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A Review on the Association between Dyslipidemia, Glucose Tolerance, and Coronary Heart Disease Risk

Xueling He, Yanyan Zhang, Qingkai Yan*

Department of Cardiology, Bishan Hospital of Chongqing Medical University, Chongqing 402760, China

**Corresponding author:* Qingkai Yan, yangingkai@hotmail.com*

Abstract: As one of the main chronic diseases in modern society, coronary heart disease, as a major disease that affects people's lives and health, has the characteristics of hidden onset and sudden onset. Coronary heart disease has relatively clear risk factors. Among them, blood lipid levels and blood sugar levels, as two main risk factors, play an important role in promoting the onset of coronary heart disease. The two complement each other in a vicious cycle, synergize and promote each other, promote the process of coronary atherosclerosis, thereby causing coronary heart disease. Multiple components in blood lipids and poor management of long-term blood sugar levels play a major role in specific clinical problems. This article reviews the different components of blood lipids and the effects of hyperglycemia on coronary heart disease, and initially expounds the mechanism by which blood lipids and blood sugar levels synergize each other to aggravate the risk of coronary heart disease, and combines them with relevant clinical issues, in order to help clinicians guide the prevention of coronary heart disease in terms of blood lipids and blood sugar levels.

Keywords: Dyslipidemia; Coronary heart disease; Glucose tolerance; Risk

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1. Introduction

Coronary heart disease still tops the list of lethal conditions worldwide, and the bulk of these deaths root in risk factors that could, at least in theory, be altered. Among the most repeatedly verified culprits, dyslipidemia and impaired glucose tolerance stand out; they now occupy centre stage whenever clinicians reconstruct how CHD first takes hold. Dyslipidemia, a collective label for blood lipid levels that drift beyond the safe interval, accelerates atherosclerosis, the slow piling-up of material within arterial walls that eventually surfaces as clinical CHD. Impaired glucose tolerance, the metabolic phase that typically heralds type 2 diabetes, piles on cardiovascular danger even while fasting glucose stays short of the diabetic cut-off. The two disturbances frequently appear side by side and amplify one another, so teasing apart their interaction and sizing up their combined load on coronary vessels has turned into a prerequisite if prevention schemes and therapeutic routines are to stay precise and deliver results.

After entering modern society, people's lifestyles have undergone significant changes, and dyslipidemia has become a long-term metabolic disorder affecting many individuals, posing serious health risks. The impact of long-term dyslipidemia on the human body is like an 'invisible bomb.' If patients do not pay attention and fail to intervene in time, the consequences are often severe, and it is highly likely to trigger various serious cardiovascular diseases, among which coronary heart disease is the most common and harmful. So, how closely is dyslipidemia linked to coronary heart disease? Dyslipidemia usually manifests as elevated low-density lipoprotein (LDL) and triglycerides, along with decreased high-density lipoprotein (HDL)[1]. In the human body, the metabolic internal environment is extremely complex, and when dyslipidemia occurs alongside conditions like insulin resistance that may cause blood sugar disturbances, the risk factors for developing coronary heart disease can be amplified [2].

Abnormal blood sugar levels are also quite common in today's society. Prolonged high blood sugar levels can simultaneously worsen and disrupt lipid levels, and abnormal blood sugar can also act as an independent risk factor contributing to the development of coronary heart disease [3,4]. In more extreme cases, when a patient with type 2 diabetes experiences significant lipid metabolism disorders, they may develop coronary heart disease as a complication [5].

Dyslipidemia and impaired glucose tolerance often lock together in a self-reinforcing loop, and the pattern is hardest to miss in patients who already carry the metabolic syndrome label or live with type 2 diabetes. Several cohort studies have shown that once the syndrome—recognised by the familiar triad of lipid anomalies, rising blood pressure, and IGT—takes hold, the odds of later coronary heart disease rise far beyond the level faced by people who escape this bundle of metabolic oddities [6]. Because the separate pieces so rarely appear alone, front-line clinicians have learned that isolating a single target seldom moves the needle; the cumulative threat to coronary arteries only contracts when several strands are reined in side by side. Routine practice, therefore turns into a coordinated bundle: steady guidance on food, activity, and weight, agents that act on both lipid and glucose pathways, and follow-up visits spaced closely enough to watch how overall cardiovascular risk drifts over time, all treated as indispensable if the heart is to stay insulated from the overlapping stress these linked disorders impose [7].

