



A SYSTEMATIC REVIEW FOR PARTIAL FULFILMENT OF  
MASTER DEGREE IN GENERAL SURGERY

# **Role of Laparoscopic Ileal Interposition Associated with Sleeve Gastrectomy in Management of Type 2 Diabetes Mellitus**

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*Ahmed El Sayed Abdel Hamid El Kordy*

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## **List of Abbreviations**

<b>Abb.</b>	<b>Full Term</b>
<b>ADA</b>	American Diabetes Association
<b>BMI</b>	Body mass index
<b>BPD</b>	Biliopancreatic diversion
<b>BPD&amp;DS</b>	Biliopancreatic diversion and duodenal switch
<b>DJB</b>	Duodenal–jejunal bypass
<b>DJB&amp;SG</b>	Duodenal–jejunal bypass with sleeve gastrectomy
<b>DMGB</b>	Diverted mini gastric bypass
<b>DS</b>	Duodenal switch
<b>FPG</b>	Fasting plasma glucose
<b>GIP</b>	Insulinotropic polypeptide
<b>GLP-1</b>	Glucagon-like peptide 1
<b>IDF</b>	International Diabetes Federation
<b>II</b>	Ileal interposition
<b>II-DSG</b>	Ileal interposition with diverted sleeve gastrectomy
<b>JII-SG</b>	Jejuno-ileal interposition with sleeve gastrectomy
<b>LAGB</b>	Laparoscopic adjustable gastric banding
<b>LSG</b>	Laparoscopic sleeve gastrectomy
<b>MGB</b>	Mini gastric bypass
<b>OAGB</b>	One anastomosis gastric bypass
<b>OHA</b>	Oral hypoglycemic agents

<b>Abb.</b>	<b>Full Term</b>
<b>OXM</b>	Oxyntomodulin
<b>PYY</b>	Peptide YY
<b>ROS</b>	Reactive oxygen species
<b>RYGB</b>	Roux-en-Y gastric bypass
<b>SADI-S</b>	Single anastomosis duodenoileal bypass with sleeve gastrectomy
<b>SASI</b>	Single anastomosis sleeve ileal bypass
<b>SG</b>	Sleeve gastrectomy
<b>T2DM</b>	Type 2 diabetes mellitus
<b>TB&amp;SG</b>	Transit bipartition and sleeve gastrectomy
<b>WHO</b>	World Health Organization



## **Introduction**

Diabetes is a debilitating, life-threatening disease accounting for the death of millions of people every year all over the world, its out breaking prevalence as well as its hazards complications such as cardiovascular disease, renal disease and infectious diseases among others makes it a major health challenge of the twenty-first century and has led to the search for novel procedures that can provide sustained glycemic control (**Harding et al., 2018**).

According to the last estimations about 415 million adults currently suffer from diabetes and this number is expected to rise to 642 million by 2040 (**Ogurtsova et al., 2019**).

The ordinary management of type 2 diabetes mellitus (T2DM) includes lifestyle modulations and oral hypoglycemic drugs, insulin or combination between both (**Chatterjee et al., 2017**).

While the medications aim to maintain acceptable blood glucose levels, the lifestyle modulations aim to achieve loss of weight to target the underlying pathophysiology of T2DM (**Tsilingiris et al., 2019**).

The significant effects of bariatric and metabolic surgery regarding loss of weight and metabolic amendment have put it as a possibility for T2DM management. By addressing many complementary pathogenic mechanisms, bariatric surgery appears to be hopeful in the reversal of the metabolic abnormalities of T2DM (**Rubino et al., 2016**).

Ileal interposition enhance the insulin sensitivity and improve the  $\beta$ -cell function in an effective way that overcomes the traditional medical therapy (**Mingrone et al., 2013**).

Laparoscopic ileal interposition associated with sleeve gastrectomy emerged as a choice for T2DM management due to its effect on glucose hemostasis as it intends to target the pathophysiology of the underlying mechanisms of the disease (**DePaula et al., 2012**).

The procedure combines between the restrictive role of the sleeve gastrectomy and the malabsorptive role of the interposed ileal segment (**Ugale et al., 2017**).

## **The aim of the work**

In this study we attempt to illustrate, through the available researches, the role of laparoscopic ileal interposition associated with sleeve gastrectomy in management of type 2 diabetes mellitus and the feasibility and efficacy of this procedure as an option in management of T2DM and assess the risk to benefit profiles.

