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Original Research Article

Molecular Analysis of Genetic Variants and Expression of the Ferroportin (SLC40A1) Gene in Hemodialysis Patients in Mosul City

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Abstract: ***Background:*** Ferroportin (FPN1), encoded by the SLC40A1 gene, is the only known cellular iron exporter and is essential for maintaining systemic iron homeostasis. In patients with end-stage renal disease (ESRD) undergoing hemodialysis, chronic inflammation and persistently elevated Hepcidin levels contribute to ferroportin degradation, leading to impaired iron export and iron-restricted erythropoiesis. However, data regarding SLC40A1 gene expression and genetic variation in this patient population remain limited. Objectives: This study aimed to evaluate the molecular characteristics of the SLC40A1 gene in hemodialysis patients from Mosul City by assessing gene expression levels and identifying sequence variants, and to clarify their potential contribution to ESRD-associated anemia. ***Materials and Methods:*** Peripheral blood samples were collected from hemodialysis patients and age-matched healthy controls. Genomic DNA and total RNA were extracted using standard EDTA-based protocols and TRIzol reagent, respectively. SLC40A1 gene expression was quantified by real-time quantitative PCR, while DNA sequencing was performed to detect sequence variations within the gene. Identified variants were compared with previously reported mutations, and novel findings were submitted to the NCBI GenBank database. ***Results:*** Quantitative PCR analysis demonstrated a marked downregulation of SLC40A1 expression in hemodialysis patients compared with healthy controls, with a fold-change value of 0.344, indicating significant transcriptional suppression of ferroportin. Sequence analysis revealed several known variants, as well as a novel insertion mutation that was successfully deposited in GenBank (accession number: LC872842.1). ***Conclusion:*** The observed downregulation of SLC40A1 expression, together with the presence of genetic variations, suggests a molecular basis for impaired iron export and intracellular iron sequestration in hemodialysis patients. These findings underscore the central role of the Hepcidin–ferroportin axis in the pathophysiology of ESRD-associated anemia and highlight the importance of molecular evaluation of iron-regulatory genes in this population.

Keywords: Ferroportin, SLC40A1, Hemodialysis, Gene Expression, Anemia.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive condition affecting more than 10% of the global population and is associated with substantial morbidity and mortality. In its advanced stage, end-stage renal disease (ESRD), renal replacement therapy such as hemodialysis becomes essential [1,2]. Anemia is one of the most common and clinically significant complications of ESRD, contributing to reduced quality of life and increased hospitalization and mortality rates [2,3].

Anemia in CKD is multifactorial, resulting from impaired erythropoietin (EPO) production, chronic inflammation, and disturbances in iron metabolism [3,4]. A key mechanism is functional iron deficiency, in which iron stores are adequate but unavailable for erythropoiesis due to Hepcidin-mediated iron sequestration [4,5]. Inflammatory cytokines, particularly interleukin-6, enhance

Hepcidin synthesis, thereby exacerbating iron restriction in hemodialysis patients [5–7].

Hepcidin regulates systemic iron homeostasis through its interaction with ferroportin (FPN1), the only known cellular iron exporter encoded by the SLC40A1 gene. Hepcidin binding induces ferroportin internalization and degradation, leading to reduced iron absorption and impaired iron release from macrophages and hepatocytes [7,8]. In ESRD, persistently elevated Hepcidin levels promote ferroportin suppression, contributing to functional iron deficiency and reduced responsiveness to erythropoiesis-stimulating agents [6–9].

Ferroportin, encoded by SLC40A1 on chromosome 2q32, is expressed in enterocytes, macrophages, and hepatocytes and plays a central role in iron homeostasis [8,10]. Genetic variants in SLC40A1 may impair iron export or alter Hepcidin sensitivity, thereby influencing iron availability and anemia severity [11,12]. However, data on SLC40A1 gene expression and genetic variants in hemodialysis populations remain limited, particularly in Middle Eastern cohorts. Therefore, this study aimed to investigate SLC40A1 gene expression and genetic variants in hemodialysis patients from Mosul City to better understand their contribution to iron dysregulation and ESRD-related anemia.

MATERIALS AND METHODS

Study Population and Blood Sample Collection

A total of 50 individuals were enrolled in this study, including 25 patients undergoing maintenance hemodialysis and 25 age- and sex-matched healthy controls. The study population included both males and females, with ages ranging from 25 to 70 years. Written informed consent was obtained from all participants prior to inclusion in the study.

