Entitlement Eligibility Guideline

Ischemic Heart Disease

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ICD-11 **codes:** BA8Z; BA8Y; BA40-43, 4Z, BA50-52, BA5Y, BA5Z, BA6Z, BA82, BA85, BA86

VAC medical code: 00708 Ischemic heart disease, coronary artery disease, arteriosclerotic heart disease, angina, coronary thrombosis, myocardial infarction

Definition

Ischemic heart disease (IHD) is a condition in which the supply of blood and oxygen to the heart tissue is inadequate to meet demand. IHD exists on a spectrum ranging from stable angina to acute coronary syndromes, myocardial infarction, and sudden cardiac death. Common to all conditions across this spectrum is the concept of a supply-demand mismatch.

For the purposes of this entitlement eligibility guideline (EEG), the following conditions are included:

- ischemic heart disease (IHD)
- coronary artery disease (CAD)
- angina, including Prinzmetal angina and unstable angina
- non-ST elevation myocardial infarction (NSTEMI)
- ST elevation myocardial infarction (STEMI)
- myocardial infarction (MI)
- acute coronary syndrome (ACS)
- spontaneous coronary artery dissection (SCAD)
- ischemia with non-obstructive coronary artery disease (INOCA).

Diagnostic standard

A diagnosis from a qualified physician or nurse practitioner (within scope of practice) is required. Documentation should be as comprehensive as possible, including specialist and/or cardiology reports and accompanying cardiac investigations.

The diagnosis of IHD can be made with a high degree of confidence from history and physical examination, and additional investigations such as bloodwork

(including cardiac markers such as troponin), electrocardiogram (ECG), exercise stress testing, echocardiogram, and invasive coronary angiography.
Exercise stress testing is a widely used initial test for the diagnosis and estimation of risk and prognosis of IHD. The exercise stress test involves a 12-lead ECG before, during, and after observed exercise, which is usually performed on a treadmill. This test involves monitoring symptoms using ECG, heart rate, and arm blood pressure readings while sustaining a standardized incremental increase in workload while exercising.

An intravenous drug challenge known as stress myocardial perfusion imaging is used to diagnose IHD in individuals who are unable to exercise. Echocardiography may be completed which involves ultrasound examination of the heart to assess valve function, wall motion, blood flow, and pump efficiency.

Coronary arteriography, also called cardiac angiography or cardiac catheterization, involves the placement of a catheter into an artery in the groin or wrist and an injection of contrast dye to allow visualization of the blood vessels of the heart. Cardiac angiography is a valuable tool for diagnosing IHD and determining the best course of treatment such as medication, angioplasty, or bypass surgery.

Anatomy and physiology

In a healthy heart (Figure 1: Arterial supply of the heart), the coronary arteries branch off the aorta, which is the main artery of the body to supply oxygen-rich blood to different areas of the heart. Under normal conditions, the arteries allow the blood to flow unobstructed. The inner layer of the artery, called the endothelium, is normally smooth to help blood flow easily and prevent blood from sticking or clotting. The myocardium is the muscular middle layer of the heart which is responsible for the pumping action necessary to maintain circulation. An adequate supply of oxygen to the myocardium is dependent on both the oxygen-carrying capacity of the blood and sufficient blood flow through the coronary arteries.

