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Original research

Plasma Homocysteine levels and cardiovascular risk Profiles in Adults with cardiovascular Disease: A retrospective observational study in Morocco

Kholoud Krimi[1,2] Dounia El Moujtahide [1,2] El-houcine Sebbar [1,2] Mohammed Choukri [1,2]

[1] *Central Laboratory, Mohammed VI University Hospital, Oujda, Morocco*|[2] *Faculty of Medicine and Pharmacy, Mohammed I University, Oujda, Morocco*

Correspondence should be addressed to Kholoud Krimi; kholoudkrimi2020@gmail.com

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Vitamin B deficiency.

ABSTRACT

Homocysteine may contribute to vascular injury and cardiovascular disease. This study described plasma homocysteine concentrations in 52 adults with cardiovascular or thromboembolic diseases at Mohammed VI University Hospital of Oujda, Morocco. Patients underwent homocysteine testing between January 2019 and July 2024. Clinical and laboratory data were analyzed descriptively according to age, sex, cardiovascular risk factors, vitamin status, and diagnosis. Mean age was 51.34 years; 28 patients were men. Mean homocysteine concentrations increased with age, from 5.5 µmol/L in patients younger than 20 years to 20.40 µmol/L in those older than 60 years. Concentrations were higher in women than men (16.87 vs 14.98 µmol/L) and highest in patients with hypertension (24.85 µmol/L). Vitamin B9 and B12 deficiencies were associated with slightly higher concentrations. These findings suggest age- and risk-factor-related variation; however, the descriptive design and lack of a control group preclude causal conclusions.

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1. Introduction

Cardiovascular diseases are among the leading causes of morbidity and mortality worldwide. According to the World Health Organization (WHO), they account for approximately 17.5 million deaths, representing nearly 30% of all global deaths (World Health Organization, 2025). Atherosclerosis plays a central role in the pathophysiology of these disorders (Paul & Baudin, 2009). It is a multifactorial process promoted by several well-established cardiovascular risk factors, including smoking, hypercholesterolaemia, arterial hypertension, diabetes mellitus, abdominal obesity and psychosocial factors.

Although these risk factors are now clearly identified, epidemiological data accumulated over the past three decades suggest the possible involvement of other biological markers in the increase of cardiovascular risk. Among these markers is homocysteine (Hcy), an amino acid discovered in 1932 by Butz, Lewis and Du Vigneaud (Ross, 1999).

Recent studies have shown that homocysteine may be involved in several mechanisms underlying vascular damage, including endothelial dysfunction, oxidative stress, inflammation, cellular proliferation and thrombosis (Tucker et al, 1996 ; Selhub et al, 1993) As early as 1969, McCully was the first to suggest that an abnormality in homocysteine metabolism could be involved in the occurrence of vascular lesions (Zerrouk, 2023).

The contribution of homocysteine to the development of cardiovascular diseases remains widely debated in the literature, particularly through numerous epidemiological and interventional studies. In this context, we conducted a retrospective, descriptive, observational study at Mohammed VI University Hospital in Oujda, Morocco.

The primary objective was to describe the distribution of plasma total homocysteine (tHcy) concentrations among patients with cardiovascular and thromboembolic diseases. We also aimed to describe variations in tHcy concentrations according to age, sex, cardiovascular risk factors, vitamin B9 and B12 status, and clinical diagnoses.

2. Materials and methods

2.1. Study Design and Setting

We conducted a retrospective, descriptive, observational study involving 52 adult patients who underwent plasma homocysteine measurement as part of the assessment of cardiovascular diseases at the Biochemistry Department of Mohammed VI University Hospital in Oujda, Morocco. The study covered a period of five years and seven months, from January 2019 to July 2024.

2.2. Study Population and Eligibility Criteria

Patients were included if they were adults, had undergone homocysteine measurement as part of their medical assessment, and had a confirmed diagnosis of one or more cardiovascular diseases.

Patients who underwent homocysteine measurement for non cardiovascular conditions, as well as those with unusable medical records, were excluded. Patients with conditions likely to influence plasma homocysteine concentrations, particularly severe chronic kidney disease, liver failure, hypothyroidism, or cancer, were also excluded.

