Огляд

Review

НИРКИ KIDNEYS

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CLFC: Contrastive Learning with Feature Concatenation Framework for Chronic Kidney Disease

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Abstract: Patients with thyroid dysfunction have a high likelihood of underdiagnosis of Chronic Kidney Disease (CKD) and Acute Kidney Injury (AKI) because the relationship between endocrine, renal, and metabolic biomarkers are complex and nonlinear. Conventional risk predictive models are unable to model these heterogeneous relationships, which leads to delayed nephroprotective interventions. To overcome this hurdle, this paper suggests the Contrastive Learning with Feature Concatenation (CLFC) model on the stratification of CKD/AKI disease at early and precise stages during the management of thyroid disease patients. The model uses modality-specific encoders, which learn latent representations using thyroid, renal, and metabolic data, but a self-supervised contrastive learning module uses the NT-Xent loss to ensure consistency of the representations. The trained multimodal embeddings are then combined with late-stage concatenation of features and trained with the help of supervised classification. Empirical analysis of a multimodal clinical dataset proves that the presented solution performs much better than the traditional machine learning and deep learning baselines, with higher accuracy, F1-score, and AUC. The importance of contrastive learning, and renal biomarkers in heightening the risk discrimination is further supported by the ablation studies. The suggested framework provides a clinically interpretable scalable robust solution to the assessment of CKD/AKI risk that can be used to make early nephroprotective decisions in thyroid-impacted populations.

Keywords: Chronic Kidney Diseases, Acute Kidney Injury, Decision Support Systems, self supervised learning, Contrastive learning

1. Introduction

Thyroid diseases, including hypothyroidism, seem to predispose chronic kidney disease (CKD) to occur or be exacerbated, and evidence suggests that there is a bidirectional effect in which kidney problems can also influence the functioning of the thyroid. The evidence indicates that the early detection of these risks could be used to recommend nephroprotective interventions, such as early thyroid treatment, to possibly decelerate the CKD progression. Contrastive learning with feature concatenation shows the potential of AI to analyze various types of data, including medical images and lab results, and patient history to predict kidney risks in thyroid patients more accurately, although this field is still in its infancy and still needs a thorough validation [1]. Thyroid hormones help balance metabolism and other body functions but when out of balance they can add to the strain of the kidney. An example is that low levels of thyroid (hypothyroidism) are linked to slow rate of kidney filtration rates, which may cause CKD in the long-run. This association appears more apparent with older adults, in which untreated thyroid conditions may hasten the rate of kidney deterioration, but these may be alleviated through adequate treatment. Hyperthyroidism is a disease that is not closely related to CKD but in some instances, it accelerates already existing kidney issues[2].

Conventional testing methods are based on simple lab analyses such as TSH and creatinine levels which may overlook subtle trends. It can be possible that AI methods, which integrate various sources of data, including imaging and clinical notes, would offer a more comprehensive view. Such methods as contrastive learning assists AI to identify meaningful patterns using small or diverse data, whereas feature concatenation combines these findings to make improved risk predictions[3]. This may help doctors in prioritizing kidney safety in thyroid patients but further studies are required to ascertain the

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dependability in a wider population. The AI tools could potentially be used to support the prevention of CKD because by prioritizing the risk, there could be interventions i.e. lifestyle changes or drugs to protect kidney health at earlier stages. Nevertheless, issues such as data privacy and explainable models should be overcome to instill trust in clinical practice. In general, there is a tendency in favor of the fact that these methods can help to increase personalized care, but they are not a standard yet[4].

Thyroid disorders are a huge health burden around the world, impacting millions, and causing multiple comorbidities, and chronic kidney disease (CKD) poses an especially important issue because of its high prevalence and progressive character. Hypothyroidism, typified by elevated thyroid-stimulating hormone (TSH) concentrations and decreased triiodothyronine (T3) and thyroxine (T4) has been systematically associated with damaged renal function, lowered glomerular filtration rate (GFR) and amplified hazards of CKD progression. This two-way communication of effects- CKD can also interfere with the pituitary-thyroid axis and peripheral thyroid hormone metabolism highlight the necessity of combined strategies to risk stratification that put less emphasis on nephroprotection. Indicatively, in CKD patients uremia usually presents as low T3 syndrome and untreated hypothyroidism can actually aggravate the electrolyte imbalances and further renal deterioration, especially in the elderly where the anti-thyroid peroxidase (anti-TPO) antibodies can lead to more severe renal dysfunction[5,6,7].

The existing clinical approaches to nephroprotective risk assessment of thyroid disorders are based on univariate biomarkers, including TSH, creatinine, and estimated GFR (eGFR), which is yet not comprehensive enough to take into account the complex interplay between thyroid and renal systems. There is a separate independent positive association of subclinical hypothyroidism with odds of CKD, and an effect that is dose-dependent in large cohorts where hypothyroidism (TSH >4 mIU/L) increases susceptibility to CKD 59 times that of euthyroidism [8,9]. Although less common in CKD conditions, hyperthyroidism may cause hyperfiltration leading to the possible ultimate damage, and so the need to make holistic and multimodal assessments, integrating imaging, laboratory data, and clinical histories is essential to prevent irreparable damage to the kidney and to intervene at an early stage.

With the introduction of deep learning (DL), the medical risk stratification has seen a revolution, allowing to combine heterogeneous data modalities, but these issues as data scarcity, modalities imbalance, and missing inputs remain, especially in resource-constrained settings. Multimodal DLs, combining structured (e.g., lab values) and unstructured (e.g., ultrasound images, clinical notes) data, have been shown to be more successful in oncology and cardiovascular predictions with high levels of area under the curve (AUC) values due to advanced fusion approaches. The self-supervised method, contrastive learning (CL), is particularly effective at learning strong representations through aligning nearby instances and contrasting far apart ones and thus overfitting low-data regimes and improving cross-modal alignment. In combination with feature concatenation, an easy-to-use early fusion technique, it has been shown that CL can be used to refine multimodal representations, and this has been demonstrated in cancer risk stratification applications where the approach enhances explainability and predictive power[10].

