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Integrating care for chronic diseases: A review of management approaches and Outcomes

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Abstract

Chronic disease are considered a major public health concern worldwide, and most of these diseases like cancer, cardiovascular, metabolic, and neurological disorders occur due to atypical regulation of multiple signaling pathways. It has also been observed that most of the currently approved therapies for these diseases fail to show prolonged efficacy due to their mono-targeted nature and are associated with the development of chemoresistance, thus restricting their utility.

Keyword: Chronic care; Disease management; Care management; Diabetes; COPD; Hypertension

1. Introduction

Chronic disease is defined by the World Health Organization (WHO) as being of long duration, generally slow in progression and not passed from person to person. The Global Burden of Disease study 2013[1] reported a substantial (42.3%) increase in the years lived with disability (YLD) from 1990 to 2013[2]. This was overwhelming due to non-communicable diseases, with no infectious diseases in the top 20 leading causes of YLDs globally in 2013. Chronic condition multi-morbidity is high in developed countries and the prevalence of it increases with age; Australian data indicate that around 40% of people aged over 44 years have chronic disease multi-morbidity, increasing to around 50% for 65–74 year olds, and 70% for 85 years or over [3].

Primary health care (PHC) has obtain been held up as vital for health system globally to cope with many health challenge, from control of new and re-emerging infection disease to helping individuals prevent and manage chronic condition. Empirical research documents a strong positive association between the "strength" of PHC and various positive health system outcome, including better health at lower per capita cost.[4] Chronic diseases have one or more of the following characteristics: they are permanent, leave residual disability, are caused by non-reversible pathological alteration, require special training of the patient for rehabilitation, or may be expected to require a long period of supervision, observation or care. This definition includes both somatic and mental disorders. Thus, the National Board of Health's recommendations focus on the organization and provision of health care and the options of strengthened and supported self-management.This article focused on chronic diseases like Diabetes,COPD & Hypertension.

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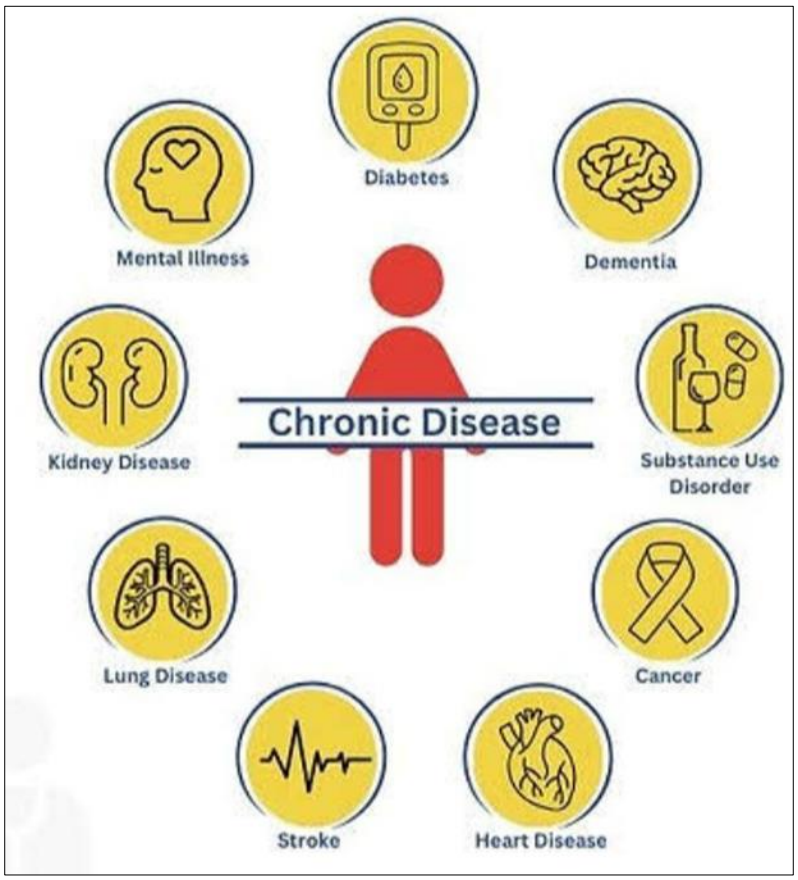