2.3.Biochemical Measurements

Plasma total homocysteine (tHcy) concentrations were measured using an ARCHITECT automated immunoassay system (Abbott Diagnostics), based on chemiluminescent microparticle immunoassay (CMIA) technology. Results were expressed in µmol/L.Vitamin B9 and B12 concentrations were obtained from available laboratory records.

2.4. Data Collection

Patient data were collected from paper-based medical records and electronic records available through the HOSIX hospital information system. Relevant information was extracted using a pre-established data collection form.

2.5.Statistical Analysis

Descriptive statistical analyses were performed using Microsoft Excel. The collected data were analysed descriptively to characterise the study population and describe the distribution of plasma tHcy concentrations.

2.6. Ethical Considerations

This retrospective observational study was conducted at Mohammed VI University Hospital in Oujda, Morocco. The study protocol was reviewed and approved by the relevant institutional ethics committee. All procedures followed applicable ethical principles and institutional regulations.

3. Results

3.1. Demographic Characteristics of the Patients

The study included 52 patients aged between 18 and 71 years, with a mean age of 51.34 years. The 40–59-year age group was the most represented (34 patients; 65.38%), followed by the oldest age group (11 patients; 21.15%) and patients aged 18–39 years (7 patients; 13.46%). The study population comprised 28 men (53.85%) and 24 women (46.15%), with a male-to-female ratio of 1.17. The main cardiovascular risk factors and comorbidities were type 2 diabetes mellitus, obesity or overweight, chronic smoking, arterial hypertension, dyslipidaemia, menopause, and a family history of cardiovascular disease. Type 2 diabetes mellitus was the most frequent comorbidity, affecting 23 patients (44.23%), including 10 patients with hyperhomocysteinaemia. Obesity or overweight was observed in 22 patients (42.31%), including 6 with hyperhomocysteinaemia. Chronic smoking was reported in 16 patients (30.77%), including 7 with hyperhomocysteinaemia.

Arterial hypertension affected 12 patients (23.08%), including 8 with hyperhomocysteinaemia, while dyslipidaemia was documented in 8 patients (15.38%), including 4 with hyperhomocysteinaemia.

Among the 24 women, 14 (58.33%) were postmenopausal. A family history of cardiovascular disease was reported in 8 patients (15.38%).

3.2. Plasma Total Homocysteine Concentrations

According to age

Mean plasma total homocysteine (tHcy) concentrations varied across age groups. After combining the two youngest age categories, the calculated mean concentration was approximately 12.70 µmol/L among patients aged 18–39 years (n = 7). The reported mean concentrations were 15.03 µmol/L among patients aged 40–59 years (n = 34) and 20.40 µmol/L in the oldest age group (n = 11). These findings showed a descriptive increase in mean tHcy concentrations across the age categories.

2.5. According to sex

The reported mean plasma tHcy concentration was slightly higher in women than in men (16.87 vs. 14.98 µmol/L).

2.6. According to cardiovascular risk factors

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4. Discussion

At the national level, a study conducted in the Cardiology A Department of Ibn Sina University Hospital in Rabat investigated the relationship between plasma homocysteine concentrations, genetic polymorphisms of enzymes involved in homocysteine metabolism, and angiographically confirmed coronary artery disease. In that study, patients were aged between 24 and 73 years, with a mean age of 52 years (Laraqui et al., 2007).

In Tunisia, a study entitled "Variation of homocysteinaemia during venous thrombosis: a study of 1045 cases of thrombosis", conducted between 2000 and 2017, included patients with a mean age of 56.3 years and an age range of 17–94 years. This study investigated homocysteine concentrations in patients with venous thrombosis (Khedher et al., 2018).

In Togo, a study entitled "Homocysteinaemia and ischaemic stroke at the Campus University Hospital of Lomé" reported a mean age of 61.5 years, with an age range of 31–85 years. The study examined homocysteine concentrations in patients with ischaemic stroke (Grunitzky et al., 2008).

A study entitled "Hyperhomocysteinaemia: An Independent Risk Factor for Vascular Disease", published in 1991, investigated the relationship between hyperhomocysteinaemia and vascular disease (Clarke et al., 1991).

Another study, entitled "A Quantitative Assessment of Plasma Homocysteine as a Risk Factor for Vascular Disease", examined the relationship between plasma homocysteine concentrations and vascular disease, as well as the potential implications of folic acid intake (Boushey et al., 1995).