When the three elements of dyslipidemia, impaired glucose tolerance, and coronary heart disease are placed side by side, their mutual influence quickly reveals a maze rather than a straight line, because each disorder keeps feeding the others through overlapping metabolic and vascular disruptions. Dyslipidemia or IGT alone already nudges CHD risk upward, yet the moment they share the same body they amplify one another, so clinicians have to knit prevention and daily care into one coherent plan instead of letting the two drift along separate rails. The next layer of work is to chart, with far higher resolution, the molecular and cellular roads that tether lipid chaos to glucose swings, and then to run pragmatic trials of interventions that can strike both routes at the same time, hoping to tilt the cardiovascular future of high-risk cohorts toward a clearly brighter curve.

2. Main body

2.1. Definition and classification of dyslipidemia

Dyslipidemia is the label clinicians give when the blood levels of fat-like substances—chiefly cholesterol and triglycerides—drift outside the interval laboratories regard as normal, and the same label applies when HDL cholesterol slips below the value cardiologists would like to see. Physicians usually begin by deciding which lipid component has wandered off course, and the first fork in the road separates primary from secondary varieties. Primary dyslipidemia, illustrated by disorders such as familial hypercholesterolemia, generally stems from germ-line DNA variants handed down through generations, while the secondary version follows in the wake of other illnesses—diabetes, adiposity, or a sluggish thyroid gland are typical culprits. After the main category is pinned down, the profile is sharpened into narrower subtypes: hypercholesterolemia when LDL or total cholesterol occupies centre stage, hypertriglyceridemia when triglycerides claim the spotlight, or an isolated HDL deficit when the good cholesterol alone is missing in action, allowing the therapeutic blueprint to focus on the exact pattern that is pushing cardiovascular risk upward and tangling the broader task of keeping the patient well [1].

2.1.1. Low-density lipoprotein cholesterol (LDL-C)

Low-density lipoprotein cholesterol, shortened in everyday notes to LDL-C, picked up the informal label "bad cholesterol" because it reliably appears at the exact spots where atherosclerotic plaques first take hold and where later cardiovascular problems quietly begin. When LDL-C lingers on the high side for years, the probability of coronary artery disease and a broader set of acute cardiac or cerebrovascular incidents rises in parallel. Because of this steady link, clinicians treat tight control of circulating LDL-C as one of the main handles for stopping atherosclerotic cardiovascular disease, or ASCVD, before it can gather speed. Present-day guidelines accordingly tell patients already classed as high-risk to push LDL-C under 70 mg/dL, a threshold that several large cohorts have tied to a visible fall in new cardiovascular events. Statins still serve as the routine first-line option for nudging LDL-C lower, yet daily experience shows that a noticeable minority of people—particularly those carrying familial hypercholesterolemia or those who cannot stomach statins—require additional help, most often from PCSK9 inhibitors or from ezetimibe, before the desired number finally slips into range [8]. Shifting to the laboratory bench, LDL-C can be measured in more than one way: the classic Friedewald equation continues to deliver an estimate by stripping HDL-C and a triglyceride-derived fraction away from total cholesterol, whereas direct homogeneous assays are now common and, in situations such as marked hypertriglyceridemia or non-fasting specimens, they usually hand back a more reliable figure [9].

2.1.2. High-density lipoprotein cholesterol (HDL-C)

High-density lipoprotein is widely labelled 'good cholesterol' by researchers, since it routinely lowers circulating triglycerides and ferries lipids back to the liver where they can be processed and cleared. This protection hinges on reverse cholesterol transport, a stepwise route that collects surplus cholesterol from peripheral tissues, shuttles it through plasma, and hands it to the liver for ultimate disposal in bile. Population studies have long linked higher HDL-C numbers with fewer heart attacks and strokes; yet fresh cohort data and Mendelian-randomisation papers now question the old assurance that simply raising HDL-C guarantees safety. A growing line of work contends that particle performance—its functionality—carries weight equal to, or greater than, the milligrams-per-decilitre figure. Chronic inflammation, oxidative pressure, or compositional remodelling can convert these supposedly anti-atherogenic spheres into dysfunctional ones that forfeit cholesterol-efflux power, turn pro-inflammatory, and erase the anticipated gain. Given these findings, many investigators hold that while HDL-C quantity still mirrors metabolic balance to some degree, markers such as cholesterol-efflux capacity or the HDL inflammatory index paint a more reliable portrait of personal cardiovascular risk [10]. Current guidelines avoid prescribing a fixed HDL-C threshold; they instead promote everyday habits—steady aerobic activity, swapping unsaturated fats for trans fats, light alcohol, and weight management—that usually lift HDL-C and, more critically, seem to revive