Figure 1: Arterial supply of the heart

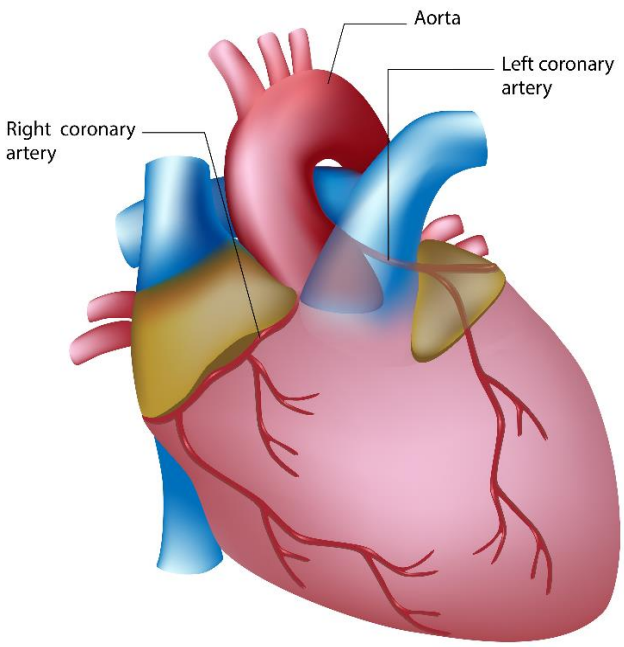

Illustration of a human heart highlighting the aorta—the main artery that carries oxygen-rich blood to the heart, supplying it with the oxygen and nutrients it needs to function. Branching from the aorta, the left and right coronary arteries. Source: Veterans Affairs Canada (2024).

Ischemia develops when the perfusion of oxygen to the myocardium cannot meet the demand for oxygen. The blood vessels supplying blood to the heart can become narrowed or blocked, referred to as atherosclerosis. Fatty deposits (plaque) build up in the coronary arteries that feed the heart tissue and this accumulation causes narrowing within the coronary arteries, reducing the flow of oxygen-rich blood to the heart.

Atherosclerosis results from an inflammatory and immunologically driven response of the arterial wall to multifactorial and repetitive injury. Complications of atherosclerosis result from progressive growth of plaques and secondary changes in the plaques that can lead to narrowing of the blood vessel (luminal stenosis), thrombosis, embolization, aneurysm formation, and vessel rupture. Progression of atherosclerosis can be influenced by a broad range of metabolic, immunological, and inflammatory factors.

Multiple risk factors are implicated in the development of atherosclerosis including ageing, dyslipidaemia, hypertension, cigarette smoking, and diabetes mellitus. An inflammatory cascade ensues that plays an important role in erosion or rupture of

vulnerable plaques leading to atherosclerosis complications which can progress slowly or rapidly depending on the presence of risk factors.

Angina develops when the heart tissue does not receive adequate perfusion of oxygen-rich blood. Angina is a type of chest pain or discomfort associated with IHD. While obstruction of the blood flow through the coronary arteries caused by atherosclerosis is the most common cause, symptoms of angina can occur without evidence of significant obstructive disease. Ischemia with non-obstructive coronary artery disease (INOCA) is a condition where a person experiences symptoms of reduced blood flow to the heart (ischemia), such as chest pain or discomfort (angina), without evidence of narrowing or blockage in the heart vessels (Figure 2: Causes of ischemic heart disease).