From each participant, 2 mL of peripheral venous blood was collected under aseptic conditions. One portion of the sample was transferred into an EDTA-containing tube and used exclusively for genomic DNA extraction. The remaining portion was immediately transferred into a tube containing TRIzol reagent for total RNA isolation. All samples were processed promptly to ensure preservation of nucleic acid integrity.

RNA Extraction and cDNA Synthesis

Total RNA was extracted using Trizol reagent (TransGen Biotech, China) according to the manufacturer's protocol, which included sequential steps of homogenization, phase separation, precipitation, and purification. The isolated RNA was dissolved in RNase-free water, quantified, and stored at –80 °C until further use [13,14].

Complementary DNA (cDNA) was synthesized from purified RNA using reverse transcriptase (TransGen Biotech, China) following the manufacturer's instructions. The reaction was performed in a final volume of 20 µL, with incubation at 42 °C for 60 min followed by enzyme inactivation at 85 °C for 5 min [15].

Gene Expression Analysis (qPCR)

Quantitative real-time PCR (qPCR) was performed to assess *SLC40A1* gene expression relative to a housekeeping gene (GAPDH). Each reaction mixture (20 µL) contained SYBR Green Master Mix (TransGen Biotech, China), gene-specific primers, and 100 ng of cDNA. The thermal cycling conditions included an initial denaturation at 95 °C, followed by 40 cycles of denaturation, annealing, and extension. Melting curve analysis was conducted to verify amplification specificity. Relative expression was calculated using the 2^–ΔΔCt method [16]. The primer sequences used for quantitative expression analysis of the SLC40A1 gene and the housekeeping gene (GAPDH) are shown in Table 1.

RESULTS

Table 1: Primers Used for *SLC40A1* Gene Expression Analysis

Primer	Sequence (5' → 3')	Target
SLC40A1-RT-F	TGAATGCCACAATACGAAGG	*SLC40A1* (Forward)
SLC40A1-RT-R	CCAAGTTCCATCCCGAAATA	*SLC40A1* (Reverse)
H.K-F	GACCCAGATCATGTTTGAG	Housekeeping (Forward)
H.K-R	CGTACAGGGATAGCACAG	Housekeeping (Reverse)

Note: Housekeeping (H.K) primers were employed as internal controls to normalize *SLC40A1* gene expression levels in qPCR analysis.

Genomic DNA Extraction and Purity Assessment

Genomic DNA was extracted from EDTA-anticoagulated blood using the TransGen Biotech DNA extraction kit, which involves Proteinase K digestion, lysis buffer treatment, ethanol precipitation, and purification through spin columns. The DNA was eluted in nuclease-free water and stored at –20 °C. DNA concentration and purity were assessed using a BioDrop spectrophotometer by measuring the A260/A280 ratio, with values between

1.8 and 2.0 considered acceptable for downstream applications [17].

DNA Sequencing of the *SLC40A1* Gene

Polymerase chain reaction (PCR) amplification of the selected exonic region of the *SLC40A1* gene was carried out using gene-specific primers. The amplification products were subsequently purified to remove unincorporated nucleotides and residual primers, ensuring high-quality templates for sequencing. Purified PCR products were then subjected to bidirectional Sanger sequencing (Psomagen, USA) to obtain accurate nucleotide reads in both forward and reverse directions. The obtained chromatograms were analyzed, and the resulting nucleotide sequences were aligned and compared with the reference *SLC40A1* sequence available in the NCBI GenBank database using the BLAST tool, in order to confirm primer specificity and identify any potential nucleotide substitutions or mutations [18,19]. The primer se

quences used for the PCR amplification of the *SLC40A1* gene prior to sequencing are presented in Table 2.

Table 2: Include the primer that have been used in DNA sequence

Primer	Sequence
SLC40A1–F	5' TCGAAGATCTTTCCCCATGA 3'
SLC40A1 –R	5' CTCCACTGCCCCAAACTTTA 3'

RESULTS AND DISCUSSION

Gene Expression of *SLC40A1*

The results of the present study indicate a significant alteration in the expression level of the *SLC40A1* gene in hemodialysis patients compared with the control group. Figure 1 illustrates the quantitative real-time PCR (qPCR) amplification curves for *SLC40A1* obtained from hemodialysis patients and healthy controls. In these curves, the x-axis represents the number of PCR cycles, while the y-axis reflects fluorescence intensity, which is directly proportional to the amount of amplified DNA. The red curves correspond to patient samples, whereas the green curves represent the control group.