HDL performance [11].

2.1.3. Triglycerides (TG)

Triglycerides, often abbreviated as TG, serve the body both as a handy fuel depot and as one of the circulating lipid fractions, and once their plasma level drifts upward, it is usually read as a signal that overall health is slipping. Clinicians pay particular attention when these lipid molecules rise above the reference interval, because the shift becomes a clear cardiovascular warning, especially when low HDL-C and elevated LDL-C travel alongside, a trio that together is called atherogenic dyslipidemia. The upward drift of triglycerides rarely has a single cause; instead, it reflects a blend of everyday factors, with obesity, insulin resistance, and repeated heavy drinking heading the list. Holding the concentration within the desirable zone, therefore, matters above all for people who already carry a label of metabolic syndrome or diabetes, because values that stay high can trigger an episode of acute pancreatitis and, over the years, keep adding to long-term cardiovascular hazard. Current care paths open with lifestyle recalibration—tightening food choices and stepping up movement—and, if that is not enough, layer in medicines such as fibrates, omega-3 fatty acids, or newer agents that block triglyceride synthesis, all chosen to pull fasting TG under 150 mg/dL so that the linked threats recede. Laboratory checks spaced at regular intervals make sense for anyone with known cardiovascular disease or classified as high-risk, since the triglyceride figure also acts as a quick glimpse into broader lipid turnover and overall vascular health [12].

2.2. The relationship between dyslipidemia and coronary heart disease

Dyslipidemia, a catch-all label for any measurable departure of blood lipid values from their usual range, sits near the top of the list of modifiable culprits that quietly prepare the ground for coronary heart disease. The relationship between wayward lipids and CHD refuses to flatten into a single track; instead, several lipid sub-fractions join forces, amplifying hemorheological shifts and nudging the body's own clotting machinery off balance, with low-density lipoproteins leading the charge. When LDL climbs too high, the delicate inner coat of small arteries takes a direct hit, and the ordinary physiology of the intima begins to tilt toward dysfunction, inviting an eclectic mix of circulating elements to settle along the wall, a sequence that ends in stiffening and the slow build-up of atherosclerotic plaque. Those plaques, once established, act as an immediate pathological driver of severe cardiac events. Fragments can shear away, slip into the coronary stream and lodge further downstream, narrowing the lumen and deepening myocardial ischemia, a chain of events that lifts the risk of a catastrophic episode sharply upward. HDL-C, in contrast, earns its reputation as a cardioprotective player mainly by shepherging excess cholesterol out of the arterial wall, ferrying it through the reverse transport route, and finally delivering it to the liver for disposal. Clinicians who hope to ease the day-to-day weight of CHD still need to map the subtle tug-of-war between these opposing lipid camps and watch how the balance tips into tangible clinical endpoints, a step that underpins any prevention plan or treatment sequence worth implementing.

2.2.1. The impact of dyslipidemia on atherosclerosis

Dyslipidemia occupies the narrative core of atherosclerosis, since the instant LDL particles exceed what the arterial wall can clear, lipids begin to linger, fibrous debris mingles with immune cells that arrive as part of the inflammatory reaction, gradually knitting together the arterial plaque. Among the lipid cast, low-density lipoprotein sharpens this pathophysiological sequence most acutely: once it lodges beneath the endothelium it readily oxidises, delivers its cholesterol cargo to tissue macrophages, and powers a self-feeding inflammatory loop that chips away at ordinary endothelial behaviour. An expanding collection of studies now indicates that long-standing dyslipidemia sustains both oxidative stress and inflammatory signalling inside the plaque milieu, twin forces that hasten lesion enlargement and undermine its stability [13]. In parallel, the unending lipid surplus trims nitric-oxide availability, loosens endothelial junctions and renders the arterial wall more porous, alterations that circle back to enlarge the plaque and further scramble local lipid management. Viewed as a whole, the reciprocal dance among climbing lipid concentrations, lingering inflammation and advancing endothelial harm clarifies why reining in dyslipidemia still serves as a workable keystone for curbing atherosclerotic hazard and, in turn, for heading off the cardiovascular events that erupt once a plaque finally breaks.