Figure 1 Different chronic diseases

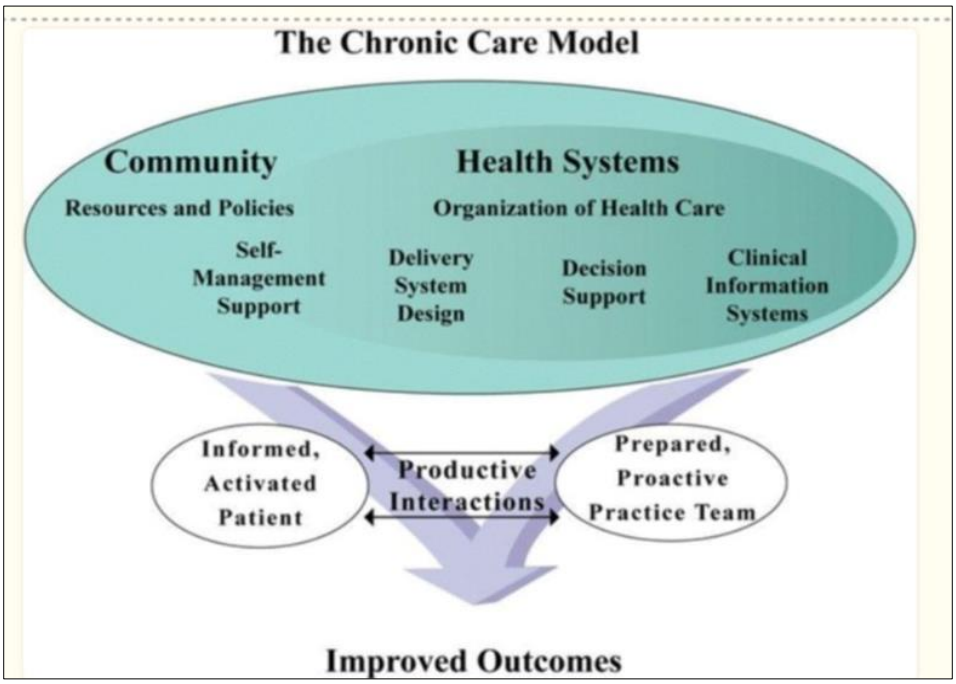

Figure 2 Chronic Care Model

2. Diabetes

Chronic hyperglycemia is a metabolic disorder caused by either a lack of insulin secretion, impaired insulin action, or both. Notably, insulin plays an important role as an anabolic hormone, affecting the metabolism of carbohydrates, lipids, and proteins. The metabolic abnormalities associated with diabetes mainly affect tissues such as adipose tissue, skeletal muscles, and the liver due to insulin resistance.[6] Impaired glucose tolerance (IGT) refers to an intermediate metabolic state between normal glucose tolerance (NGT) and diabetes mellitus (DM). Its prevalence in mainland China has been steadily increasing, reaching an estimated rate of 13.4% in 2021.1 This highlights the significant burden of IGT in these populations, with individuals in this intermediate state at a heightened risk of developing diabetes.2,3 Furthermore, individuals with diabetes are at an increased risk of developing cardiovascular (CV) events and have higher mortality rates compared with those with NGT.4–6 Notably, emerging evidence suggests that IGT may contribute to an elevated risk of CV events and mortal it [7]

Figure 3 Food chart

2.1. Causes of Diabetes

Diabetes mellitus is classified into four broad categories: type 1, type 2, gestational diabetes, and "other specific types". The "other specific types" are a collection of a few dozen individual causes. The term "diabetes", without qualification, usually refers to diabetes mellitus.[8]Type 1 diabetes develops when your immune system mistakenly attacks and destroys cells in your pancreas that make insulin. This destruction can happen over months or years, ultimately resulting in a total lack (deficiency) of insulin. Although scientists don't yet know the exact cause of Type 1 diabetes, they believe there's a strong genetic component. The risk of developing the disease with no family history is approximately 0.4%. If your biological mother has Type 1 diabetes, your risk is 1% to 4%, and your risk is 3% to 8% if your biological father has it. If both of your biological parents have Type 1 diabetes, your risk of developing the condition is as high as 30%. Scientists believe that certain factors, such as a virus or environmental toxins, can trigger your immune system to attack cells in your pancreas if you have a genetic predisposition for developing Type 1 diabetes.