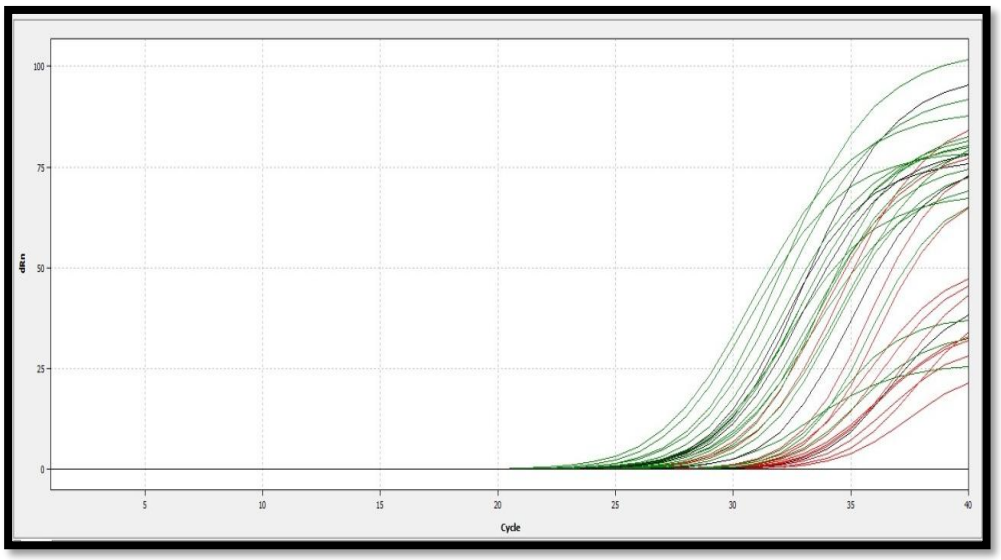

Figure 1. The curve for q-PCR

Analysis of the amplification profiles revealed a consistent delay in signal detection in patient samples, as evidenced by higher cycle threshold (Ct) values, indicating reduced baseline expression of the *SLC40A1* gene. Relative quantification further supported this observation, showing marked downregulation of *SLC40A1* mRNA expression in all patient samples when normalized to the control value of 1.0.

Gene expression analysis demonstrated a significant downregulation of *SLC40A1* in hemodialysis patients compared with healthy controls. The mean fold-change value was **0.344**, (**Figure 2; Table 4**) indicating a marked reduction in ferroportin transcriptional expression among patients. This consistent decrease in *SLC40A1* expression supports the presence of impaired iron export at the molecular level in hemodialysis patients.

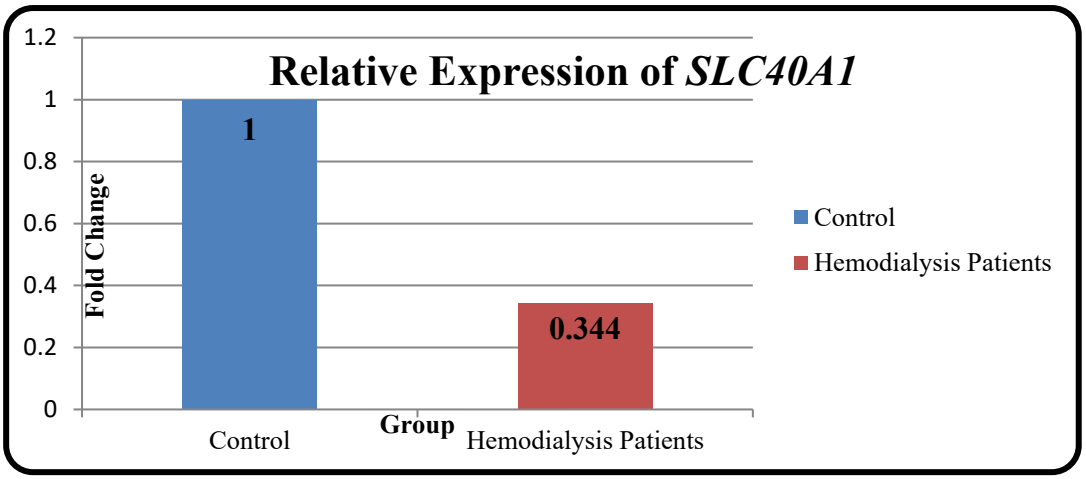

Figure 2. shows the level of gene expression of *SLC40A1* gene in patients and the control group.