2.2.2. The correlation between LDL-C and coronary heart disease risk

For decades, clinicians have taken it as given that the concentration of LDL-cholesterol moves in step with the probability of coronary heart disease, and the literature has grown so large that every fresh meta-analysis merely sharpens the same outline: when low-density lipoprotein particles accumulate beyond their usual range, cardiac risk climbs in near-lockstep. Keeping that excess in place year after year erodes the mechanical integrity of the intima lining the smaller arteries, and once that lining weakens, the path toward arteriosclerosis needs no further invitation. Geneticists have added a sharper angle, pooling dozens of cohorts to show that individuals whose DNA nudges LDL-C upward carry a noticeably higher lifetime incidence of manifest CHD [14]. The mirror image appears in trials where statins or the newer lipid-lowering agents push the same metric downward, producing fewer events such as myocardial infarction or stroke, a symmetry that has turned the lipid hypothesis into common currency across cardiology departments. Abnormal lipid profiles therefore serve a dual role: they flag future risk and, more importantly, feed the disease process itself. The newest data refine the picture further, revealing that the impact of low-density lipoprotein can be tempered or amplified by existing atherosclerotic plaque and by the broader lipid landscape, a reminder that therapy should address the whole pattern rather than a solitary number in patients who already face high baseline hazard [15].

2.2.3. The protective role of HDL-C and its mechanisms

High-density lipoprotein cholesterol, abbreviated HDL-C, has long been casually called the “good cholesterol” because epidemiologists repeatedly observe that higher circulating amounts seem to wrap the vasculature in a buffer against atherosclerosis and its downstream companion, coronary heart disease. The maneuver most textbooks highlight is reverse cholesterol transport: the lipoprotein scours peripheral tissues for cholesterol that has wandered out of place, ferries the cargo through the bloodstream, and unloads it in the liver for ultimate excretion, a sequence that quietly prevents lipid from stacking inside arterial walls. Yet this cleanup operation is only one entry on its résumé; HDL also donates anti-inflammatory signals, supplies antioxidant enzymes, and shields the endothelial lining from stress, talents that together widen its cardioprotective footprint [16]. Recent cohort data, however, reveal that the HDL-C tale contains more wrinkles than earlier imagined, suggesting that the functional calibre of each particle can outweigh the sheer concentration measured in plasma. When the lipoprotein shifts into a “dysfunctional” state and its cholesterol efflux capacity slackens, cardiovascular hazard may paradoxically rise, a twist that redirects attention toward how effectively HDL operates rather than how many milligrams drift in the circulation [17]. Charting the intricate molecular choreography that underlies these protective maneuvers, therefore, stays high on the research agenda, because any future therapy that hopes to restore or amplify HDL performance will first need the granular map of what to fix before clinical benefit can follow.

2.3. Definition and clinical significance of abnormal glucose tolerance

Impaired glucose tolerance, abbreviated AGT, sits one step shy of outright diabetes. In this state the body can no longer keep blood sugar within a safe corridor for years on end, so glucose lingers at levels higher than ideal, quietly setting the scene for heavier downstream damage, above all type 2 diabetes mellitus and a cluster of cardiovascular diseases. Clinicians normally uncover AGT with a routine oral glucose tolerance test: two hours after the sweet drink, a plasma glucose value that stops anywhere from 140 to 199 mg/dL earns the label of impaired glucose tolerance, while a result that edges past 200 mg/dL crosses the line into outright diabetes. Doctors watch this narrow band so closely because AGT behaves like an early flare rather than a harmless flicker; cohort data keep showing that people who land here face roughly double or triple the risk of marching on to full T2DM when their long-term outcomes are lined up against peers whose two-hour values never leave the normal lane [18]. The glucose trouble seldom travels alone. Blood pressure drifts upward, lipids tilt toward dyslipidemia, and extra weight settles around the waist in the pattern of central obesity; these linked shifts steadily raise the probability that coronary artery disease will follow [19].