2.2. Classification of diabetes

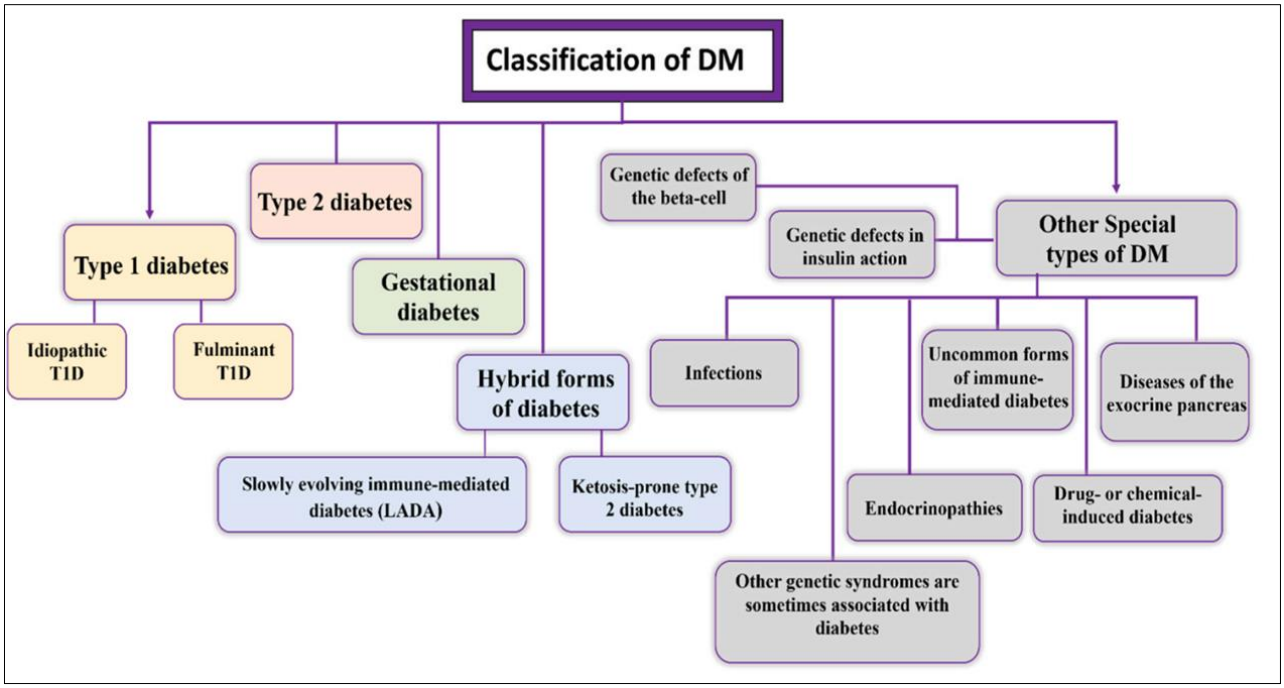

Figure 4 Classification Diabetes Mellitus

2.3. Type 1

Type 1 account for 5 to 10% of diabetes cases and is the most common type diagnosed in patients under 20 years; however, the older term "juvenile-onset diabetes" is no longer used as onset in adulthood is not unusual. The disease is characterized by loss of the insulin-producing beta cella of the pancreatic islets, leading to severe insulin deficiency, and can be further classified as immune- medicated or idiopathic (without known cause). The majority of cases are immune-mediated, in which a T- cell mediated autoimmune attack causes loss of beta cells and thus insulin deficiency. Patients often have irregular and unpredictable blood sugar levels due to very low insulin and an impaired counter-response to hypoglycemia.