Table 4: CT values, the level of gene expression for the *SLC40A1* gene and the housekeeping gene levels for patients are compared with the control group.

Gene Expression folding calculate							
Sample		CT target gene	CT housekeeping gene	Δ CT target gene	Δ CT control	Δ Δ CT	Gene Expression folding
Control	C1	29.10	27.67	1.43	1.43	0	1
	C2	29.05	27.60	1.45	1.43	0	1
	C3	29.20	27.75	1.45	1.43	0	1
	C4	29.00	27.55	1.45	1.43	0	1
	C5	29.15	27.70	1.45	1.43	0	1
	Mean						**1**
patients	1	33.60	31.39	2.21	1.43	0.78	0.58
	2	33.10	29.11	3.99	1.43	2.56	0.17
	3	35.58	28.56	7.02	1.43	5.59	0.02
	4	33.80	30.42	3.38	1.43	1.95	0.26
	5	33.83	29.31	4.52	1.43	3.09	0.12
	6	34.49	32.41	2.08	1.43	0.65	0.64
	7	33.61	31.12	2.49	1.43	1.06	0.48
	8	33.80	29.25	4.55	1.43	3.12	0.11
	9	33.22	33.10	0.12	1.43	−1.31	2.48
	10	32.33	26.34	5.99	1.43	4.56	0.04
	11	34.21	29.06	5.15	1.43	3.72	0.07
	12	33.80	28.26	5.54	1.43	4.11	0.06
	13	32.61	30.01	2.60	1.43	1.17	0.44
	14	34.12	25.49	8.63	1.43	7.20	0.01
	15	34.12	28.47	5.56	1.43	4.22	0.05
	16	33.10	29.25	3.85	1.43	2.42	0.19
	17	35.08	32.20	2.88	1.43	1.45	0.37
	18	32.80	29.81	2.99	1.43	1.56	0.34
	19	30.08	28.39	1.69	1.43	0.26	0.84
	20	33.08	27.83	5.25	1.43	3.82	0.07
	21	32.07	27.27	4.80	1.43	3.37	0.09
	22	33.50	30.20	3.30	1.43	1.87	0.27
	23	32.90	29.90	3.00	1.43	1.57	0.34
	24	34.00	30.80	3.20	1.43	1.77	0.29
	25	33.20	29.90	3.30	1.43	1.87	0.27
	Mean						**0.344**

Sequencing of the *SLC40A1* Gene

The integrity of the extracted genomic DNA and the specificity of the PCR amplification were verified using agarose gel electrophoresis, as shown in Figures 3 and 4.

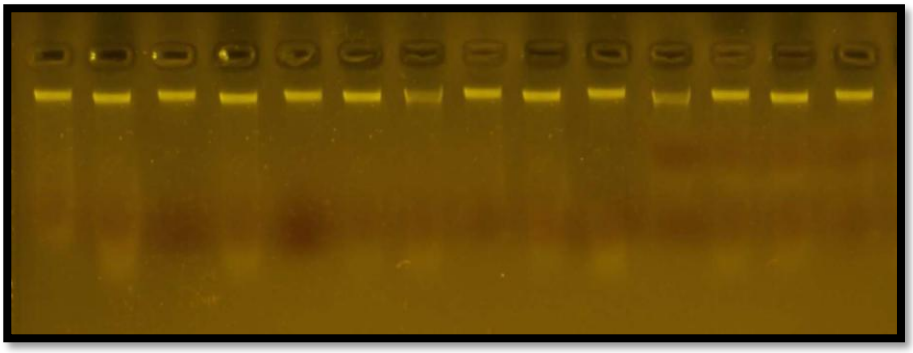

Figure 3. Genomic DNA extracted from peripheral blood samples showing clear, intact DNA bands separated on 1% agarose gel electrophoresis. (This figure confirms the successful extraction and purity of genomic DNA, which was subsequently used for PCR and sequencing analyses.)