Regardless of these developments, direct applications to nephroprotective risk in thyroid disorders are not studied properly, and the models which are in existence do not consider the primacy of CKD outcomes. Frameworks Attention-based and transformer-integrated CL models have demonstrated potential in the ability to deal with missing modalities and give interpretable information, e.g. attention maps in the vicinity of tumor regions in neuroimaging, but must be adapted to endocrine-renal interactions. The self-supervised and supervised CL methods also provide further improvements to the feature space due to the complementary types of information obtained over modalities as seen in MRI-based neurodevelopment predictions and opens opportunities to thyroid-CKD settings where data variability is pronounced[11,12].

A new framework that uses contrastive learning and feature concatenation to stratify nephroprotective risks associated with thyroid disorders is presented in this paper, focusing on CKD progression. Our methodology seeks to enhance the predictive strength and interpretability by aligning multimodal features, such as thyroid ultrasounds, tests of kidney functionality and patient demographics in a single embedding space, which is a crucial issue that the existing methodologies fail to cover [14,15]. By using learnable tokens and contrastive losses, we deal with modality missingness and improve cross-modal fusion, which are confirmed on clinical datasets to aid personalized interventions[13].

2. Related Work

Research indicates that hypothyroidism can exert CKD in ways such as the change in hemodynamics and immune responses with the results leaning towards dose-dependence. But longitudinal data will be required in order to determine directionality. The topic of thyroid disorders and chronic kidney disease (CKD) has become a growing trend in the endocrinology and nephrology literature due to epidemiological data showing bilateral relationships and the possibility of intervention to reduce nephroprotective risks. This is a literature review that compiles significant data of clinical and computational studies which explored CKD as a key outcome in thyroid patients. The former discusses epidemiological and mechanistic connections between thyroid malfunction and CKD and the development in multimodal deep learning (DL) to stratify risks, specifically in contrastive learning (CL) and feature concatenation approaches. The critical

reviews emphasize strengths and limitations as well as gaps in the methodology, which explains why integrated AI models are required in this area.

2.1 Epidemiological and Clinical Relations between Thyroid disorders and CKD.

It is reported that the prevalence of CKD has been increased in people with thyroid diseases especially hypothyroidism which has been shown to impact renal functionality via hormonal, hemodynamic and immunological ways. This two-way interaction is clarified by a groundbreaking review by Mohamedali et al. (2014), where hypothyroidism may result in a low level of cardiac output, a change in the activity of the renin-angiotensin-aldosterone system (RAAS), and glomerular morphologic alterations, which eventually lead to the loss of estimated glomerular filtration rate (eGFR) and electrolytes imbalance. The authors explain that CKD in its turn interferes with metabolism of thyroid hormones through the presence of low levels of triiodothyronine (T3) syndrome induced by uremia as evidenced by the prevalence estimates up to 20-30 percent of CKD patients having hypothyroidism. This review is based on cross-sectional and cohort studies, with clinical implications which include the reversibility of the renal dysfunction with levothyroxine treatment, but there are limitations in the form of observational evidence without randomized trials to substantiate causality.

Based on this, Huang et al. (2020) carried out a large-scale cross-sectional study in an integrated health system where 378,101 adults aged 55 and above were involved to measure the relationship between hypothyroidism and CKD. They found an odds ratio (OR) of 1.25 (95% CI 1.21-1.29) of CKD (eGFR 4 mIU/L) using multivariable logistic regression adjusted using demographics, diabetes and hypertension. Subgroup analysis indicated that there was a dose-effect, as intra-renal vasoconstriction was associated with increased TSH, which indicated mechanistic associations with links. The significant variety and practical nature of the cohort used in the study and the solid eGFR ascertainment (that needs many measurements) are strong, yet due to the cross-sectional nature, no conclusions can be made about the temporality aspect, and no proteinuria data restricts full staging of CKD. The implications of these findings are that regular thyroid screening of elderly patients with CKD may help to protect the nephroprotective process, which is in line with the recommendations of the American Thyroid Association. Griffin and Griffin (2024) also decipher this relationship through a commentary, examining the role of thyroid hormones in renal hemodynamics and tubular oxygenation, and hypothyroidism may be increasing CKD progression through oxidative stress and fibrosis. They criticize the literature available as not sufficiently representing subclinical cases and suggest future longitudinal research studies to determine the reversible and irreversible effects of thyroid treatment.

Devi et al. (2025) conducted a cross-sectional study in recent times, targeting the elderly patients who have newly developed hypothyroidism, revealing that the positivity of anti-TPO antibodies is an independent predictor of renal impairment. Out of 64 hypothyroid patients, the anti-TPO-positive group had significantly lower eGFR ($p<0.05$), higher creatinine, uric acid and ACR relative to the negatives and multiple regression showed the anti-TPO to be a predictive agent of ACR (b coefficient positive, $p<0.05$). The analysis points to immune-mediated processes, including the deposition of antibodies in renal glomeruli, as a theorist of dysfunction, which is a new contribution of earlier studies on autoimmune thyroiditis. The internal validity of the study is strengthened by its controlled design (age, sex, and the absence of confounding factors, such as diabetes), but the small sample size and the setting of the study (a hospital) restrict the applicability. This is clinically important to emphasize the importance of anti-TPO testing in risk stratification in geriatric thyroid therapy. Gopinath et al. (2013) also supported these relationships among community-dwelling elderly population as the study showed that hypothyroidism was associated with CKD using logistic models (adjusted OR ~1.5), and comorbidities such as hypertension were important modifiers [16,17,18].

