

# Hyperglycemia Syndromes



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## KEYWORDS

- Diabetes • Hyperglycemia • Complications of diabetes
- DKA (diabetic ketoacidosis) • HHS (hyperglycemic hyperosmolar state)
- Glycemic control

## KEY POINTS

- Diabetes is one of the leading causes of organ failure in the world with high rates of microvascular and macrovascular disease.
- Diabetic ketoacidosis and hyperglycemia hyperosmolar syndrome are two serious complications of diabetes that can lead to significant morbidity and mortality.
- Individualized, patient-centered care is crucial to avoiding complications of diabetes; people with well-controlled diabetes can live long and healthy lives.

## INTRODUCTION

Diabetes mellitus (DM) has become a global pandemic. In 2014, it was estimated that more than 422 million people had diabetes worldwide<sup>1</sup> and it is predicted that approximately 592 million people globally will have diabetes by 2035.<sup>2</sup> Additionally, nearly 1.5 million Americans are diagnosed with diabetes every year, and 23.8% of people with diabetes are undiagnosed.<sup>3</sup> The financial burden of diabetes in the United States is approximately \$245 billion a year, including direct and indirect costs, a staggering number that continues to climb.<sup>4</sup> In 2014, there were 300 million deaths associated with diabetes and it is the seventh leading cause of death in the United States.<sup>5</sup> Diabetes is one of the leading causes of organ failure, and timely, evidence-based patient education and management is essential to preventing complications.

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## **PATHOPHYSIOLOGY OF DIABETES**

DM is characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both.<sup>6</sup> There are several pathophysiologic processes that occur leading to the development of type 2 DM (T2DM). These processes are described as follows.

### ***Beta-Cell Failure***

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The  $\beta$ -cells of the pancreas are no longer producing enough insulin to match physiologic needs, leading to greater amounts of circulating glucose and hyperglycemia.

### ***Insulin Resistance***

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Insulin may have a reduced effect, leading to lessened peripheral glucose uptake and higher blood glucose (BG).

### ***Inappropriate Hormone Release***

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In T2DM, the feedback systems for hepatic glucose production and glucagon release are defective, leading to inappropriate glucose release from the liver and glucagon release from the alpha cells of the pancreas, even when sugars are normal or high.

### ***Decreased Incretin Effect***

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The incretin hormones of the intestines aid in food digestion and promoting feelings of satiety. The 2 main incretin hormones, GIP and GLP-1, are instrumental in promoting insulin release and inhibiting glucagon production. Individuals with T2DM have a poor response to incretin hormones, leading to poor carbohydrate absorption and delivery and hyperglycemia.

The development of T2DM is a heterogenous condition involving multiple factors including genetics and environment.<sup>7</sup> T1DM is an autoimmune process in which the islet cells of the pancreas are attacked by the body, leading to no insulin production. Similar to T2DM, there are many factors likely involved in the development of T1DM including genetic susceptibility, ineffective immune response, and inflammation.<sup>6</sup>

## **DIAGNOSIS OF TYPE 1 DIABETES VERSUS TYPE 2 DIABETES**

Although there are at least 5 classifications of diabetes including prediabetes, T1DM and T2DM are the 2 predominant types. Evidence affirms that T2DM accounts for approximately 90% to 95% of diabetes cases.<sup>8</sup> The symptoms of T1DM and T2DM are similar; however, there are key clinical findings ([Table 1](#)) that are specific to each type that are helpful in guiding a correct diagnosis. These defining characteristics are necessary to properly guide assessment, diagnosis, planning, interventions, evaluations, treatment regimens, and research efforts.

## **MANAGEMENT OF TYPE 1 DIABETES VERSUS TYPE 2 DIABETES**

Although diagnostic criteria are similar for both T1DM and T2DM, the treatment approach is considerably different. Patients with T1DM require exogenous insulin throughout their lifetimes due to the autoimmune destruction of the beta cells, leaving the body deficient of endogenous insulin. Subsequently, administration of basal and bolus insulin via multiple injections or insulin pump devices is necessary to sustain life in patients with T1DM.

For people with T2DM, metformin, along with diet and lifestyle changes, is typically initiated as first-line therapy. Developments of new pharmacologic agents often pose

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