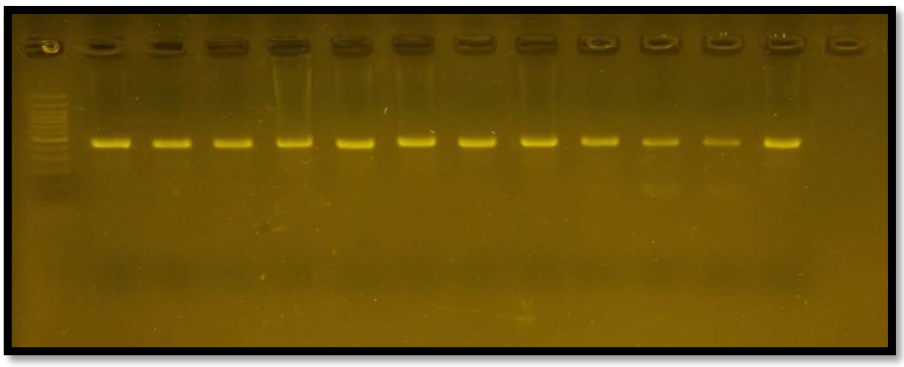

Figure 4. PCR amplification product of the *SLC40A1* gene (430 bp) separated on 2% agarose gel electrophoresis. (A single sharp band at the expected size confirms successful amplification of the target gene prior to sequencing.)

33PCR amplification of the *SLC40A1* gene produced a clear and specific DNA fragment of approximately 430 base pairs, as shown in Figure 3. Sequencing of this fragment from a healthy control sample revealed complete alignment with the NCBI reference sequence (NG_009027.1), showing 100% identity and no nucleotide variations. This perfect match served as an internal control confirming the high specificity of the designed primers and the overall accuracy of the sequencing process Table 5, In contrast, sequencing results from hemodialysis patient samples displayed multiple nucleotide substitutions and insertion variants, as illustrated in Figures 5–10 and summarized in Table 4. The insertion mutations were detected at positions 6218, 6230, 6231, 6242, 6262, and 6264, and are predicted to cause frameshift alterations, potentially leading to premature stop codons and truncated ferroportin proteins. Additionally, several transition mutations were identified, including C→T (position 6340) and C→A (position 6498), which may result in missense changes that could modify amino acid composition and affect the functional conformation of the ferroportin protein. A few variants of uncertain significance (VUS) were also observed, such as ambiguous substitutions T→N or —→N at positions 6214, 6215, and 6301, which may require further sequencing validation for clarification. Interestingly, several patient sequences showed complete identity with the reference gene sequence, serving as additional internal validation controls for sequencing accuracy and confirming the reliability of the amplification and analysis process.

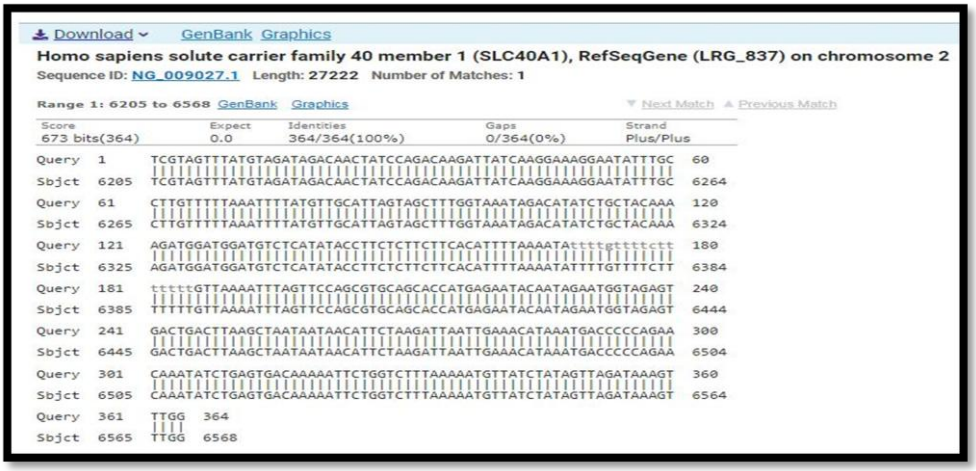

Figure 5. Results of aligning the *SLC40A1* gene sequences with reference sequences from NCBI (Sample 1).