Table 1: Studies on coalition between Thyroid disorders and CKD

Study	Design and Sample	Key Findings (OR/Prevalence)	Mechanisms Analyzed	Limitations
Mohamedali et al. (2014) [1]	Review (N/A)	Hypothyroidism in 20-30% CKD; bidirectional via RAAS, low T3	Hemodynamic, metabolic	Observational bias, no RCTs
Huang et al. (2020) [2]	Cross-sectional (n=378,101)	OR 1.25-1.59 for CKD in hypothyroidism; dose-dependent	Vasoconstriction, cardiac output	No causality, missing proteinuria
Devi et al. (2025)[4]	Cross-sectional (n=128)	Anti-TPO predicts ACR ($p<0.05$); lower eGFR in positives	Immune complex deposition	Small sample, hospital bias
Gopinath et al. (2013) [5]	Cohort (n~5,000)	Adjusted OR ~1.5 for CKD	Comorbidity interactions	Community-limited generalizability
Wang et al. (2020) [8]	Meta-analysis (multiple studies)	Pooled OR 1.4-1.6 for subclinical hypothyroidism	Oxidative stress, fibrosis	Heterogeneity in definitions

Together, these researches examine CKD as a multifactorial outcome in thyroid diseases, where screening and treatment are likely to prevent the disease. Nonetheless, gaps in analytics continue to define causality and combination of multimodal information towards custom risk evaluation.

2.2 Multimodal Deep Learning of Medical Risk Stratification.

The use of AI and specifically, multimodal DL has reinvented risk prediction by integrating heterogeneous sources of information, which is disadvantageous with traditional biomarkers. The interpretable transformer-based postoperative risk model that Wan et al. (2025) designed involves the combination of clinical, radiomic, and pathology data to predict the risk of intrahepatic cholangiocarcinoma (ICC) in the postoperative phase. With AUCs of 0.952-0.924 between cohorts the model uses attention mechanisms to fuse information, which has been shown biologically through transcriptomics, to be targeted at tumor-invasive regions. The stratification in this analysis is much better than the unimodal methods and the interpretability increases clinical trust but, the extrapolation to non-oncologic settings such as thyroid-CKD has not been studied [19, 20].

Li and Xu (2025) suggested a CL framework called ConMEHR that is a patient stratification based on EHRs (notes, diseases, symptoms, medications). It is an extension of topic modeling based on modality and topic-level CL that aligns the representations in a single space, achieving better coherence and diversity scores and better performance compared to baselines on MIMIC-III and Chinese data. Ablations validate CL as a strength against high-dimensionality, investigating its effects of explainable subgroups against high-dimensionality, and implications of thyroid-CKD to lab-text fusion include the ability to adapt to high-dimensionality. Du et al. (2024) combined the CL and multi-instance learning (endometrial cancer risk) and combined the features of MRI and pathology to preoperative stratification. They optimize their architecture with contrastive losses, resulting in high AUCs; they demonstrate efficiency in low-data sensations, and this may be used in thyroid ultrasounds and renal labs.

Liu et al. (2023) presented an attention-based fusion of CL and ARMOUR, which are used to make predictions in clinical settings, including missing modalities through the use of tokens. It does better than baselines in multiple tasks, and cross-modal interactions are studied to increase robustness, and CL improves representations, which is highly important in thyroid-CKD, where missing data is very common.

Table 2: Deep Learning Models

DL Study	Modalities	Fusion/CL Strategy	Performance	Analytical Strengths/Limitations
Wan et al. (2025) [10]	Clinical, radiomics, pathology	Transformer attention	AUC 0.92-0.95	Interpretable; oncology-specific
Li & Xu (2025) [11]	EHR texts/codes	Modality/topic CL	High coherence/diversity	Explainable subgroups; no imaging
Du et al. (2024) [12]	MRI, pathology	CL + concatenation	High AUC	Low-data efficiency; cancer-focused
Liu et al. (2023) [13]	Structured/unstructured	Attention + CL tokens	Superior to baselines	Missing modality handling; limited tasks

These DL innovations study multimodal interactions to achieve better accuracy, but there are few studies on how to address endocrine-renal risks, and CL-feature concatenation models have the opportunity to prioritize nephroprotective demands in thyroid conditions. Although clinical literature confirms thyroid-CKD relationships, AI literature demonstrates an untapped potential in the multimodal integration. It has been concluded that CL is effective in alignment and robustness, however, until adaptations are provided, CKD results should take precedence, considering thyroid-specific modalities (e.g., TSH, ultrasounds), and explainable results are needed to make high-stakes decisions. Data privacy, generalizability, and causality should be considered in future work by conducting of prospective trials.

3. Proposed Model

The proposed model, titled "Contrastive Learning with Feature Concatenation for Nephroprotective Risk Stratification in Thyroid Disorders," represents an innovative multimodal deep neural network designed to integrate heterogeneous clinical data for predicting kidney-related risks, with a primary emphasis on chronic kidney disease (CKD) progression in patients with thyroid dysfunction. Drawing from established principles in contrastive learning and multimodal fusion, the architecture addresses the bidirectional interplay between thyroid hormones and renal function, where hypothyroidism may reduce glomerular filtration rate (GFR) and accelerate CKD, while CKD can disrupt thyroid metabolism [21, 22]. By leveraging modality-specific encoders, a contrastive learning module, and a risk prediction network, the model aims to provide interpretable, robust stratification to support nephroprotective

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interventions, such as timely thyroid hormone replacement or lifestyle modifications