2.3.1. Diagnostic criteria for type 2 diabetes

Over the past decades, the yardsticks for labeling a person with T2DM have drifted noticeably, and both the American Diabetes Association and the World Health Organization now issue guidance that keeps hammering home the same message: unless the diagnosis is spot-on, any chance of starting therapy while the disease is still in its earliest chapter slips away. The ADA presently treats any one of four laboratory signals as decisive proof: a fasting plasma glucose that sits at 126 mg/dL or climbs higher, a two-hour reading that reaches 200 mg/dL or more during a standard oral glucose tolerance test, a hemoglobin A1c figure that lands at 6.5 % or above, or—when unmistakable hyperglycemic symptoms accompany the visit—a random plasma glucose that touches 200 mg/dL or beyond [2]. These thresholds quietly nudge clinicians to cast a wider screening net, especially over groups already freighted with extra risk, among them people whose glucose tolerance has begun to wobble and who, if no one intervenes, frequently tip into full-blown diabetes with unsettling speed. Picking up T2DM even a few months earlier than usual unlocks room for lifestyle adjustments and, should they prove insufficient, for pharmacologic help, a tandem that can trim the probability of later harm—think myocardial infarction, stroke, or a slow slide toward renal failure—by a clear margin [20].

2.3.2. Impact of abnormal glucose tolerance on cardiovascular health

Researchers have already traced the connection between abnormal glucose tolerance and cardiovascular health in considerable detail, and a steady stream of studies keeps sounding the same alarm: once blood sugar drifts into the impaired glucose tolerance zone, the likelihood of coronary heart disease climbs sharply. The problem rarely arrives unaccompanied; hypertension, hyperhomocysteinemia, and dyslipidemia usually tag along, and the resulting mix of elevated lipids, raised blood pressure, and other metabolic disturbances multiplies the baseline danger. Lipid panels offer a clear snapshot of this shift: individuals who have crossed into the AGT range tend to present with higher fasting triglycerides and, in the same breath, lower protective HDL-cholesterol, a pairing that clinicians recognise as a slow but steady eroder of arterial integrity [21]. The risk is not confined to laboratory figures; cohort data show that people with AGT register markedly more myocardial infarctions and ischaemic strokes, a trend that underlines why a glucose tolerance test ought to sit routinely within any thorough cardiovascular risk assessment. On a more hopeful note, targeted measures—structured exercise plans, careful dietary tweaks, or glucose-lowering medication—can nudge tolerance back toward normal and, in the process, pare down a measurable slice of cardiac risk, implying that attentive control of AGT functions as a real, not merely theoretical, shield for the heart [22]. Spotting the condition promptly and responding without delay therefore emerges as a pivotal prevention move, above all in population segments already juggling a double load of metabolic and cardiovascular liabilities.

2.4. Interaction between glucose tolerance and dyslipidemia

When it comes to mechanism, the way dyslipidemia and erratic glucose values talk to each other has moved to the front of the current debate and carries weight both on the bench and at the bedside. Work to date shows that people who carry dyslipidemia almost always display measurable insulin resistance, a defect that then nudges fasting and post-prandial glucose further upward [1,2]. This two-way downward spiral, which also magnifies coronary risk, is most visible in individuals with marked obesity or with chronically low daily step counts. Metabolic chaos feeds itself, accelerating lipid-rich plaque growth within coronary trees and setting the stage for hard endpoints. For those who see patients every day, keeping an eye on metabolic read-outs and stepping in early—whether lipids are off or glucose tolerance is already wobbling—has become central to daily practice. More mapping of the molecular traffic between lipid and glucose circuits will clearly feed back into smarter molecules and into guidelines that are refreshed more often.