In our series, the mean age was 51.34 years, which was relatively close to that reported in several previous studies. Nevertheless, differences in inclusion criteria, clinical characteristics, and study populations limit direct comparisons.

In our study, a slight male predominance was observed. Among the 52 patients included, 28 were men (53.85%) and 24 were women

The highest reported mean plasma tHcy concentration was observed in patients with arterial hypertension (24.85 µmol/L), followed by those with obesity or overweight (20.18 µmol/L), dyslipidaemia (19.49 µmol/L), diabetes mellitus (18.96 µmol/L), postmenopause (18.79 µmol/L), and chronic smoking (16.96 µmol/L). Patients without documented cardiovascular risk factors had a reported mean tHcy concentration of 15.36 µmol/L. These differences were interpreted descriptively, without establishing statistical significance.

2.7. According to vitamin status

Among the 34 patients reported as having undergone vitamin B9 testing, 26 had normal vitamin B9 levels, with a reported mean plasma tHcy concentration of 12.34 µmol/L, whereas 8 patients had vitamin B9 deficiency, with a reported mean concentration of 14.09 µmol/L.

Similarly, among the 34 patients reported as having undergone vitamin B12 testing, 26 had normal vitamin B12 levels, with a reported mean tHcy concentration of 12.39 µmol/L, compared with 13.86 µmol/L in the 8 patients with vitamin B12 deficiency.

Higher mean tHcy concentrations were reported in patients with vitamin B9 or B12 deficiency than in those with normal vitamin levels. These differences were descriptive and were not evaluated for statistical significance.

2.8. According to cardiovascular diagnoses

Myocardial infarction was the most frequently reported cardiovascular diagnosis (16 patients; 30.77%), followed by stroke (14 patients; 26.92%), deep vein thrombosis (12 patients; 23.08%), and pulmonary embolism (10 patients; 19.23%). The four diagnostic categories accounted for the 52 patients included in the study (Fig 1).

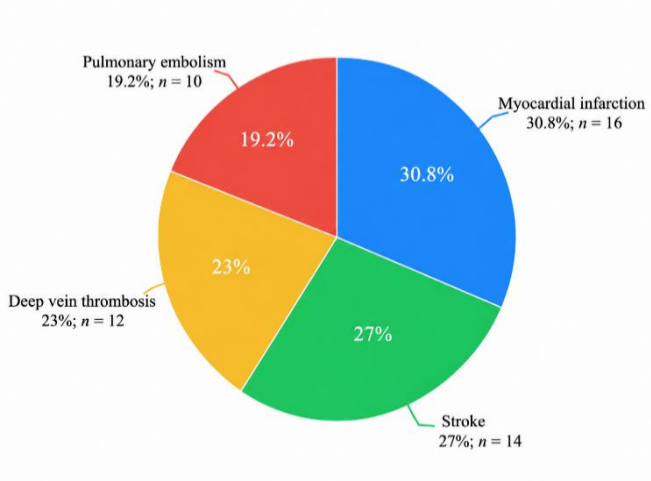


Figure 1: Distribution of cardiovascular diseases and mean homocysteine concentration

(46.15%). This distribution describes the composition of our hospital-based sample and does not establish a higher population-level prevalence of cardiovascular disease among men.

A prospective Chinese study conducted by Zhang et al. (Zhang et al., 2021) reported hyperhomocysteinaemia in 61.85% of hypertensive participants, suggesting a potential relationship between arterial hypertension and elevated homocysteine concentrations. Similarly, another Chinese study by Wang et al. (Wang et al., 2014), entitled "Homocysteine as a risk factor for hypertension", reported hyperhomocysteinaemia in 22.9% of hypertensive patients.

In our series, 8 of the 12 hypertensive patients (66.67%) had hyperhomocysteinaemia. Although this proportion can be compared descriptively with previous findings, differences in population characteristics, sample sizes, and diagnostic thresholds should be considered.