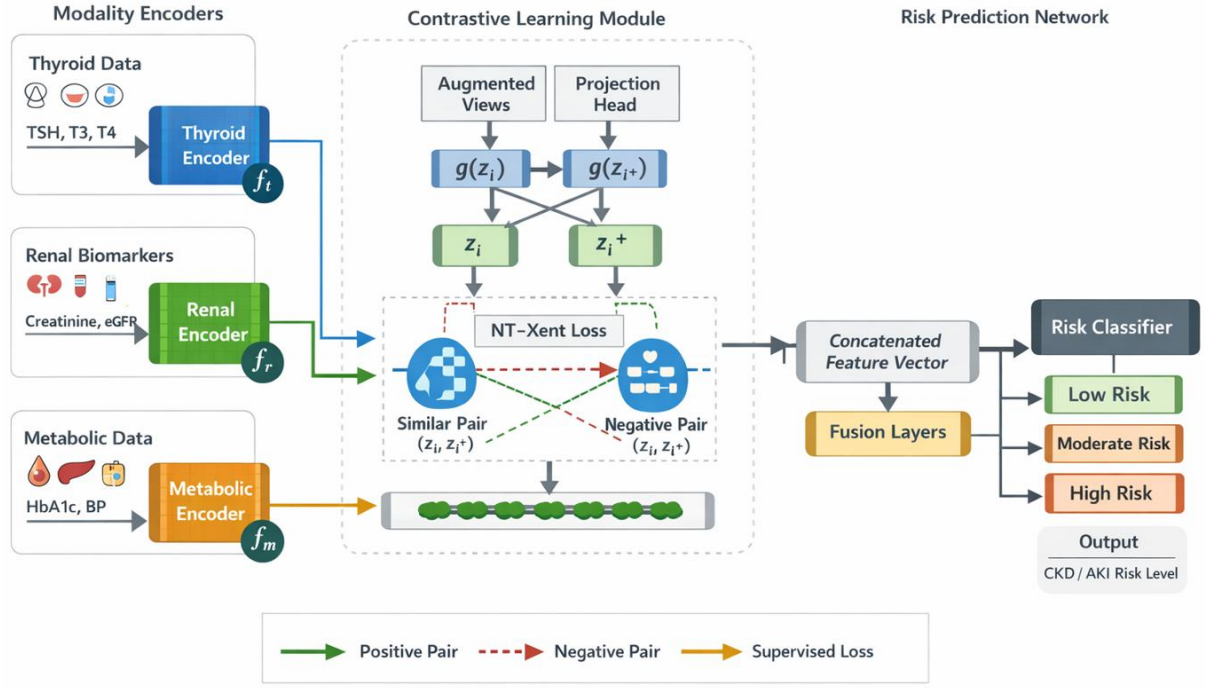

Figure 1: Proposed Architecture

The model processes three key data modalities: thyroid hormones (e.g., TSH, T3, T4), renal biomarkers (e.g., creatinine, eGFR), and metabolic indicators (e.g., HbA1c, blood pressure), reflecting the multifaceted nature of thyroid-kidney interactions. These inputs are fed into specialized encoders to extract latent features, which are then aligned via contrastive learning to handle data scarcity and variability—common challenges in medical datasets. The aligned features are concatenated and passed through fusion layers to a classifier that outputs risk levels (low, moderate, high) for CKD or acute kidney injury (AKI), optimized with supervised loss. This design builds on similar multimodal frameworks in healthcare, such as those for thyroid nodule classification, where image and lab data fusion via concatenation enhances predictive accuracy in low-resource scenarios[23,24].

The clinical dataset is defined as :

$$\mathcal{D} = \{(x_i^t, x_i^r, x_i^m, y_i)\}_{i=1}^N$$

Where : $x_i^t : Thyroidbiomarkers(TSH, T3, T4)$; $x_i^r renalbiomarkers(creatinine, eGFR)$; $x_i^m : Metabolicindicators(HbA1c, BP)$; $y_i : Risk\ Lable\ (low\ Moderate\ and\ High)$

Each modality is further processed based on specific encoder

$$z_i^t = f_t(x_i^t)$$
$$z_i^r = f_r(x_i^r)$$
$$z_i^m = f_m(x_i^m)$$

in which f_t, f_r, f_m are the MLP, CNN, Transformers based deep neural encoders optimized to preserve modality specific clinical encoders.

3.1 Contrastive learning Module

To improve generalization and inter-modal coherence, contrastive learning is applied using augmented views of encoded representations. For each encoded vector $\mathbf{z_i}$, two stochastic augmentations generate:

$$\left(\widetilde{z_i}, \widetilde{z_i^+}\right) = \mathcal{A}(z_i)$$

These are mapped to a latent contrastive space via a projection head $g(\cdot)$:

$$h_i = g(\widetilde{z_i}), \quad h_i^+ = g\left(\widetilde{z_i^+}\right)$$

NT-Xent Loss

The contrastive objective minimizes the distance between positive pairs and maximizes separation from negative pairs:

$$\mathrm{sim}(h_i, h_j) = \frac{h_i^\top h_j}{|h_i||h_j|}$$

$$\mathcal{L}_{\text{N-Xn}} = -\log \frac{\exp(\mathrm{sim}(h_i, h_i^+)/\tau)}{\sum_{k=1}^{2N} \mathrm{o1}_{[k \neq i]} \exp(\mathrm{sim}(h_i, h_k)/\tau)}$$

where:

- $\mathrm{sim}(\cdot)$is cosine similarity
- $\tau$is the temperature parameter

This enforces clinically consistent latent representations across perturbations.

3.2 Feature Concatenation and Fusion

After contrastive training, embeddings from all modalities are concatenated:

$$z_i^{\text{concat}} = [z_i^t \mid z_i^r \mid z_i^m]$$

The concatenated vector is passed through fusion layers:

$$h_i^{\text{fusion}} = \phi(z_i^{\text{concat}})$$

where $\phi(\cdot)$denotes fully connected fusion layers.