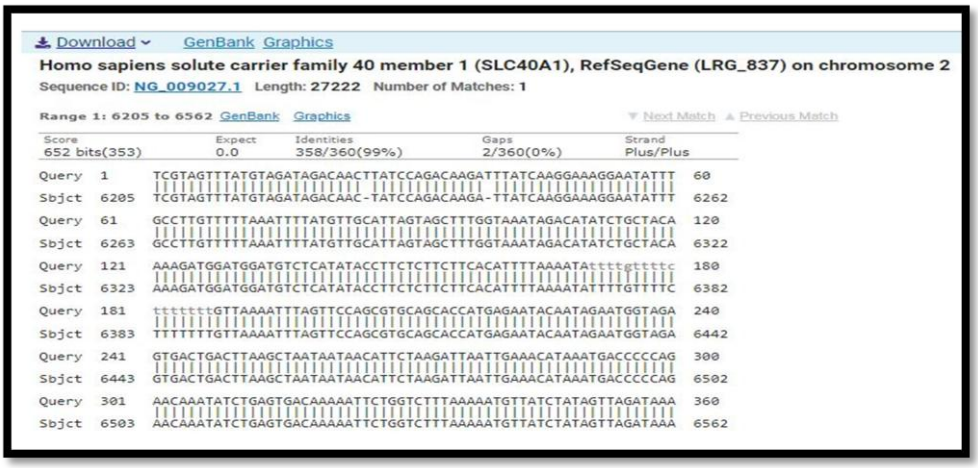

Figure 6. Results of aligning the *SLC40A1* gene sequences with reference sequences from NCBI (Sample 2).

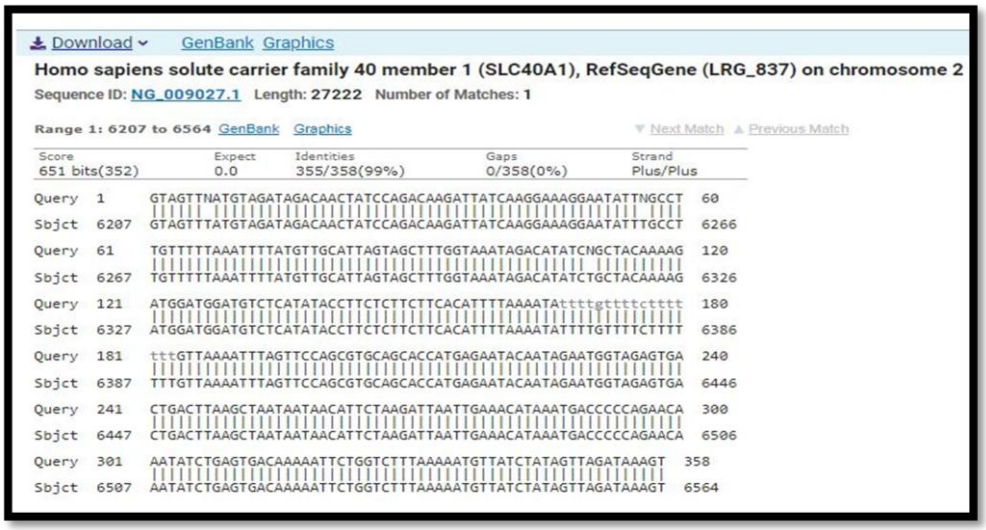

Figure 7. Results of aligning the *SLC40A1* gene sequences with reference sequences from NCBI (Sample 3).