Figure 2: Causes of ischemic heart disease

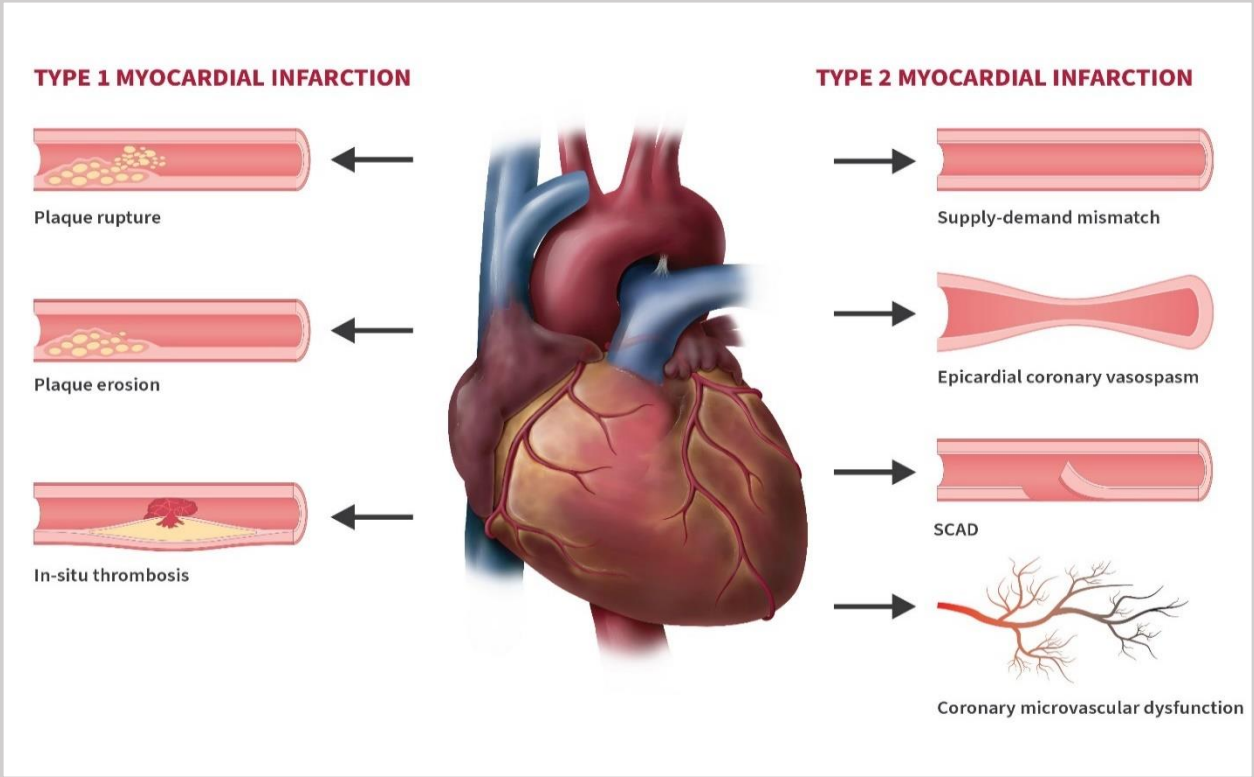

Illustration of Type 1 and Type 2 myocardial infarctions (MI), commonly known as a heart attack. Type 1 infarctions happen due to plaque rupture, plaque erosion or thrombosis. Type 2 infractions are caused by a supply-demand mismatch of oxygen to the heart muscle, often due to epicardial coronary vasospasm, a tear in the blood vessel known as spontaneous coronary artery dissection (SCAD) or coronary microvascular dysfunction (CMD). Source: From *A Guide to Your MINOCA* [Figure 2], by University of Ottawa Heart Institute (2024), A Guide to Your MINOCA | University of Ottawa Heart Institute. Copyright 2024 by University of Ottawa Heart Institute. Reprinted with permission.

In INOCA, although the main coronary arteries appear normal or have only mild blockages, there may still be underlying issues affecting blood flow to the heart. These underlying issues may include dysfunction in the small blood vessels (microvascular dysfunction), abnormalities in the lining of the blood vessels (endothelial dysfunction), or spasms in the coronary arteries (coronary artery vasospasm). The pathophysiology behind INOCA is believed to be multifactorial with coronary microvascular dysfunction and/or coronary vasospasm thought to be present in a majority of INOCA cases.

Coronary microvascular dysfunction (CMD) is a condition that affects the tiny blood vessels (micro-vessels) in the heart, specifically in the smaller coronary arteries which play a crucial role in delivering oxygen-rich blood to the myocardium. People with CMD experience typical anginal symptoms with evidence of ischemia, though they are not noted to have significant obstruction on further assessment of their coronary arteries.

An infrequent cause of acute MI is spontaneous coronary artery dissection (SCAD). SCAD occurs when there is a sudden split or tear in the wall of one of the coronary arteries without any obvious reason. This split or tear is often referred to as a dissection and can create a false passageway in the blood vessel compromising blood flow to the heart resulting in ischemia or infarction. SCAD predominantly affects females under the age of 50.

Please consult Table 1: Ischemic heart syndromes for coronary syndromes categorized into chronic versus acute.