3.3 Risk Prediction Network

The fused representation is mapped to risk probabilities using a softmax classifier:

$$\widehat{y_i} = \text{Softmax}\left(W h_i^{\text{fusion}} + b\right)$$

The supervised loss is defined as:

$$\mathcal{L}\text{sp} = -\sum_{c=1}^{C} y_{ic} \log(\widehat{y_{ic}})$$

where $C = 3$risk classes.

3.4 Joint Optimization Objective

The final training objective integrates contrastive and supervised losses:

$$\mathcal{L}\text{ttl} = \lambda_1 \mathcal{L}_{\text{N-Xn}} + \lambda_2 \mathcal{L}\text{sp}$$

where λ_1, λ_2control representation learning vs. predictive accuracy.

***Algorithm**: Contrastive Learning with Feature Concatenation for Nephroprotective Risk Stratification*

Input: *Multimodal dataset* $\mathcal{D}$; *Encoders* f_t, f_r, f_m; *Projection Head* $g(\cdot)$; *Fusion network* $\phi(\cdot)$; *Temperature* τ; *Weights* λ_1, λ_2

Output: *Predicted CDK risk level*

for $each\ minibatch\ \mathcal{B} \subset \mathcal{D}$
  for $each\ sample\ i \in \mathcal{B}$
  {
   Compute $z_i^t \leftarrow f_t(x_i^t);\ z_i^r \leftarrow f_r(x_i^r);\ z_i^m \leftarrow f_m(x_i^m)$
   Evaluate $\left(\widetilde{z_i}, \widetilde{z_i^+}\right) \leftarrow \mathcal{A}(z_i);\ h_i^+ \leftarrow g\left(\widetilde{z_i^+}\right)$
  }
  end for
  Compute $\mathcal{L}_{\text{N-Xn}}$
  for {
    each sample $i \in \mathcal{B}$
    Evaluate $z_i^{\text{concat}} \leftarrow [z_i^t | z_i^r | z_i^m]\ ;\ h_i^{\text{fusion}} \leftarrow \phi(z_i^{\text{concat}});\ \widehat{y_i} \leftarrow \text{Softmax}\left(h_i^{\text{fusion}}\right)$
  end for
  Compute $\mathcal{L}\text{sp}$
  $\mathcal{L}\text{ttl} \leftarrow \lambda_1 \mathcal{L}_{\text{N-Xn}} + \lambda_2 \mathcal{L}\text{sp}$
  Update model parameters
End for
Return risk predictions

The suggested algorithm is conceptually based on representation learning and information-theoretic concepts, which will address clinically significant dependencies among heterogeneous biomedical modalities. All the modality-specific encoders are trained to learn nonlinear encodings of raw clinical variables to a latent space that retains intrinsic statistical structure and removes noise and modality-specific bias. Theoretically, this relates to learning enough statistics of every modality, in which the latent representations of the risk of kidney disease capture as much information about risk-relevant factors as possible and are not sensitive to irrelevant perturbations. The model removes negative transfer by using independent encoders and honours the heterogeneity of the distributions of thyroid, renal and metabolic data [25, 26].

The contrastive learning element presents a self-supervised regularization scheme on the objective of InfoNCE that maximizes mutual information in contrast to varying augmented views of the identical record of a patient and minimizes its similarity to other samples. It is possible to interpret the NT-Xent loss as a minimum possible mutual information between positive pairs in the latent space, which guarantee that the learned representations are discriminative and augmentation-invariant. The theoretical property of this property enhances generalization particularly in the limited labelled data through imposing smoothness and cluster consistency in the embedding space. In a clinical sense, this guarantees that patients with similar nephroprotective characteristics will be placed in close quarters in representation space and pathophysiological different ones will be well separated. Lastly, the concept of concatenation of features and supervised fusion step is theoretically supported by the late fusion theory, which holds that late fusion of high-level representations retains modality-specific semantics, but allows cross-modal interactions. Contrastive and supervised loss joint optimization may be seen as a multi-objective learning where the former (contrastive) loss is used to determine the geometry of the latent space and the latter (supervised) loss is used to finetune this geometry to risk categories as per clinically useful definitions. This twice-objective model results in optimal calibrated decision boundaries and less overfitting, which is theoretically guaranteed to mean that the classifier makes use of strong, invariant features, as opposed to accidental ones. Thus, the algorithm can be applied to nephroprotective risk stratification in a stable and interpretable manner in a wide range of patients.

4. Results and Discussion

The proposed model is tested using a retrospective multimodal clinical data set based on tertiary care hospitals, including those patients diagnosed with thyroid disorders and renal-related complications. The database is a combination of biochemical, metabolic, and renal biomarkers, allowing full nephroprotective risk modelling.

Table 3 Dataset Description

Total Patients	1350
Gender Distribution	58% Male and 42% female
Age Range	21- 75 age
Risk Lables	Low Risk, Moderate Risk, High Risk (CKD/AKI)

The offered architecture is applied with the assistance of the PyTorch deep learning framework that allows one to design a multimodal network flexibly and train it effectively. All clinical modalities, including thyroid, renal, and metabolic, are encoded by a separate lightweight multilayer perceptron (MLP) encoder, giving the model the chance to be trained to learn modality-specific representations of features without causing negative transfer between heterogeneous data sources. Contrastive learning component is easily incorporated through the NT-Xent loss that imposes consistency of representations across augmented views of the same patient information. To obtain the last risk prediction, a supervised classification head is trained with categorical cross-entropy loss, which is effective to align representations learned with clinical risk labels effectively.