Insulin resistance quietly steers the entire derailment of lipid balance, and it does so by nudging liver cells away from their usual choreography; once the hepatic rhythm slips, the organ no longer finishes the daily task of sorting lipids with its former precision. Everyone who follows metabolic literature already accepts that insulin resistance sits at the hub of glucose intolerance, yet the same defect, while nudging glucose upward, also muffles the liver's talent for clearing low-density lipoprotein particles. When the liver's grip on lipid traffic loosens, triglycerides climb, high-density lipoprotein shrinks, and the twin stresses of hyperglycaemia and disordered lipids stir the clotting cascade inside the vessel wall, encouraging plaque to take root. The sequence makes it clear that dyslipidaemia can feed back into glucose intolerance, but the pivotal insult—insulin resistance—can just as easily push lipids further off course. A steady programme of everyday movement and deliberate lifestyle choices nudges both glucose and lipid figures back toward their usual corridors [23].

2.5. Prevention and intervention strategies

2.5.1. Lifestyle interventions

Day-to-day habits still give clinicians the clearest, least expensive line of defence against coronary heart disease and diabetes, particularly once fasting glucose edges into the impaired tolerance band. Study after study shows that when people edge their routines toward cardioprotective patterns, the likelihood of stepping into outright diabetes or landing in the emergency ward with a myocardial event shrinks in a visible way. The shield that research keeps pointing to is neither exotic nor new: it is a trio of ordinary moves—plate changes you can spot across the dinner table, minutes of movement you can tally on a watch face, and a final break with tobacco. One fresh meta-analysis, for instance, calculated that nudging meals toward a Mediterranean template—filling the weekly grocery list with intact grains, seasonal fruit, and pulses while treating meat as a side actor—restores lipids and fasting glucose well enough to trim coronary risk [24]. On the exercise ledger, accumulating a modest 150 min of brisk walking, cycling, or any activity that raises the pulse is repeatedly tied to fewer cardiac admissions and to welcome side benefits such as softer blood-pressure readings and a lipid panel that no longer alarms the laboratory [25]. When counsellors add behavioural glue—perhaps a step-count wager among friends or a simple paper log of meals—participants usually stay with the regimen long enough for genuine shifts in risk to emerge [11]. Collectively, these strands of evidence show why community-wide lifestyle programmes merit front-row status in any serious plan to ease the shared burden of diabetes and heart disease, and they also clarify why public-health messages keep returning to the same quiet plea: eat a little more thoughtfully, move a little more often, and let the cigarettes go.

2.5.2. Recent advances in pharmacotherapy

Drug treatment has moved forward in the past few years, and the advance has given clinicians a noticeably firmer hold on coronary heart disease as well as on diabetes, particularly for individuals whose glucose tolerance is already beginning to slide. The newer classes—sodium-glucose co-transporter-2 inhibitors and glucagon-like peptide-1 receptor agonists—no longer stop at glycaemic control; they also hand clinicians measurable cardiovascular benefits that older agents could not deliver. Real-world trials illustrate the point: when an SGLT2 inhibitor is added to background therapy, the combined rate of heart-failure hospitalisations and major adverse cardiac events in adults with type 2 diabetes drops [7]. GLP-1 receptor agonists manage a comparable dual task, easing glucose levels downward, trimming excess weight, and still leaving patients with cleaner vascular endpoints [26]. Because these molecules interfere with several metabolic steps at once, they erode the tight bundle of risk factors that usually nourish both CHD and diabetes. While this is happening, investigators are busy testing cocktails that park the newcomers next to familiar workhorses—statins, ACE inhibitors, or beta-blockers—to learn whether the mixture can drive event curves still lower [1]. Clinics, for their part, are experimenting with personalised plans that fold age, renal function, body-mass index and other comorbidities into the equation before the first tablet is dispensed, a shift that should keep regimens from sliding into one-size-fits-all territory [27]. Viewed together, the shifting landscape of pharmacotherapy still looks like one of the more promising avenues for keeping high-risk patients clear of cardiac catastrophes.