Table 1: Ischemic heart syndromes

Chronic coronary syndrome	Acute coronary syndrome			
Stable Angina	NSTEMI	STEMI	SCD	Unstable angina
▸ Underlying CAD ▸ CMD	▸ IC thrombus/ plaque with CAD ▸ CMD ▸ Type 2 from other causes	▸ IC thrombus/ plaque with CAD ▸ Other causes	▸ Acute MI, ischemia ▸ Scar-mediated	▸ IC thrombus/ plaque with CAD ▸ Supply demand mismatch ▸ CMD

Note: AMI: Acute myocardial infarction, CAD: coronary artery disease, CMD: coronary microvascular dysfunction, IC: intracoronary, NSTEMI: non ST-elevation myocardial

infarction, SCD: sudden cardiac death, STEMI: ST-Elevation myocardial infarction. (Adapted from Concistre, G., 2023).

During episodes of inadequate perfusion, the mechanical, biochemical, and electrical functions of the heart tissue are disturbed. Ischemia causes characteristic changes on an ECG and the pumping function of the heart can become impaired. Cardiac troponin is a group of proteins found in heart muscle cells that play a crucial role in regulating contraction of the heart muscle. When heart muscle cells are damaged during reduced blood flow or ischemia, troponin is released into the blood stream making it an important biomarker for detecting myocardial ischemia.

Risk factors for IHD can be classified into modifiable and non-modifiable.

Non-modifiable risk factors include, but are not limited to:

- age
- biological sex
- genetic vulnerability
- family history
- ethnicity.

IHD is more prevalent in older age groups, males, Black, Hispanic, Latino, and Southeast Asian ethnicities, and those with a family history of IHD—particularly if cardiac disease is present in family members younger than 50 years of age. Young and middle-aged females with MI have poorer morbidity, mortality, and quality of life relative to comparable males, which is unexplained by traditional risk factors, comorbidities, and treatments.

Modifiable risk factors also have a significant role in the development of IHD and include aspects related to lifestyle or health that can be changed or controlled to reduce the risk of developing IHD.

Known modifiable risk factors include, but are not limited to:

- smoking
- diet
- physical activity
- obesity
- stress
- hypertension, including hypertensive disorders of pregnancy
- dyslipidemia
- diabetes.

In addition to known risk factors, there are several emerging risk factors for IHD under investigation, including inflammation, air pollution, and sleep disorders. For claims related to emerging risk factors of IHD, consultation with a disability consultant and/or medical advisor is recommended.

Clinical features

The clinical presentation of myocardial ischemia is most often acute chest discomfort. Angina pectoris, or angina for short, is the term used when chest discomfort is thought to be caused by myocardial ischemia. In individuals with myocardial ischemia, chest discomfort is often, but not always, present. Other associated symptoms with ischemia may be present, such as exertional shortness of breath, nausea, sweating, and fatigue. These symptoms have been called anginal equivalents.

The typical presentation of angina is a male over 50 or a female over 60 who reports episodic chest discomfort, usually described as heaviness, pressure, squeezing, smothering, or choking and rarely as frank pain. When asked to identify the location, the individual often places a hand over the sternum, sometimes with a clenched fist to indicate a squeezing, central, substernal discomfort. Angina typically lasts two to five minutes and can radiate to either shoulder or to both arms. Angina can also arise in or radiate to the back, shoulder blade region, base of the neck, jaw, teeth, or epigastrium.

Angina is often triggered by activities and situations that increase myocardial oxygen demand. These activities or situations may include physical activity, cold, emotional stress, sexual intercourse, meals, or laying down which results in an increase in blood return to the heart and increased stress to the heart walls. Typical angina is often relieved with discontinuation of the triggering factor.

The term acute coronary syndrome (ACS) includes acute myocardial infarction, ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial Infarction (NSTEMI), and unstable angina. MI is commonly called a heart attack.