## **Chapter I: Pathophysiology of Type 2 Diabetes Mellitus (T2DM)**

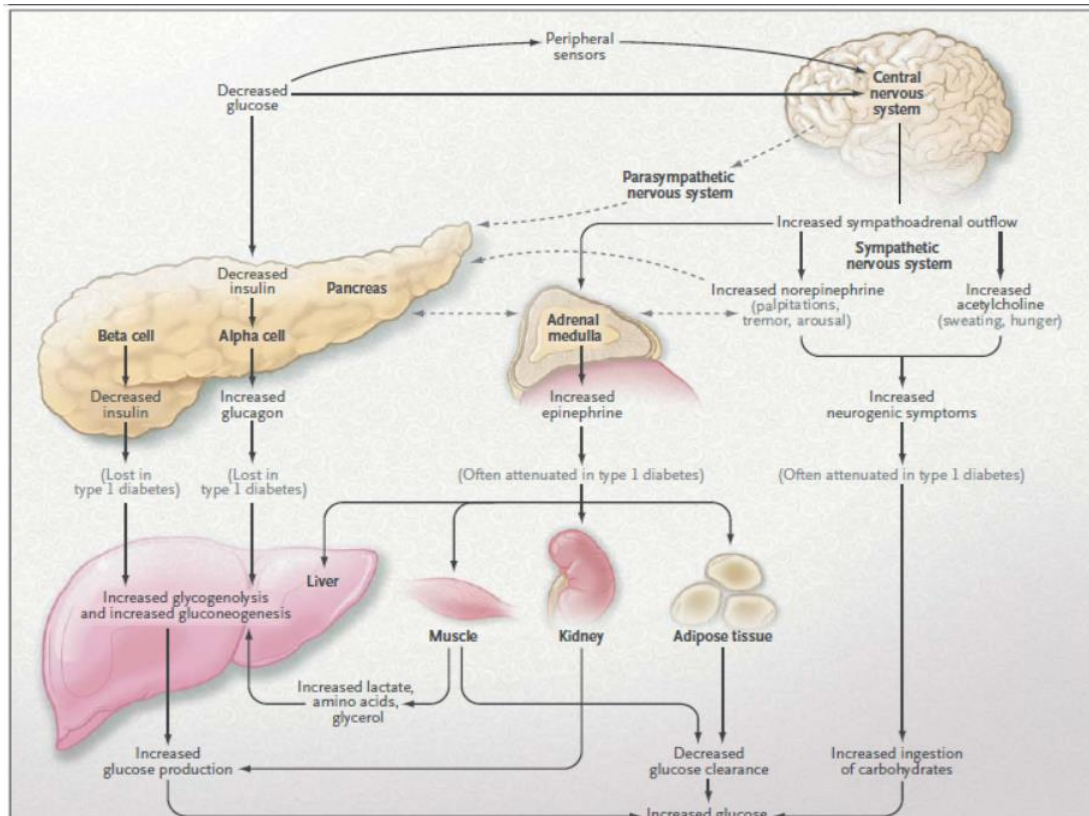
T2DM is a heterogeneous metabolic disorder characterized by lack of control of glucose hemostasis due to failure of the body to produce sufficient levels of insulin due to pancreatic islet beta cell dysfunction or ineffective usage of insulin due to insulin resistance (**Zaccardi et al., 2016**).

T2DM represents about 85% to 95% of all cases of diabetes making it the most common type of diabetes all over the world (**World health organization (WHO), 2019**).

### ➤ **Pathogenesis:**

The etiology of hyperglycemia in T2DM is a complex multifactorial process (**Javeed and Matveyenko, 2018**).