2.4. Type 2

Type 2 diabetes is due primarily to lifestyle factors and genetics. A number of lifestyle factors are known to be important to the development of type 2 diabetes, including obesity (defined by a body mass index of greater than thirty), lack of physical activity, poor diet, stress, and urbanization. Excess body fat is associated with 30% of cases in those of Chinese and Japanese descent, 60-80% of cases in those of European and African descent, and 100% of Pima Indians and Pacific Islanders. Those who are not obese often have a high waist–hip ratio.[9]

2.5. Treatments of diabetes

For both T1DM and T2DM, the cornerstone of therapy is diet and exercise. A diet low in saturated fat, refined carbohydrates, high fructose corn syrup, and high in fiber and monounsaturated fats needs to be encouraged. Aerobic exercise for a duration of 90 to 150 minutes per week is also beneficial. The major target in T2DM patients, who are obese, is weight loss.If adequate glycemia cannot be achieved, metformin is the first-line therapy. Following metformin, many other therapies such as oral sulfonylureas, dipeptidyl peptidase-4 (DPP-4) inhibitors. Glucagon-like peptide-1 (GLP-I) receptor agonists, Sodium-glucose co-transporter-2 (SGLT2) inhibitors, pioglitazone, especially if the patient has fatty liver disease, alpha-glucosidase inhibitors, and insulin, are available.[10]

Impaired glucose metabolism is a substantial burden on population health, and our findings emphasise the need for more effective prevention strategies, which should be implemented as soon in a person's life as possible. The substantial lifetime risk of prediabetes and diabetes in lean individuals also supports risk factor control in non-obese individuals.[11]

3. COPD:-chronic obstructive pulmonary disease

COPD comprises a diverse group of clinical syndromes that share the common feature of limitation of expiratory airflow. The American Thoracic Society defines COPD in terms of chronic bronchitis and emphysema. Chronic bronchitis is characterized by the clinical symptoms of excessive cough and sputum production; emphysema refers to chronic dyspnea, resulting from enlarged air spaces and destruction of lung tissue. The GOLD initiative defines COPD as "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases." Asthma is also characterized by airflow obstruction and inflammation, but in addition it involves hyperresponsiveness of the airways to stimulus; therefore, the reversibility of functional deficits in asthma differentiates it from COPD.[12]