While chest pain is the most common symptom of ACS, other symptoms of ischemia may be present. Chest pain is not required for the diagnosis of ACS. Many people with ACS present with symptoms such as dyspnea or malaise, either alone or in addition to chest pain. Females are more likely to have associated dyspnea than males, and individuals who are older or have diabetes are more likely to present with dyspnea without chest pain. Pain from myocardial ischemia is more often characterized as non-focal chest discomfort or pressure rather than pain, is generally gradual in onset, and is exacerbated by activity. Discomfort that radiates, particularly to arms, should increase suspicion for ACS.

The term acute MI is used when there is acute myocardial injury (STEMI or NSTEMI) with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cardiac troponin values. Acute MI is further categorized into types with type one and type two being the most common. A type one MI is caused by atherothrombotic disease of the coronary arteries which is usually triggered by erosion or rupture of the atherosclerotic plaque. A type two MI results from a mismatch between oxygen supply and demand and can include a variety of potential mechanisms, including coronary artery dissection, vasospasm, emboli,

microvascular dysfunction, as well as increases in demand with or without underlying CAD.

There are significant differences between females and males in the epidemiology, diagnosis, response to therapy, and prognosis of IHD. The importance of some metabolic, behavioural, and psychosocial risk factors may differ by biological sex.

Males more often experience the classic symptoms of IHD that include chest pain with radiation to the back and arms. Females more often present with atypical symptoms of IHD such as shortness of breath, chest pain at rest, nausea, and back or jaw pain. Symptoms experienced by females are often not readily recognized as diagnostic for IHD. Females present more often than males with signs and symptoms of IHD in the absence of obstructed coronary arteries.

Female Veterans have been shown to have higher rates of cardiovascular disease than their civilian counterparts. Younger females with previous MI have a two-fold likelihood of developing psychological stress induced myocardial ischemia compared to their male counterparts. Among transgender women, estrogen therapy is associated with higher triglyceride levels contributing to increased risk of cardiovascular disease, including MI.

Entitlement considerations

Section A: Causes and/or aggravation

For Veterans Affairs Canada (VAC) entitlement purposes, the following factors are accepted to cause or aggravate the conditions included in the Definition section of this EEG, and may be considered along with the evidence to assist in establishing a relationship to service. The factors have been determined based on a review of up-to-date scientific and medical literature, as well as evidence-based medical best practices. Factors other than those listed may be considered, however consultation with a disability consultant or medical advisor is recommended.

The timelines cited below are for guidance purposes. Each case should be adjudicated on the evidence provided and its own merits.

Factors

1. Having a diagnosis of **hypertension** before the clinical onset or aggravation of IHD.

2. Having a diagnosis of **diabetes mellitus** before the clinical onset or aggravation of IHD.

3. Having a diagnosis of **dyslipidemia** before the clinical onset or aggravation of IHD.

4. Having a diagnosis of **severe chronic kidney disease** before the clinical onset or aggravation of IHD.

   Note: Severe chronic kidney disease is defined as having a glomerular filtration rate (GFR) of less than 30 mL/minute/1.73m^2 for at least three months.

5. Having a clinically significant **psychiatric condition** for at least five years prior to clinical onset or aggravation of IHD.

   Note: For VAC purposes, clinically significant means requiring ongoing treatment and clinical management.

6. Being treated with a **medication** from the specified list below at the time of clinical onset or aggravation of IHD. The medications include, but are not limited to, the following:

   - antiandrogen therapy with a gonadotropin releasing hormone agonist for at least seven days prior to aggravation or onset
   - antipsychotic
   - aromatase inhibitor
   - bevacizumab
   - capecitabine
   - docetaxel
   - erlotinib
   - fluorouracil
   - paclitaxel
   - sorafenib.

   Note:

   - Individual medications may belong to a class of medications. The effects of a specific medication may vary from the class. The effects of the specific medication should be considered.
   - If it is claimed a medication resulted in the clinical onset or aggravation of IHD, the following must be established:

     - The medication was prescribed to treat an entitled condition.
     - The individual was receiving the medication at the time of the clinical onset or aggravation of the IHD.
     - The current medical literature supports the medication can result in the clinical onset or aggravation of IHD.
     - The medication use is long-term, ongoing, and cannot reasonably be replaced with another medication or the

medication is known to have enduring effects after discontinuation.