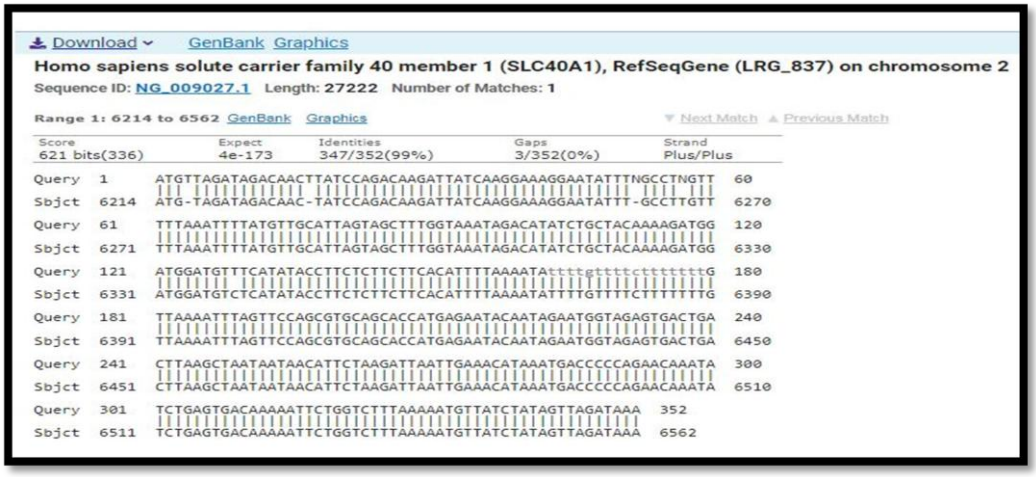

Figure 8. Results of aligning the *SLC40A1* gene sequences with reference sequences from NCBI (Sample 4).

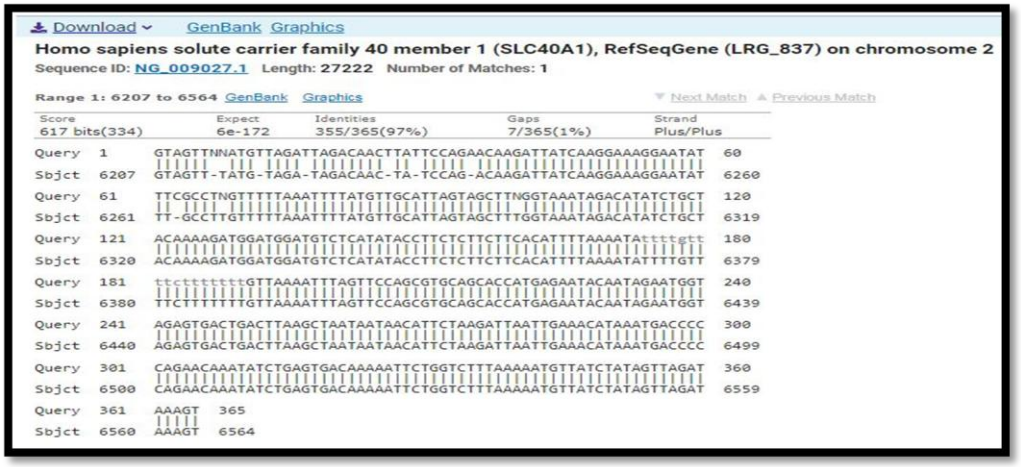

Figure 9. Results of aligning the *SLC40A1* gene sequences with reference sequences from NCBI (Sample 5).

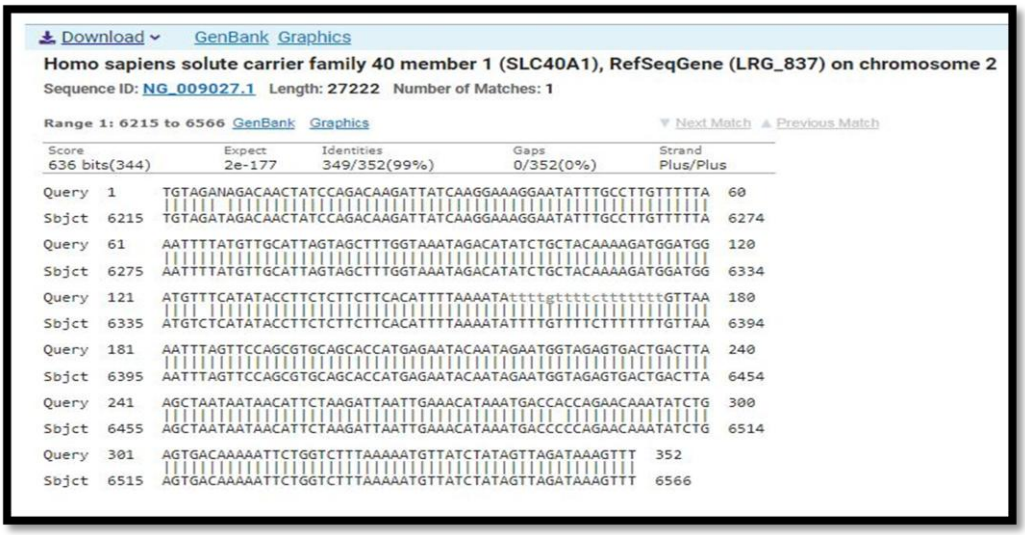

Figure 10. Results of aligning the *SLC40A1* gene sequences with reference sequences from NCBI (Sample 6).