Regarding type 2 diabetes mellitus, 10 of the 23 diabetic patients in our series had hyperhomocysteinaemia, corresponding to 43.48%. A Moroccan study entitled "Hyperhomocysteinaemia: prevalence in a population of diabetic subjects", conducted at the Military Hospital of Marrakech (Belkhair et al., 2019), reported a prevalence of 38.8% among diabetic patients. Buysschaert et al. (Buysschaert et al., 2000) in Belgium and Goldstein et al. (Goldstein et al., 2004) reported prevalences of 31% and 32%, respectively. Higher prevalences were reported by Shaikh et al. (Shaikh et al., 2012) in Pakistan and Otmane et al. (Otmane et al., 2007) in Algeria, at 60% and 53%, respectively, whereas other studies reported lower percentages (Emoto et al., 2001; Chico et al., 1998). Such variations may reflect differences in clinical characteristics, sampling methods, and definitions of hyperhomocysteinaemia.

Regarding dyslipidaemia, a cross-sectional study conducted in China by Momin et al. (Momin et al., 2017) examined the relationship between homocysteine concentrations and lipid profiles and reported hyperhomocysteinaemia in 24.4% of dyslipidaemic patients. In our series, 4 of the 8 patients with dyslipidaemia (50%) had hyperhomocysteinaemia. Given the small number of patients, this finding should be interpreted cautiously.

Regarding obesity or overweight, 6 of the 22 affected patients in our series had hyperhomocysteinaemia (27.27%). This proportion was relatively close to the 23.3% reported by Elguedri et al. (Elguedri et al., 2014). Regarding chronic smoking, 7 of the 16 smokers in our sample had hyperhomocysteinaemia (43.75%). A study involving young adults with coronary artery disease (Sun et al., 2021) reported a prevalence of 39.1% among active smokers. These comparisons remain descriptive and cannot demonstrate a causal relationship between smoking, obesity, and increased homocysteine concentrations.

Regarding age, a Togolese study reported mean homocysteine concentrations of 17.41 µmol/L in patients younger than 45 years, 20.45 µmol/L in those aged 45–60 years, and 18.77 µmol/L in patients older than 60 years (Grunitzky et al., 2008). Similarly, Clarke et al. (Clarke et al., 1991) described variations in homocysteine concentrations across age categories. In our study, the calculated mean concentration was approximately 12.70 µmol/L among patients aged 18–39 years, while the reported means were 15.03 µmol/L among those aged 40–59 years and 20.40 µmol/L in the oldest age group. These findings indicate a descriptive increase in mean homocysteine concentrations across age categories. However, no statistical trend analysis was performed.

Regarding sex, Clarke et al. (Clarke et al., 1991) reported higher homocysteine concentrations in men than in women, while the Togolese study reported mean values of 20.66 µmol/L in men and 17.15 µmol/L in women (Grunitzky et al., 2008). In contrast, our cohort showed slightly higher mean concentrations in women than in men (16.87 vs. 14.98 µmol/L). This difference may reflect variations in age distribution, hormonal status, comorbidities, or sample composition, although these possible explanations were not specifically investigated.

Regarding cardiovascular risk factors, hypertensive patients in a Togolese study had a mean homocysteine concentration of 19.31 µmol/L, close to that of non-hypertensive patients (Grunitzky et al., 2008). Wang et al. (Wang et al., 2014) reported mean concentrations of approximately 23.4 µmol/L in hypertensive men and 16.6 µmol/L in hypertensive women. Mean concentrations of 22.33 µmol/L in hypertensive patients and 12.59 µmol/L in non-hypertensive individuals have also been attributed to Boushey et al. (Boushey et al., 1995). In our study, the reported mean homocysteine concentration among hypertensive patients was 24.85 µmol/L. However, this observation does not establish a statistically significant association or demonstrate that homocysteine is an independent cardiovascular risk factor.

In patients with type 2 diabetes mellitus, Buysschaert et al. (Buysschaert et al., 2000) reported a mean homocysteine concentration of 15.5 µmol/L and examined its relationship with macroangiopathy and nephropathy. Shaikh et al. (Shaikh et al., 2012) reported 15.22 µmol/L, while Emoto et al. (Emoto et al., 2001) reported 16 µmol/L. In our diabetic patients, the reported mean concentration was 18.96 µmol/L. This finding allows a descriptive comparison with previous studies but does not establish an independent relationship between diabetes, homocysteine concentrations, and cardiovascular risk.

