney disease, the matrix is already degraded by mineral dysregulation and secondary hyperparathyroidism [8, 16], so a constitutional weakness of the collagen scaffold is superimposed on an abnormal substrate. A dose-dependent model accounts for the gradient: a single copy of the allele suffices to reach the osteoporotic phenotype once CKD-related matrix injury is present, whereas two copies cluster in the subgroup in which the additional neurohumoral, inflammatory, and nutritional burden of heart failure is

also imposed [21]. The result stands in apparent contrast to the published literature, in which this variant has performed poorly. Two meta-analyses found, at most, marginal associations with bone mineral density and no association with fracture [6, 24], and a recent study in postmenopausal women reported no association under any genetic model [1, 9]. Those studies, however, were conducted in populations whose bone matrix was not otherwise injured. The present data suggest that the variant is not unconditionally weak but context-dependent, becoming visible only where matrix quality is already compromised — a gene–environment interaction rather than a contradiction. Two observations temper this interpretation. The effect sizes reported here greatly exceed anything described for this locus elsewhere, and the minor allele frequency in our controls is lower than that reported in European series. Therefore, comparison with published estimates is not straightforward, and regional reference data are required before the figures are used clinically.

Mineral indices already carry prognostic information in this setting: parathyroid hormone and phosphate predict fracture in chronic kidney disease, although both show substantial heterogeneity between studies [12], and they fluctuate with hydration, diuretic dose, and acute decompensation. A genotype determined once does not fluctuate and would add a stable layer to a risk assessment otherwise based on variable biochemistry. Given that bone disease in heart failure carries independent prognostic weight [22] and that CKD-associated osteoporosis is now treated as an entity in its own right [8], identifying A/A homozygotes among cardiorenal patients may help determine in whom correction of vitamin D deficiency, control of secondary hyperparathyroidism, and fall prevention should begin earliest. The cross-sectional design precludes any statement about the causal sequence or the rate of bone loss. The patient groups are small and drawn from a single centre; the confidence intervals for the A/A contrasts are correspondingly wide, with the estimates based on twelve and one homozygote, respectively, and must be regarded as provisional until replicated. Specific bone turnover markers were not measured, so the relative contributions of impaired formation and increased resorption remain undetermined [3]. Finally, no group of patients with heart failure and kidney disease but without osteoporosis was available, which prevents the separation of a true gene–environment interaction from mediation through the cardiac phenotype itself. Assembling such a group is the next stage of this work.

CONCLUSION

1. The genotype distribution of the COL1A1 1997C/A (rs1107946) polymorphism conformed to Hardy–Weinberg equilibrium in all three groups, which allows the control frequencies to serve as local reference values for the population examined. 2. The minor A allele was significantly associated with osteoporosis in chronic kidney disease in both patient groups, that is, irrespective of the presence of chronic heart failure, and heterozygous C/A carriage was over-represented to almost the same degree in each group. 3. The association was graded, and the gradient was produced by the homozygotes: A/A was strongly associated with the combined cardiorenal phenotype and did not differ from controls among patients without heart failure. 4. Allele A of COL1A1 1997C/A may therefore be regarded as a matrix-related risk marker of osteoporosis in chronic kidney disease, while its homozygous form identifies the cardiorenal subgroup. Given the wide confidence intervals and the single-centre design, these estimates require replication in larger regional cohorts.

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