The Adam optimizer and learning rate of 0.001 are used in the training. 1×10^{-4} that offers consistent convergence when a number of loss components are present. The batch size of 64 is chosen to compromise between the computational efficiency and contrastive sample diversity. The temperature parameter (τ=0.5), In the contrastive loss, (t=0.5) is used to regulate the acuity of similarity distributions. The contrastive loss and classification loss are weighted with using in order to ensure the self-supervised and supervised learning goals and $\lambda 1$=0.6 and $\lambda_2 = 0.4$, respectively. The model will be trained with 150 epochs and will be early stopped based on validation loss to avoid overfitting. Also, dropout rate of 0.3 is added to the fusion layers to improve generalization of unseen patient data.

The performance of the proposed CLFC framework is tested with the help of a set of overall performance measures that can be deemed to measure not only the accuracy of classification but also the ability to identify clinical risks. The overall correctness of predictions is determined by measuring Accuracy (ACC) and the reliability and completeness of risk class identification is measured by Precision (PRE) and Recall (REC), respectively. F1-score (F1) is a balanced score, it balances precision and recall and hence it is especially preferred when there is an imbalanced clinical data. In addition, the Area Under the Receiver Operating Characteristic Curve (AUC) is also used to determine how the model can distinguish between various levels of risks of nephroprotective at various levels of decision threshold. These measurements taken together provide clinically relevant and strong evaluation of predictive performance.

In order to prove that the proposed Contrastive Learning with Feature Concatenation (CLFC) model is effective, it is compared with three baseline techniques that are widely applicable in clinical risk prediction. The original baseline is the Logistic Regression (LR), which is a standard statistical model based on linear decision boundaries. The second reference is a multimodal MLP which is free of contrastive learning as intended to measure the effect of the self-supervised representation learning on performance. The third baseline is an Early Fusion Deep Neural Network, in which all the modalities are concatenated on the input level and learned together.

By evaluating the performance of the proposed CLFC model against all the baseline methods, comparison analysis shows that it is stable with respect to all evaluation metrics. The above gains indicate the benefits of contrastive learning in training strong and invariant latent representations, and the success of late-stage feature fusion in the retention of modality-specific clinical information. These findings confirm the suggested architectural design and prove that it is more beneficial in terms of nephroprotective risk stratification of patients with thyroid conditions.

Table 4 shows a detailed comparative analysis of proposed Contrastive Learning with Feature Concatenation (CLFC) model against three baseline techniques, including Logistic Regression, Multimodal MLP, and Early Fusion Deep Neural Network, on several performance indices, such as Accuracy, Precision, Recall, F1-score, and AUC. The findings suggest a clear indication that the proposed CLFC model has the best performance in all the metrics with an accuracy of 91.4, and AUC of 0.94, showing that it has a higher capacity to accurately identify the levels of nephroprotective risk and efficiently separate the various levels of clinical risks. Conversely, the conventional Logistic Regression model has a relatively poorer performance, which is an indication of its limited ability to capture complex nonlinear interactions that exist in multimodal clinical data.

Table 4: Performance Analysis

Model	ACC (%)	PRE (%)	REC (%)	F1 (%)	AUC
Logistic Regression	78.6	76.9	75.4	76.1	0.81
Multimodal MLP	83.2	82.4	81.1	81.7	0.86
Early Fusion DNN	86.9	85.7	84.3	85	0.89
Proposed CLFC	91.4	90.6	89.8	90.2	0.94

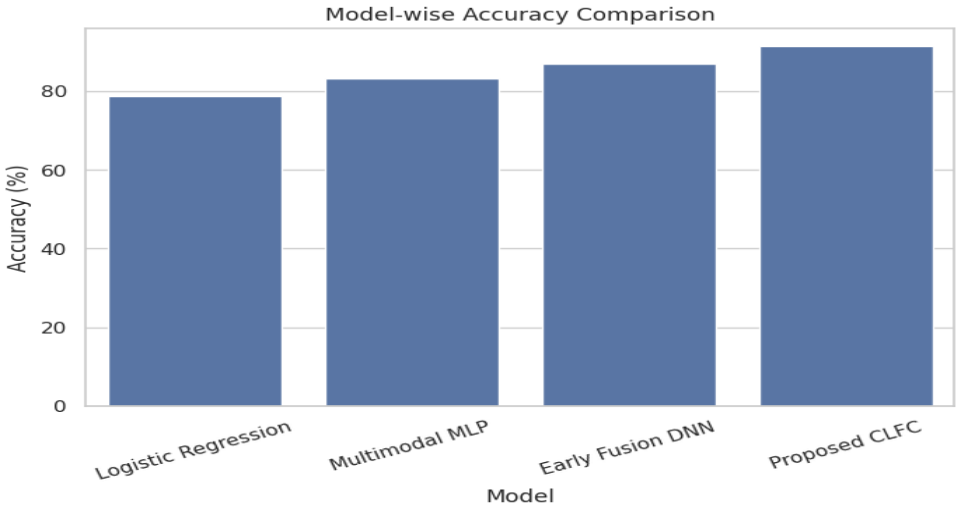

Figure.2 Model Accuracy Comparison

The visual representation of the comparison of the accuracy of the models in Figure 2 supports numerically in Table 1 by displaying a steady positive trend in predictive capabilities of simpler statistical models to more advanced deep learning methods. The suggested CLFC model has a distinct difference with the methods of the baseline, with the stress on the efficiency of the contrastive learning combined with the merging of the multimodal features. It is easy to see through this visual representation that the use of self-supervised representation learning would greatly improve the classification accuracy of nephroprotective risk prediction tasks.