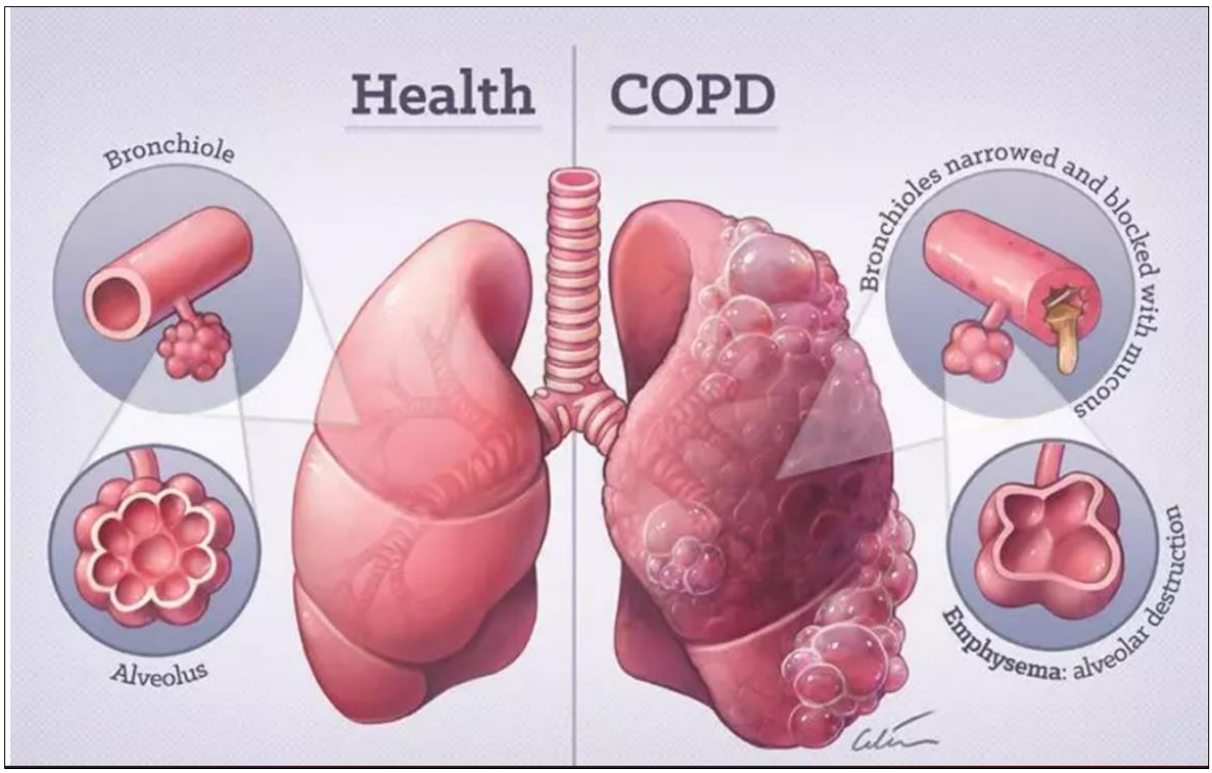

Figure 5 COPD

3.1. Pathophysiology of COPD

According to the classic paradigm proposed by Fletcher and Peto in 1977, COPD has been traditionally understood as a self-inflicted disease caused by tobacco smoking characterized by an accelerated decline in lung function. This view of COPD has changed recently thanks to new knowledge obtained in epidemiological, clinical, and imaging studies. In 2015, a study of subjects followed over time in three different cohorts, found that close to 50% of patients diagnosed with COPD at 62 years of age.[13]

As previously discussed, the primary purpose of identifying the MetS was to identify a clustering of features that were associated with increased CVD risk. As the term syndrome implies, a specific causative etiology to the MetS is not clear, nor was a common, unifying pathophysiological cause of the MetS necessarily intended. Nevertheless, abdominal adiposity and insulin resistance appear to be at the core of the pathophysiology of the MetS and its individual components. Thus, the purpose of this section is to review how abdominal adiposity and insulin resistance may contribute to the pathophysiology of the MetS. [14]

3.2. History and physical of COPD

COPD will typically present in adulthood and often during the winter months. Patients usually present with complaints of chronic and progressive dyspnea, cough, and sputum production. Patients may also have wheezing and chest tightness. While a smoking history is present in most cases, there are many without such history. They should be questioned on exposure to second-hand smoke, occupational and environmental exposures, and family history. Those

with a confirmed diagnosis of COPD should be asked about previous exacerbations, nighttime awakenings, inhaler usage, and the impact of the disease on activity level. Patients should be questioned on their past medical history for other diseases such as asthma, allergies, and childhood respiratory infections. Those with liver disease, basilar emphysema, and a family history of emphysema should raise suspicion for alpha-1 antitrypsin deficiency. Acute exacerbations of COPD usually present with increased dyspnea, productive cough, and wheezing.

Patients with COPD may have multiple physical findings as follows:

- General
  - Significant respiratory distress in acute exacerbations
  - Muscle wasting
- Lungs
  - Accessory respiratory muscle use
  - Prolonged expiration
  - Wheezing
  - Pursed-lip breathing
- Chest
  - Increased anterior-posterior chest wall diameter (barrel chest)
- Skin
  - Central cyanosis when arterial oxygenation is low
- Extremities
  - Digital clubbing
  - Lower extremity edema in right heart failure [15]

3.3. Treatment of COPD

Data from recent large-scale randomized clinical trials (RCTs) on triple combination therapy (long-acting ß-adrenergic agonist/long-acting muscarinic antagonist/inhaled corticosteroid [LABA-LAMA-ICS]) for COPD have improved our understanding of the correct use of available drugs, added value of ICSs, and eosinophil count as a potential biomarker.[16] The primary goals of treatment are to control symptoms, improve the quality of life, and reduce exacerbations and mortality. The non-pharmacological approach includes smoking cessation and pulmonary rehabilitation.

Annual influenza vaccination is recommended in all patients with COPD. Patients aged 65 and over should receive the 13-valent pneumococcal conjugate vaccine (PCV13) and the 23-valent pneumococcal polysaccharide vaccine (PPSV23) at least one year apart. The PPSV23 is recommended for those aged 64 and younger with significant comorbidities (e.g., diabetes mellitus, chronic heart disease, chronic lung disease).