7. Having had exposure to **tobacco smoke** in a confined space. VAC recognizes that exposure to tobacco smoke is a risk factor for the development of IHD. For VAC purposes, entitlement may be considered for second-hand tobacco smoke exposure in a non-tobacco smoker where exposure has been for at least two hours per day, most days, in an enclosed space for at least one year duration at the time of the clinical onset of IHD.

For acute myocardial infarction only:

8. **Directly experiencing a traumatic event(s)** within 24 hours before the clinical onset or aggravation of IHD.

   Traumatic events include, but are not limited to:

   - exposure to military combat
   - threatened or actual physical assault
   - threatened or actual sexual trauma
   - being kidnapped
   - being taken hostage
   - being in a terrorist attack
   - being tortured
   - incarceration as a prisoner of war
   - being in a natural or human-made disaster
   - being in a severe motor vehicle accident
   - killing or injuring a person
   - experiencing a sudden, catastrophic medical incident
   - experiencing an acute, severe, emotional stressor.

9. Undertaking **strenuous physical exertion** within an hour of clinical onset or aggravation of IHD.

   Note: For VAC purposes, strenuous physical exertion means eight or more METs, and examples include running (approx. 8 km/hr), climbing hills with more than a 10kg load, and climbing more than one flight of stairs at a fast pace.

10. Inability to obtain **appropriate clinical management** of IHD.

Section B: Medical conditions which are to be included in entitlement/assessment

Section B provides a list of diagnosed medical conditions which are considered for VAC purposes to be included in the entitlement and assessment of IHD.

- Coronary artery disease (CAD)
- Angina, including Prinzmetal angina and unstable angina
- Non-ST elevation myocardial infarction (NSTEMI)
- ST elevation myocardial infarction (STEMI)
- Myocardial infarction (MI)
- Acute coronary syndrome (ACS)
- Spontaneous coronary artery dissection (SCAD)
- Ischemia with non-obstructive coronary artery disease (INOCA)
- Congestive heart failure (CHF)
- Cardiac arrhythmias, including, but not limited to:
    - Atrial fibrillation
    - Atrial flutter
    - Heart block
    - Ventricular tachycardia
    - Ventricular fibrillation
- Post infarction pericarditis

Section C: Common medical conditions which may result, in whole or in part, from ischemic heart disease and/or its treatment

No consequential medical conditions were identified at the time of the publication of this EEG. If the merits of the case and medical evidence indicate that a possible consequential relationship may exist, consultation with a disability consultant or medical advisor is recommended.

Links

Related VAC guidance and policy:

- Adjustment Disorder – Entitlement Eligibility Guidelines
- Anxiety Disorders – Entitlement Eligibility Guidelines
- Bipolar and Related Disorders - Entitlement Eligibility Guidelines
- Depressive Disorders - Entitlement Eligibility Guidelines
- Feeding and Eating Disorders - Entitlement Eligibility Guidelines
- Hypertension- Entitlement Eligibility Guidelines

- Posttraumatic Stress Disorder - Entitlement Eligibility Guidelines
- Schizophrenia - Entitlement Eligibility Guidelines
- Substance Use Disorders - Entitlement Eligibility Guidelines
- Pain and Suffering Compensation – Policies
- Royal Canadian Mounted Police Disability Pension Claims – Policies
- Dual Entitlement – Disability Benefits – Policies
- Establishing the Existence of a Disability – Policies
- Disability Benefits in Respect of Peacetime Military Service – The Compensation Principle – Policies
- Disability Benefits in Respect of Wartime and Special Duty Service – The Insurance Principle – Policies
- Disability Resulting from a Non-Service Related Injury or Disease – Policies
- Consequential Disability – Policies
- Benefit of Doubt – Policies

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