Among smokers, Sun et al. (Sun et al., 2021) reported a mean concentration of 18.7 µmol/L, compared with 16.96 µmol/L in our series. In patients with dyslipidaemia, Momin et al. (Momin et al., 2017) reported a mean concentration of 11.98 µmol/L, compared with 19.49 µmol/L in our sample. El Guedri et al. reported a mean concentration of 15.60 µmol/L in obese patients, whereas the corresponding value in our patients with obesity or overweight was 20.18 µmol/L (Elguedri et al., 2014). Differences in population characteristics, comorbidities, and measurement conditions should be considered when interpreting these comparisons.

Regarding menopause, Zhu et al. reported hyperhomocysteinaemia prevalences of 43% in premenopausal women, 26.4% during the menopausal transition, and 45% in postmenopausal women, with a mean concentration of 17.6 µmol/L after menopause. In our sample, the reported mean concentration among postmenopausal women was 18.79 µmol/L. However, the small sample size and descriptive design do not allow conclusions regarding an independent effect of menopausal status on homocysteine concentrations.

Regarding vitamin B9, Selhub et al. (Selhub et al., 1999) reported higher homocysteine concentrations among individuals with folate deficiency than among those with normal folate status. The relationship between vitamin deficiency and homocysteine concentrations has also been discussed by Stabler et al. (Stabler et al., 2013). Studies investigating homocysteine lowering through folic acid supplementation have also been reported (Homocysteine Lowering Trialists' Collaboration, 1998). In our series, the reported mean tHcy concentration was 14.09 µmol/L among patients with vitamin B9 deficiency and 12.34 µmol/L among those with normal vitamin B9 levels. This difference was descriptive and was not evaluated for statistical significance.

Regarding vitamin B12, Selhub et al. (Selhub et al., 1999) described variations in homocysteine concentrations according to vitamin status. Hvas and Nexo (Hvas & Nexo, 2006) and Stabler et al. (Stabler et al., 2013) discussed biochemical markers used to evaluate vitamin B12 deficiency.

Alterations in homocysteine metabolism in the context of cobalamin deficiency have also been described (Allen et al., 1993). In our series, the reported mean tHcy concentration was 13.86 µmol/L among patients with vitamin B12 deficiency and 12.39 µmol/L among those with normal vitamin B12 levels. These findings should be interpreted cautiously because vitamin measurements were unavailable for some patients and the group means have not yet been reconfirmed following correction of the subgroup counts.

Finally, previous research has investigated the relationship between elevated homocysteine concentrations and arterial cardiovascular diseases and venous thromboembolic disorders, including deep vein thrombosis and pulmonary embolism (den Heijer et al., 1996). In our sample, myocardial infarction was the most frequently recorded diagnosis, followed by stroke, deep vein thrombosis, and pulmonary embolism. However, because all included patients had cardiovascular or thromboembolic conditions and no control group was available, our study cannot establish an association between plasma homocysteine concentrations and the occurrence of these diseases.

Study Limitations

This study has several limitations. First, its retrospective, single-center design and relatively small sample size (n = 52) limit the

generalizability of the findings. Second, the absence of a control group prevents the assessment of cardiovascular disease risk associated with plasma total homocysteine concentrations. Third, homocysteine measurements were performed according to clinical indications rather than through systematic population-based screening, potentially introducing selection and indication bias.
Vitamin B9 and B12 measurements were not available for all patients, limiting the interpretation of the findings related to vitamin status. The inclusion of patients with both arterial cardiovascular diseases and venous thromboembolic disorders also introduced clinical heterogeneity. Potential confounding factors, including medication use and nutritional status, could not be fully assessed using the available information.
Finally, the analysis was limited to descriptive statistics, without inferential testing or adjustment for potential confounding variables. Consequently, differences observed between patient subgroups should not be interpreted as statistically significant or causal associations. Further studies involving larger samples, appropriate control groups, and more comprehensive statistical analyses are needed.

5. Conclusion

This retrospective, descriptive study provides an overview of plasma total homocysteine concentrations among 52 patients with cardiovascular or thromboembolic diseases in a single Moroccan hospital. Variations in homocysteine concentrations were observed across age groups, sex, cardiovascular risk factors, and vitamin B9 and B12 status.
However, the small sample size, absence of a control group, and descriptive nature of the analysis prevent conclusions regarding the independent role of hyperhomocysteinaemia in cardiovascular disease. Further studies involving larger populations, appropriate control groups, and comprehensive statistical analyses are needed to clarify the clinical relevance of plasma homocysteine measurements in patients with cardiovascular and thromboembolic diseases.