The classes of commonly used medications in COPD include bronchodilators (beta2-agonists, antimuscarinics, methylxanthines), inhaled corticosteroids (ICS), systemic glucocorticoids, phosphodiesterase-4 (PDE4) inhibitors, and antibiotics.[17] The single most important intervention in modifying the course of COPD in patients who smoke is smoking cessation. The Lung Health Study reported a progressive decline in postbronchodilator FEV1 in men and women who continued to smoke over an 11-year period.[18]

CF is a devastating genetic disease resulting in premature death of patients, usually because of respiratory failure. The dysfunctional innate immune response, specifically the interplay between neutrophils and macrophages, may have an important role in the pathophysiology of CF lung disease. We hypothesise NETs are a hindrance rather than a help in CF, contributing to inflammation and lung damage rather than working effectively in their anti-microbial capacity. Further studies are essential to investigate the pro-inflammatory nature of NET constituents with the aim of identifying precise, new treatment strategies for CF.[19]

4. Hypertension

Hypertension is the most common chronic disease in the developed world affecting up to 25% of the adult population. It remains the most important modifiable risk factor for coronary heart disease, stroke, congestive heart failure, renal disease, and peripheral vascular disease. Suboptimal blood pressure control is responsible for 62% of cerebrovascular disease, 49% of ischemic heart disease, and an estimated 7.1 million deaths a year. Because of the associated morbidity, mortality and economic cost to society early diagnosis and treatment within the established guidelines is imperative. A

sizeable percentage of the hypertensive population does not manage to achieve adequate control in spite of receiving 3 or more antihypertensive medications. These are the patients with resistant hypertension.[20]

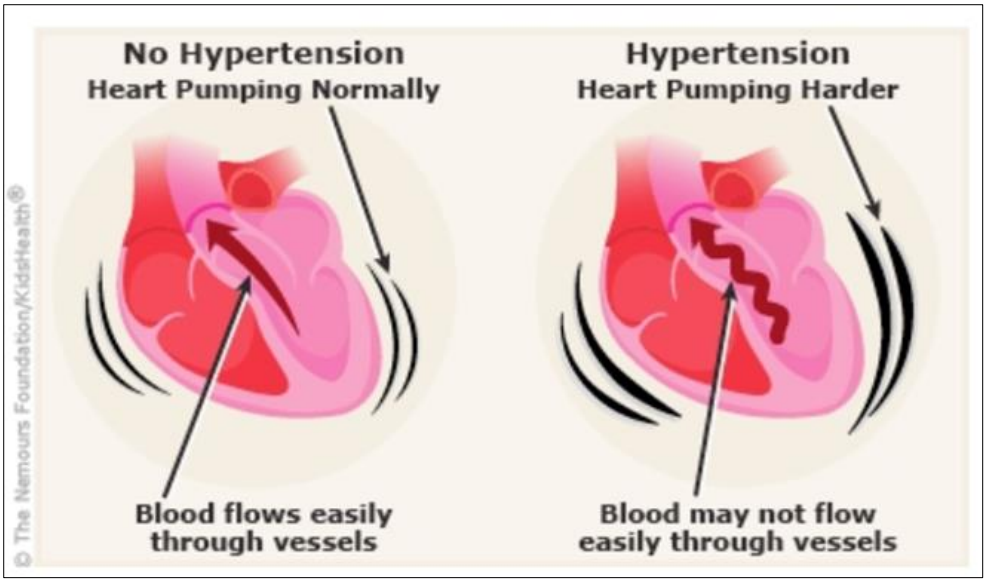

Figure 5 Heart pumping normally /Harder

The circulatory system is responsible for providing oxygen and other nutrients essential for life to all organs in the body. When sustained high blood pressure occurs in the circulatory system, one of two types of hypertension can be diagnosed: essential hypertension and secondary hypertension. In the 90– 95% of cases in which no identifiable cause of elevated blood pressure can be discerned, the condition is called essential hypertension. In contrast, secondary hypertension results from an identifiable cause, like presence of underlying kidney disease, coarctation (narrowing) of the aorta, metabolic disorders, or specific enzyme deficiencies.[21]

Table 1 Hypertension Prevalence and treatment and Body Mass Index (BMI) Among Persons 35 to 64 Years old in 6 European Countries, Canada and the United States.