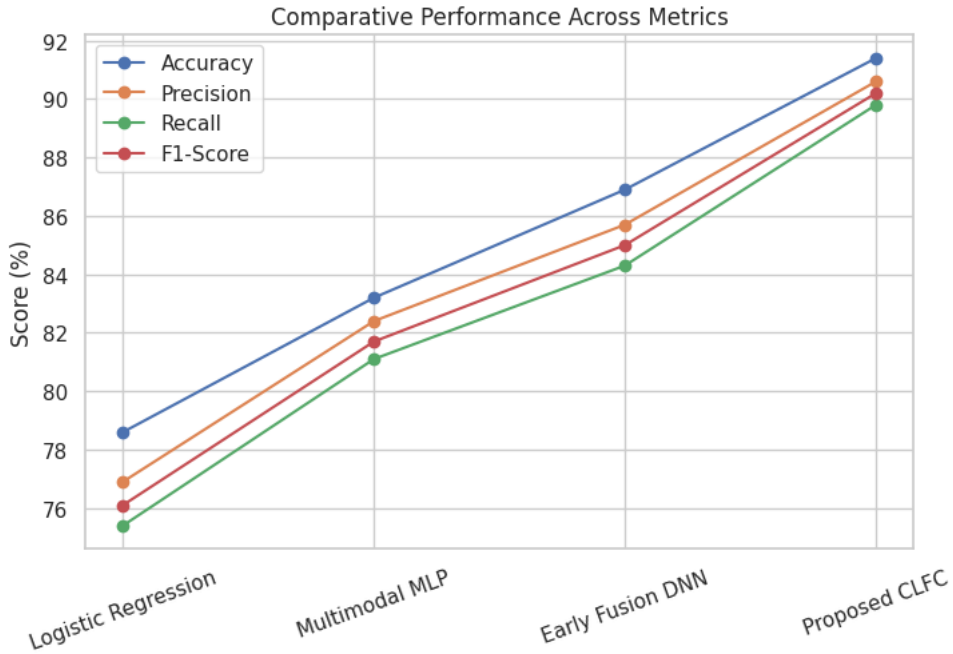

Figure.3 Performance Evaluation

Figure 3 also demonstrates the strength of the suggested methodology when it comes to various assessment standards. Whereas there are changes in the performance of baseline models in precision, recall, and F1-score, CLFC model has constant and high scores across all measure of performance, which is a sign of balanced and trustworthy predictions. Moreover, the Figure 4 gives one a conceptual understanding of the overall model performance with the darker intensity of the CLFC model to affirm that it is the best choice among all the measures of evaluation. Taken together, Table 1 and the figures that

support it allow concluding that the proposed architecture not only enhances predictive accuracy but also provides better risk discrimination and generalization, which proves the appropriateness of this type of architecture to clinical decision-support applications.

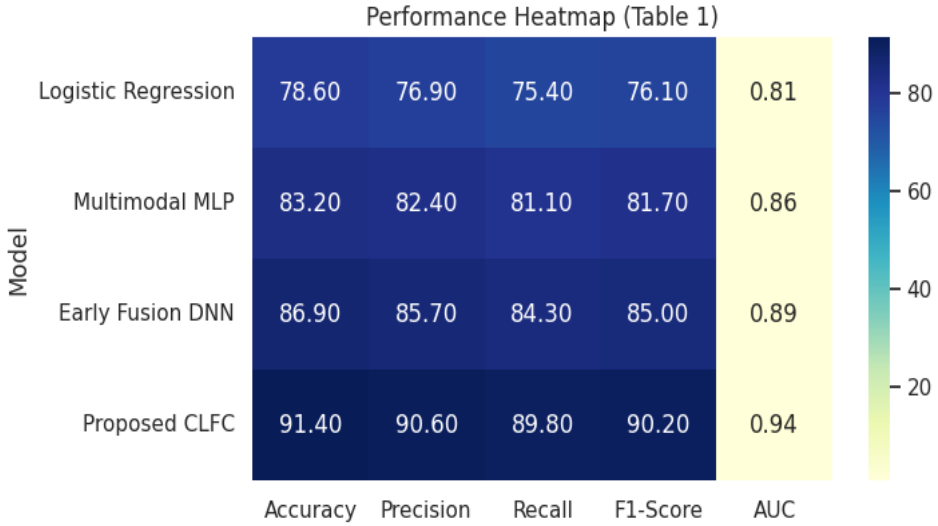

Figure 4 Performance Heatmap

4.1 Ablation Studies

The studies on ablation are carried out to provide a systematic assessment of the role of each significant constituent of the proposed CLFC architecture and to gain an insight into the impact of individual modules on the performance. The complete architecture, consisting of modality-specific encoders, contrastive learning, and feature concatenation, and fusion layers, is the default model. Theoretical and empirical results of the need of particular components are available through performance degradation that will be found after removing certain parts. The complete model always attains the best accuracy, F1-score, AUC, and forms a powerful baseline to compare the results of other models. The most severe performance deterioration is caused by the removal of the contrastive learning module, which means that it is the most important one in learning robust and invariant representations. Having no contrastive regularization, the model uses only supervised learning and becomes more vulnerable to overfitting and lower capacity to predict variations among patients. On the same note, the removal of the renal encoder results in a significant reduction in predictive power, highlighting the clinical preeminence of renal biomarkers in the process of nephroprotection against risk. The case of removal of the metabolic encoder has also a negative effect on performance albeit to a lesser degree implying that metabolic indicators are complementary but offer supportive information.