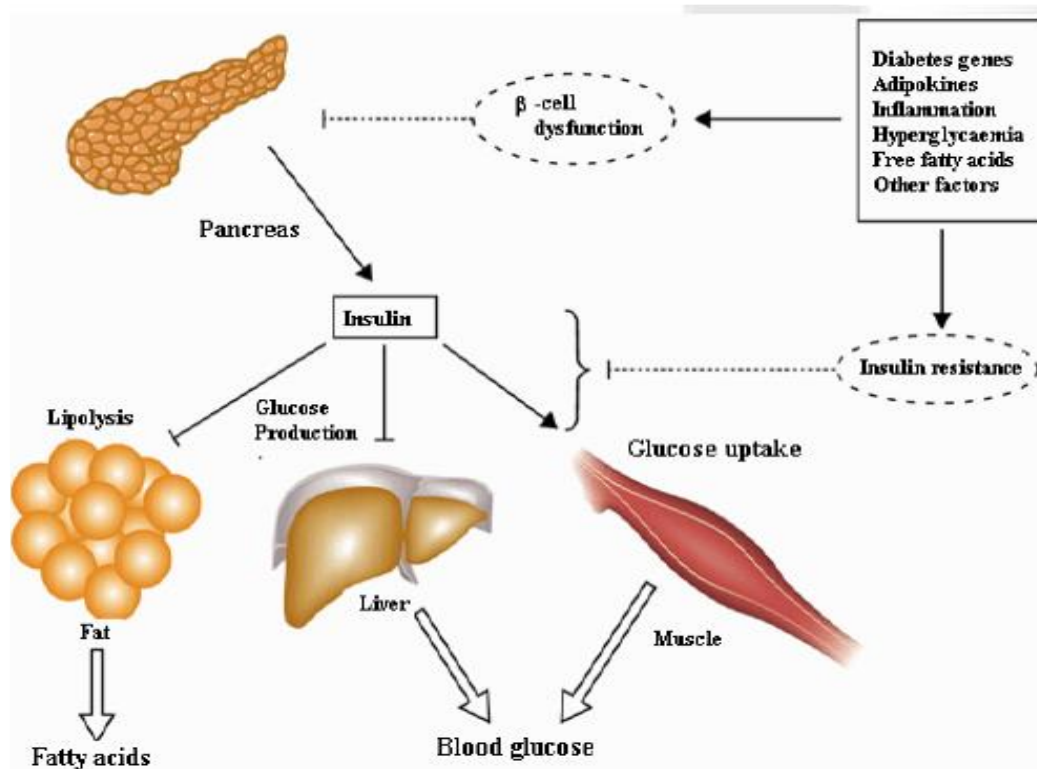
Blood glucose level is maintained by a feedback system, by which islet beta cells of the pancreas respond to the elevation of blood glucose level by secreting insulin (**Kahn et al., 2014**).



**Fig. (1):** Physiologic and Behavioral response of hyperglycemia (Baynest, 2015).

- Insulin resistance: The visceral fat is a major source of inflammatory cytokines that affect insulin action. These cytokines impair the insulin- glucose uptake process, and so the plasma glucose level rises (Baynest, 2015).
- Pancreatic beta cell dysfunction: The islet beta cells of the pancreas respond to the high glucose level by producing more insulin in an attempt to maintain glucose homeostasis, over the time these cells become exhausted and die, so insulin production diminishes and inadequate glucagon suppression happens, leading to failure of maintaining glucose concentration at its normal range (4-7mmol/L) (Kahn et al., 2014).

- The infiltration of the pancreatic beta cells with fat and amyloid deposits, beside the abnormal alpha cell function (glucagon secretion) determines the hyperglycemic state (**Carrera Boada and Martinez-Moreno, 2013**).



**Fig. (2):** Pathophysiology of Hyperglycemia and increased circulating fatty acids in type 2 Diabetes (**Baynest 2015**).

### ➤ Predisposing factors of T2DM:

There are several factors participating in the incidence of T2DM such as;

- Genetic factors: The overwhelming advances in genetic science and technology have participated in the identification of genes that are linked to the onset of T2DM. But it is still too early to use genetics for the prediction of T2DM incidence (**Kahn et al., 2014**).

- Sedentary lifestyle: High-calories diets and lack of physical activity are associated to the sedentary lifestyle. The high energy and fast foods with high levels of fat and sugar contributes significantly in the epidemic widespread of T2DM in developed countries (**Ley et al., 2016**).

Moreover, these diets are lacking essential vitamins as vitamin D, vitamin B12 and folic acid which have been implicated in the pathogenesis of T2DM by their deficiency (**Nolan et al., 2011**).

- Race: Black and minority races are six times more likely to have T2DM compared to the white Caucasian race as they use more abdominal fat storage and do not store as much fat as other races in less metabolically active sites such as the thighs and buttocks. The central fat distribution and the increase in the adiposity of these races increase the body resistance to insulin and the ability to develop T2DM (**Gholap et al., 2011**).
- Age: T2DM was rarely seen in children and adolescents until recently. There is a significant increase in the numbers of children and adolescents being diagnosed with T2DM corresponding to an increase in obesity levels in this age group (**Reinehr, 2013**).

The prevalence of T2DM in the age between 10 to 19 years increased in the United States by 30.5% in the last decade (**Dabelea et al., 2014**).

- Endocrine-disrupting chemicals: Many chemical substances such as pesticides, food and cosmetic preservatives, components used in the plastics industry, consumer products and products of waste incineration can alter the function of the endocrine system and cause a various effects including T2DM (**Chevalier and Fénichel, 2015**).