Country	Prevalence, %			Hypertensive Persons Taking Medications, %	BMI
	All	Men	Women		
North America	27.6	30.4	24.8	44.4	27.1
United States	27.8	29.8	25.8	52.5	27.4
Canada	27.4	31.0	23.8	36.3	26.8
Europe	44.2	49.7	38.6	26.8	26.9
Italy	37.7	44.8	30.6	32.0	26.4
Sweden	38.4	44.8	32.0	26.2	26.5
England	41.7	46.9	36.5	24.8	27.1
Spain	46.8	49.0	44.6	26.8	27.4
Finland	48.7	55.7	41.6	25.0	27.1
Germany	55.3	60.2	50.3	26.0	27.3

*Age adjusted. BMI calculated as weight in kilograms divided by the height in meters squared.

4.1. Hypertension prevalence

In Table, We present the age and sex adjusted prevalence of hypertension at the standard threshold (ie, BP 140/90mm Hg or treatment with antihypertensive medication). The prevalence was highest in Germany (55%), Followed by Finland (49%), spain (47%), England (42%), Sweden (38%), and Italy (38%). Prevalences in the United States and Canada were half of the rate in Germany (28% and 27%, respectively). The prevalence of hypertension for the European average was 44.2% compared with 27.6% in North America. The BMI was the only lifestyle measure available to our

analysis. The range was narrow (26.4-27.4) an weakly correlated with hypertension prevalence (r =0.22). The BMI in North America was 27.1 vs 26.9 in Europe, suggesting that obesity explains little of the overall difference in BP.

4.2. Hypertension control

Hypertension treatment in the Europran countries was on average lower than in the North American countries. The rank order for the proportion of persons undergoing treatment with a BP lower than 140/90mm Hg in the European countries was England, Italy, Germany, Finland, Sweden, and Spain; however, the variation was small, ranging from 5% to9%. For the European countries, on average only 8% of hypertensive individuals had their condition controlled copared with 23% in Canada and the United States (persons 35-64 of age)[22]

4.3. Management of hypertension

Acute hypertension is common after major surgery and may be associated with an increased risk of serious cardiac and neurologic, complications. Indeed perioperative hypertension often occurs in conjunction with one of the following events: during the induction of anesthesia; intraoperatively as associated with acute pain-induced sympathetic stimulation leading to vasoconstriction; in the early postanesthesia period, associated with pain induced sympathetic stimulation, hypothermia, hypoxia or intravascular volume overload from excessive intraoperative fluid therapy; and in the 24 – 48 h postoperatively, as fluid is mobilized from the extravascular space. In addition, BP elevation secondary to discontinuation of long-term antihypertensive medication may occur postoperatively[23].

4.4. Treatment of hypertension

Although hypertension has many dangerous medical consequences, it is easily treated, and a multitude of interventions have demonstrated efficacy in lowering blood pressures. Treatment of hypertension typically follows a step-wise approach with lifestyle modifications recommended as the first step and pharmacological treatment initiated after lifestyle modifications have failed to reduce blood pressure for most patients (James et al., 2014; National Heart, Lung, and Blood Institute, 2004). For patients with stage 2 hypertension, pharmacological treatment is often initiated immediately due to the urgent need to reduce blood pressure rapidly. Pharmacologic treatment is initiated after a failure to achieve blood pressure reduction using lifestyle modification alone or concurrent to lifestyle modification efforts. Thiazide-type diuretics are often recommended and are sufficient for controlling blood pressure for most patients as well as preventing cardiovascular diseases associated with hypertension[24].

5. Conclusion

To further improve the responsiveness of DMPs to patients needs, we suggest to monitor patient relevant outcomes that might be based on the ICF-model. To address the needs of patients with multimorbidity, we propose a generic comprehensive model, embedded in primary care. A goal-oriented approach provides the opportunity to prioritize goals that really matter to patients [25]. Hypertensive emergencies have the potential for permanent end organ damage and significant morbidity and mortality. Patients with hypertensive crises may require immediate reduction in elevated BP to prevent and arrest progressive end-organ damage [26]. DM is a complex metabolic disorder that affects multiple organ systems and leads to various complications, including kidney disease, cardiovascular disease, immune dysfunction, retinopathy, and neuropathy. The knowledge of the underlying mechanisms and pathways involved in these complications has opened new avenues for future perspectives and potential treatments to inhibit the burden of diabetes and its complications [27].

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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