Declarations

Ethics approval and consent to participate

The protocol involving humans was also approved by the Ethical Review Board of Faculty of Medicine and Pharmacy, Mohammed I University in accordance with the Declaration of Helsinki for medical research involving human subjects. All methods were performed in accordance with the relevant guidelines and regulations outlined in the Declaration of Helsinki for medical research involving human subjects. Informed consent to participate in the study was obtained from participants or , if participants are under 18, from a parent and/or legal guardian

Competing interests

The authors declare that they have no competing interests.

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Author's Contribution

Kholoud Krimi contributed to study conceptualization, methodology, data collection, analysis, and manuscript drafting. Dounia El Moujtahide participated in data collection, investigation, and manuscript editing. El-houcine Sebbar contributed to data interpretation, validation, and critical revision. Mohammed Choukri supervised the study, validated the findings, and critically revised the manuscript. All authors reviewed and approved the final manuscript.

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Data Availability

Data are available from the corresponding author upon reasonable request

References.

Grunitzky, E. K., Balogou, A. A. K., Barque-Barry, R., Kombate, D., & Amouzou, E. K. (2008). Homocystéinémie et accidents vasculaires cérébraux ischémiques au CHU Campus de Lomé. African Journal of Neurological Sciences, 27(2). Ref

Allen, R. H., Stabler, S. P., Savage, D. G., & Lindenbaum, J. (1993). Metabolic abnormalities in cobalamin (vitamin B12) and folate deficiency. The FASEB Journal, 7(14), 1344–1353. Crossref

Boushey, C. J., Beresford, S. A. A., Omenn, G. S., & Motulsky, A. G. (1995). A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: Probable benefits of increasing folic acid intakes. JAMA, 274(13), 1049–1057. Crossref

Buysschaert, M., Dramais, A. S., Wallemacq, P. E., & Hermans, M. P. (2000). Hyperhomocysteinemia in type 2 diabetes: Relationship to macroangiopathy, nephropathy, and insulin resistance. Diabetes Care, 23(12), 1816–1822. Crossref

Chico, A., Perez, A., Cordoba, A., Arcelus, R., Carreras, G., de Leiva, A., et al. (1998). Plasma homocysteine is related to albumin excretion rate in patients with diabetes mellitus: A new link between diabetic nephropathy and cardiovascular disease? Diabetologia, 41(6), 684–693. Crossref

Clarke, R., Daly, L., Robinson, K., Naughten, E., Cahalane, S., Fowler, B., et al. (1991). Hyperhomocysteinemia: An independent risk factor for vascular disease. New England Journal of Medicine, 324(17), 1149–1155. Crossref

den Heijer, M., Koster, T., Blom, H. J., Bos, G. M. J., Briet, E., Reitsma, P. H., et al. (1996). Hyperhomocysteinemia as a risk factor for deep-vein thrombosis. New England Journal of Medicine, 334(12), 759–762. Crossref

Elguedri, S., Ben Khalifa, F., Elamouri, B., Oueslati, I., Oueslati, S., Hachicha, H., et al. (2014). Metabolic syndrome and homocysteine. Annales d'Endocrinologie, 75(5–6), 466. Crossref

Emoto, M., Kanda, H., Shoji, T., Kawagishi, T., Komatsu, M., Mori, K., et al. (2001). Impact of insulin resistance and nephropathy on homocysteine in type 2 diabetes. Diabetes Care, 24(3), 533–538. Crossref

Goldstein, M., Leibovitch, I., Yeffimov, I., Gavendo, S., Sela, B. A., & Loewenstein, A. (2004). Hyperhomocysteinemia in patients with diabetes mellitus with and without diabetic retinopathy. Eye, 18(5), 460–465. Crossref

Homocysteine Lowering Trialists' Collaboration. (1998). Lowering blood homocysteine with folic acid-based supplements: Meta-analysis of randomised trials. BMJ, 316(7135), 894–898. Ref

Hvas, A. M., & Nexo, E. (2006). Diagnosis and treatment of vitamin B12 deficiency: An update. Haematologica, 91(11), 1506–1512. Ref

Khedher, M., Said, F., Hamzaoui, A., Khanfir, M., & Houman, M. H. (2018). Variation in homocysteinemia during venous thrombosis: A study of 1045 cases of thrombosis. La Revue de Médecine Interne, 39(Suppl. 1), A131. Crossref