Table 5: Comparison of the DNA sequence of the (*SLC40A1*) gene in the s study samples to the original gene sequence at NCBI.

Figure	Sequence ID	Nucleotide	Location	Mutation Type	Identify	Gaps
Figure (5)	NG-009027.1	No variation	-----	No mutation detected	100%	0%
Figure (6)	NG-009027.1	- → T	6230	Addition	99%	0%
	NG-009027.1	- → T	6244	Addition	99%	0%
Figure (7)	NG-009027.1	T→ N	6214	Unknown	99%	0%
	NG-009027.1	T→ N	6263	Unknown	99%	0%
	NG-009027.1	T→ N	6316	Unknown	99%	0%
Figure (8)	NG-009027.1	- → T	6218	Addition	99%	0%
	NG-009027.1	- → T	6231	Addition	99%	0%
	NG-009027.1	- → N	6262	Addition	99%	0%
	NG-009027.1	T → N	6267	Unknown	99%	0%
	NG-009027.1	C → T	6340	Transition	99%	0%
Figure (9)	NG-009027.1	T → N	6222	Unknown	99%	0%
	NG-009027.1	C → A	6498	Transition	99%	0%
Figure (10)	NG-009027.1	- → N	6214	Addition	97%	1%
	NG-009027.1	T → N	6215	Unknown	97%	1%
	NG-009027.1	- → T	6219	Addition	97%	1%
	NG-009027.1	- → T	6224	Addition	97%	1%
	NG-009027.1	- → T	6233	Addition	97%	1%
	NG-009027.1	- → T	6236	Addition	97%	1%
	NG-009027.1	- → A	6242	Addition	97%	1%
	NG-009027.1	- → C	6264	Addition	97%	1%
	NG-009027.1	T → N	6269	Unknown	97%	1%
	NG-009027.1	T → N	6301	Unknown	97%	1%

DISCUSSION

At the molecular level, quantitative real-time PCR analysis demonstrated a marked downregulation of SLC40A1 expression in hemodialysis patients compared with healthy controls. This finding supports disruption of the Hepcidin–ferroportin axis, in which elevated Hepcidin levels promote ferroportin internalization and degradation, thereby limiting iron export and reducing plasma iron availability for effective erythropoiesis [20]. Persistent inflammation

in chronic kidney disease, particularly mediated by interleukin-6, further enhances Hepcidin synthesis and reinforces ferroportin suppression [21]. These observations are in agreement with previous studies linking reduced ferroportin expression to functional iron deficiency and impaired erythropoietic response in renal anemia [22,23].

Sequence analysis of the SLC40A1 gene revealed the presence of several nucleotide substitutions and insertion mutations among patient samples. Insertions predicted to induce frameshift changes and premature stop codons may result in truncated or non-functional ferroportin proteins [24,25], while transition mutations may alter amino acid residues essential for iron transport activity and protein stability [26,27]. Such genetic variations could exacerbate intracellular iron retention and contribute to iron dysregulation in affected patients. However, the identification of patient samples with sequences identical to the reference gene indicates that structural genetic alterations are not universal, underscoring the predominant role of inflammatory and post-translational regulatory mechanisms, particularly excessive Hepcidin activity, in disrupting iron homeostasis in chronic kidney disease [28].

Taken together, the transcriptional and genetic findings of this study indicate that iron dysregulation in hemodialysis patients arises from a complex interplay between reduced SLC40A1 expression, chronic inflammation, Hepcidin-mediated ferroportin inhibition, and, in some cases, gene-level variants. This multifactorial disruption ultimately restricts iron bioavailability and contributes to impaired erythropoiesis in end-stage renal disease.

CONCLUSION

Anemia in hemodialysis patients arises from reduced erythropoietin production, chronic inflammation, and Hepcidin-mediated suppression of ferroportin. Decreased SLC40A1 expression and associated mutations may further impair iron export, leading to iron sequestration and functional iron deficiency. These findings highlight the role of the Hepcidin–ferroportin axis in renal anemia and support the value of integrating SLC40A1 molecular assessment into routine clinical evaluation.

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Ethical clearance: The Ethics Committee of the Nineveh Health Directorate (Department of Health, Mosul, Iraq) approved the study under official document No. 1538, dated 13 January 2025.
Conflict of interest: None.
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