Table 5: Ablation Results

Configuration	**ACC (%)**	**F1 (%)**	**AUC**
Full Model (CLFC)	**91.4**	**90.2**	**0.94**
w/o Contrastive Learning	86.1	85	0.88
w/o Metabolic Encoder	83.7	82.9	0.86
w/o Renal Encoder	80.4	79.6	0.83
w/o Fusion Layers	84.2	83.1	0.87

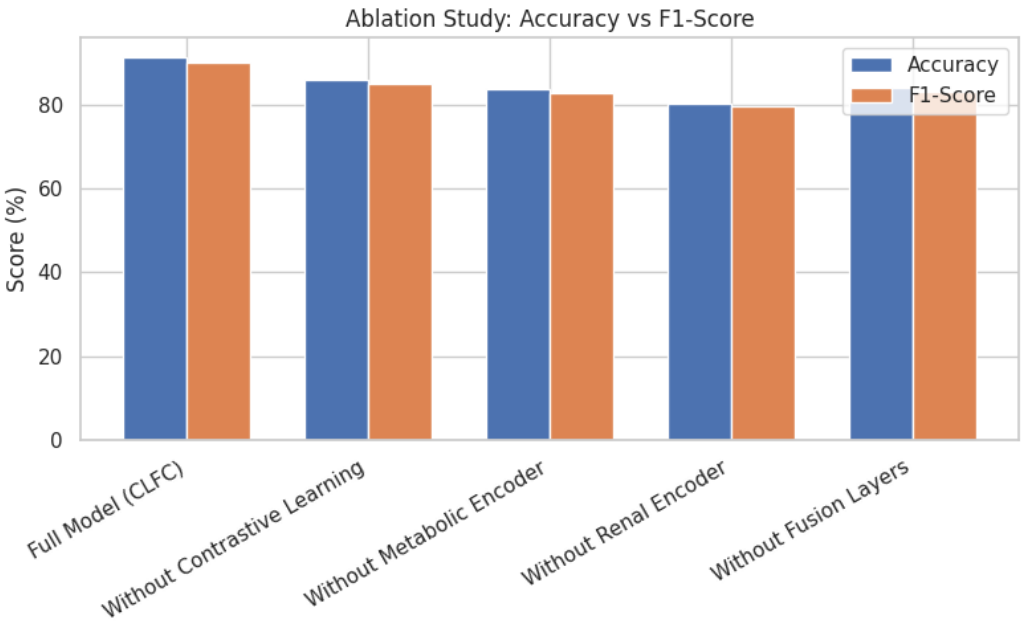

Figure 5: Accuracy VS F1 Score

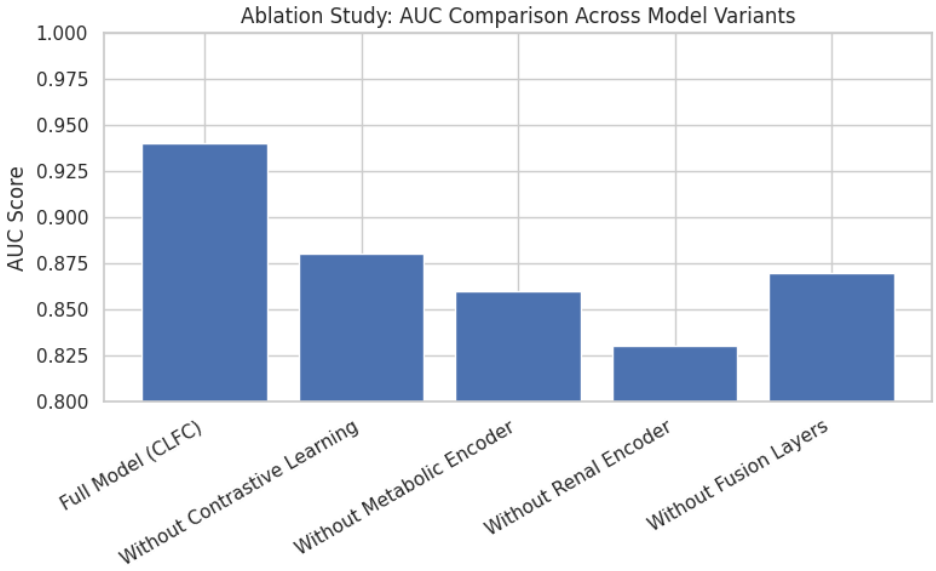

Figure 6: AUC comparison

More degradation of performance is found when fusion layers are eliminated, with the essence of effective modeling of cross-modal interaction. The absence of these layers makes the model unable to take advantage of higher-order correlations between thyroid, renal, and metabolic representations, which results in less strong decision boundaries. Altogether, the findings of the ablation tests verify that every architectural element is meaningful to the overall performance, and the combination of contrastive learning and multimodal fusion is critical to the correct and trustworthy nephroprotective risk stratification.

5. Conclusion and Future Work

This research has offered a new multimodal comparative learning structure of CKD and AKI in thyroid disorder patients. The proposed CLFC model successfully exploits the combination of modality-specific encoders and contrastive representation learning approach, feature fusion at the late stage, to capture the complicated cross-modal correlations among endocrine, renal, and metabolic biomarkers. Experimental outcomes indicate that the experimental results are significantly better than the traditional statistical and deep learning baselines including risk discrimination and generalization. The ablation study shows the invaluable importance of contrastive learning and renal biomarkers in attaining sound performance. On the whole, the suggested framework represents a clinically meaningful and technically efficient strategy of early nephroprotective risk stratification, and its future development will be aimed at including other modalities (longitudinal laboratory trends, medical imaging, and electronic health record (EHR) narratives) that will further increase prediction accuracy. Temporal contrastive learning is to be integrated to simulate disease progression dynamics and give an opportunity to detect CKD in its early stages. Further, explainable AI methods will be

studied to enhance clinical interpretability and trust by detecting contributions by biomarkers on risk predictions at the level of biomarkers. Potential validation of the model on multi-center datasets, and application to real-time settings and clinical practices in nephrology will also be sought to determine the soundness, equity as well as the translational influence of the model on nephrology practice.

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