Laraqui, A., Allami, A., Carrie, A., Coiffard, A. S., Benkouka, F., Benjouad, A., et al. (2007). Relation between plasma homocysteine, gene polymorphisms of homocysteine metabolism-related enzymes, and angiographically proven coronary artery disease. European Journal of Internal Medicine, 18(6), 474–483. Crossref

Belkhair, J., Sebbani, M., Lachgar, F., Baizri, H., Amine, M., & Chellak, S. (2019). L'hyperhomocystéinémie chez une population diabétique type 2 : prévalence et son association aux paramètres clinicobiologiques. *[Titre de la revue à confirmer], 13*(6), 556–560. Crossref

Momin, M., Jia, J., Fan, F., Li, J., Dou, J., Chen, D., et al. (2017). Relationship between plasma homocysteine level and lipid profiles

in a community-based Chinese population. Lipids in Health and Disease, 16, Article 54. Crossref

Otmane, A., Makrelouf, M., Bouchama, R., Boucelma, M., Berrah, A., & Zenati, A. (2007). Measurement of total homocysteine in a cohort of patients with type 2 diabetes. Revue Francophone des Laboratoires, 2007(396, Suppl. 1), 21–24. Crossref

Paul, J. L., & Baudin, B. (2009). Pathophysiology of atherosclerosis and early markers. Revue Francophone des Laboratoires, 2009(409), 41–50. Crossref

Ross, R. (1999). Atherosclerosis—An inflammatory disease. New England Journal of Medicine, 340(2), 115–126. Crossref

Selhub, J., Jacques, P. F., Rosenberg, I. H., Rogers, G., Bowman, B. A., Gunter, E. W., et al. (1999). Serum total homocysteine concentration in the third National Health and Nutrition Examination Survey, 1991–1994: Population reference ranges and contribution of vitamin status to high serum concentrations. Annals of Internal Medicine, 131(5), 331–339. Ref

Selhub, J., Jacques, P. F., Wilson, P. W., Rush, D., & Rosenberg, I. H. (1993). Vitamin status and intake as primary determinants of homocysteinemia in an elderly population. JAMA, 270(22), 2693–2698. Crossref

Shaikh, M. K., Devrajani, B. R., Shaikh, A., Shah, S. Z. A., Shaikh, S., & Singh, D. (2012). Plasma homocysteine level in patients with diabetes mellitus. World Applied Sciences Journal, 16(9), 1269–1273. Ref

Stabler, S. P., Korson, M., & Jethva, R. (2013). Metabolic profiling of total homocysteine and related compounds in hyperhomocysteinemia: Utility and limitations in diagnosing the cause of puzzling thrombophilia in a family. *JIMD Reports, 11*, 149–163. Crossref

Sun, J., Han, W., Wu, S., Jia, S., Yan, Z., Guo, Y., et al. (2021). Combined effect of hyperhomocysteinemia and smoking on the severity of coronary artery disease in young adults ≤35 years of age: A hospital-based observational study. BMC Cardiovascular Disorders, 21(1), Article 484. Crossref

Tucker, K. L., Selhub, J., Wilson, P. W., & Rosenberg, I. H. (1996). Dietary intake pattern relates to plasma folate and homocysteine concentrations in the Framingham Heart Study. The Journal of Nutrition, 126(12), 3025–3031. Crossref

Wang, Y., Chen, S., Yao, T., Li, D., Wang, Y., Li, Y., et al. (2014). Homocysteine as a risk factor for hypertension: A 2-year follow-up study. PLOS ONE, 9(10), Article e108223. Crossref

World Health Organization. (2025). Cardiovascular diseases (CVDs). WHO – Cardiovascular diseases Ref

Zerrouk, F. (2023). Impact of hyperhomocysteinemia on cardiovascular diseases in a murine model [Dissertation, Mouloud Mammeri University of Tizi Ouzou]. Ref

Zhang, Z. Y., Jiang, Y. Y., Wang, Z., Li, L., Shi, W., Ji, J. G., et al. (2021). Homocysteine, hypertension, and risks of cardiovascular events and all-cause death in the Chinese elderly population: A prospective study. Journal of Geriatric Cardiology, 18(10), 796–808. Ref