2.5.3. Importance of regular screening

Keeping up a routine of cardiovascular and diabetes checks is still among the handful of measures that can pick up brewing problems before they gather speed, particularly for people whose years, body mass, or inherited background already nudge them into a clearly higher-risk lane. A pattern that surfaces again and again in the literature is that adults who carry impaired glucose tolerance—abbreviated simply as IGT—face a noticeably steeper probability of sliding into outright type 2 diabetes and, in parallel, of meeting some manifestation of cardiovascular illness, so identifying them at the earliest moment is not merely useful but close to decisive [4]. The workhorse investigations most outpatient rooms reach for—an overnight fasting glucose sample and the somewhat lengthier oral glucose tolerance procedure—manage reasonably well at highlighting these borderline persons, allowing counselling, dietary shifts, or even an initial pharmaceutical step to begin before injury fans out. Moving past glucose, watching the familiar cardiovascular cast—blood pressure numbers, a standard lipid profile, and a swift height-and-weight stop to derive body mass index—turns out to be equally indispensable for anyone who has already lived through a coronary episode or walks around with an established label of coronary heart disease [3]. Once clinics, or the wider health networks that support them, put in place a coherent screening timetable instead of leaving encounters to chance-driven appointments, the benefit shows up in plain figures: doctors can start discussions on modest lifestyle edits or hand out preventive prescriptions months, sometimes years, earlier, and that early lead converts into a measurable drop in heart attacks, strokes, and other major adverse cardiovascular events as the calendar pages turn [28]. Professional guidelines drive the message home, recommending that every person who can check off either a first-degree family thread of cardiac disease or a constellation of textbook risk markers should turn up for these assessments on a tighter schedule, because the interval in which proactive care remains possible stays broader when surveillance is steady rather than sporadic [7]. Read side by side, the accumulated evidence offers a single pragmatic lesson: scheduled screening acts as the pivot on which worthwhile prevention turns, handing clinicians an opening to step in while the pathological process can still be reeled back and, by doing so, shifting the long-range prognosis toward a stretch of additional years free from complicated illness for those who stand closest to the fire.

3. Conclusion

Dyslipidemia and elevated glucose each push up coronary heart disease rates on their own, yet they also amplify one another inside the wider metabolic upset, piling up the danger in a joint fashion. The review first walks through the key clinical steps that mark how this illness begins and then expands. It offers fresh angles for bench scientists while urging front-line doctors to look harder at the overlap between disordered lipids and erratic glycemic readings.

Researchers who spend their days poring over lipid panels and fasting-glucose spreadsheets often remind newcomers that the published record on dyslipidemia and glucose intolerance resembles a patchwork quilt rather than a single seamless blanket. Dozens of papers draw a confident arrow between these twin metabolic glitches and coronary heart disease, yet the slope of that arrow tilts the moment you switch cohorts, so any clinic that wants to import the numbers into daily care has to keep one foot on the brake. A person's DNA, the ethnic story carried by grandparents, the small daily habits that never make it into a questionnaire, plus the air quality and neighbourhood income curve all steer how dyslipidemia or a blunted glucose spike will act once inside one particular body. With that mosaic in view, the coming studies could profit from slowing the camera down to single-human resolution, mapping how each tile fits, so that tomorrow's prevention recipe or drug sequence can be cut to exact measurements and, once applied, keep cardiovascular events low for good.

By threading endocrinology, cardiology, nutrition and neighbouring disciplines into the same fabric, we let the portrait of CHD thicken little by little, revealing a disorder that is nudged along by several intertwined influences rather than a single culprit. A habit of working across these boundaries lets teams assemble wider models that still keep an eye on how lipid shuttling, insulin signalling and the heart's moment-to-moment condition keep feeding back on one another. Once such living maps are in hand, they can serve as a day-to-day compass for sketching prevention bundles or therapy sequences that are trimmed to fit the exact risk fingerprint, daily routines and cultural habits that separate one population slice from another.

Researchers have sketched a fairly coherent outline of how dyslipidemia and impaired glucose tolerance jointly nudge coronary heart disease forward, yet a tangle of unanswered questions still dangles at the edges, so sustaining the investigative push remains every bit as urgent as it was at the outset. Tangible benefits will surface only if forthcoming studies keep refining preventive and therapeutic blueprints by quietly registering each person's idiosyncrasies while also scanning the broader population canvas on which those quirks appear. Once that delicate equilibrium is held, the clinical kit for intercepting—or at least tempering—CHD should gain a visibly keener edge, patients ought to face a lighter outlook, and the everyday burden this exceptionally prevalent condition imposes on hospitals, clinics, and households should ease a little.

Disclosure statement

The authors declare no conflict of interest.

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