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# Potential Therapeutic Effects of a GLP-1 Analogue on Blood Pressure in Morbidly Obese Patient: A Case Report

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**Case Report**

**Keywords:** inflammation, obesity, hypertension, cardiovascular risk factors, GLP-1 analogues

**Posted Date:** September 4th, 2026

**DOI:** https://doi.org/10.21203/rs.3.rs-10856515/v1

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**Additional Declarations:** The authors declare no competing interests.

# Abstract

Obesity is a global pandemic associated with various cardiometabolic disorders. Several new medications are being developed for the treatment of obesity. Glucagon-like peptide-1 (GLP-1) is naturally secreted in the intestine and plays an important role in regulating insulin and glucagon secretion. It is also responsible for managing post-prandial glucose excursions and reduced appetite. Currently, GLP-1 analogues are being used for the treatment of obesity. This is a case of a morbidly obese hypertensive patient with a body mass index (BMI) of 62.84 kg/m², who was treated with a GLP-1 analogue, and experienced significant weight loss. Initially, the patient was taking three different antihypertensive medications to manage his blood pressure. As the patient's BMI declined, the blood pressure readings also began to improve. Eventually, the patient was taken off antihypertensive medications and maintained a mean blood pressure of 113/74 mmHg at a BMI of 47.37 kg/m².


# INTRODUCTION

Inflammation is the body's natural protective system to fight against harmful irritants, infections and insults, maintaining the integrity of cells and normal homeostasis. There are two types of inflammation: acute and chronic inflammation. Acute inflammation is developed over a short time, resulting in edema and leukocyte migration. Whereas, chronic inflammation, as the name indicates, lasts for a long time leading to the accumulation of lymphocytes and macrophages, as well as, blood cells and connective tissue proliferation [1].

The pathophysiology of metabolic syndrome is based on inflammation, and a characteristic feature of metabolic syndrome is obesity. Obesity is defined as an excess amount of adipose tissue. Obesity is classified into three classes: Class I obesity is BMI of 30.0 to 34.9 kg/m², Class II obesity is BMI of 35.0 to 39.9 kg/m², and Class III obesity, formerly known as morbid obesity, is BMI of 40.0 kg/m² or above [2]. Obesity has become an epidemic, contributing to 2.8–3.4 million deaths annually [3]. The future projection indicates that by 2035, a quarter of the world's population, almost 2 billion people, will be obese or overweight [4]. When the energy consumption is greater than the expenditure, the body starts storing the excess energy in the form of adipose tissue. There are two main types of adipose tissue: brown and white adipose tissues. In newborns, brown adipose tissue is involved in thermogenesis, and in adults, it is inversely related to body mass index (BMI). White adipose tissue is involved in physiological and pathological processes. Adipose tissue is responsible for producing and releasing various inflammatory factors, such as leptin, IL-6, TNF-alpha (TNF-$\alpha$), and cytokines.The overexpression of the pro-inflammatory markers in adipose tissue results in inflammation in patients with obesity and inflammation serves as a hub for metabolic syndrome, leading to an increased risk of cardiovascular events, cancers, and multiple endocrine disturbances.

The prevalence of obesity has increased from 34.7 million in 1990 to 107 million in 2022, in individuals 20 years or older [5]. This epidemic of obesity and its impact on comorbidities raises serious concerns, and calls for a solution. One of the associated comorbidities of obesity is hypertension caused by

stimulation of the sympathetic nervous system, activation of renin-angiotensin aldosterone system and activation of immune inflammatory system. In 2010, an estimated 1.39 billion adults had hypertension, equally distributed between men and women [6]. Abnormal adipokine secretion from adipose tissue results in sympathetic nervous system (SANS) activation by stimulating alpha- and beta-adrenergic receptors, resulting in elevated heart rate, increased cardiac output, and renal sodium tubular reabsorption. The pharmacological blockade of adrenergic receptors in obese patients results in greater reduction in systolic blood pressure compared to lean hypertensive patients. Therefore, SANS plays an important role in causing hypertension in obese patients. Obese individuals also express higher levels of renin which results in activation of the renin angiotensin aldosterone system (RAAS) leading to vasoconstriction and hypertension. RAAS also activates the

SANS system, which is reciprocal as well. Leptin, an adipokine, has also shown stimulation of the SANS system further contributing to obesity-related hypertension [7].

Newer and effective weight management medications stemmed from global concern about obesity, which includes GLP-1 analogues. GLP-1 (glucagon-like peptide-1) is naturally secreted by L-cells of the small intestine and colon and broken down by dipeptidyl peptidase-4. It helps regulate blood glucose level and postprandial glucose excursions by stimulating insulin secretion and inhibiting glucagon secretion; it also slows down intestinal motility, reducing appetite and food intake [8]. All of these factors contribute to weight loss and reduction in the inflammatory burden. In the recent clinical trials, GLP-1 analogues contributed to 15–25% of body weight over the period of 12–18 months [9].

GLP-1 analogues are emerging for their use in the treatment of obesity and weight reduction. They also function as anti-inflammatory agents. The NF-κB pathway is a central pathway for inflammation, and GLP-1 analogues inhibit NF-κB pathway, reducing inflammation. GLP-1 analogues also reduce the production of reactive oxygen species, resulting in a further decrease in inflammation. Therefore, it is crucial to understand their therapeutic role in several chronic inflammatory conditions, such as metabolic syndrome, cancers, and hypertension. We assessed a hypertensive obese patient who showed improved blood pressure control, leading to discontinuation of antihypertensive medication after using GLP-1 analogue for weight loss management. This one case is not sufficient to establish causality but may help us understand an important link between GLP-1 analogues and hypertension, as well as, their role in treating morbidly obese patients with hypertension.

# CASE PRESENTATION

A 60-year-old Caucasian male with a known history of hypertension and acute diastolic congestive heart failure, diagnosed on 11th Nov, 2024, presented in the outpatient clinical setting with concerns about excessive weight. His past medical history was also notable for obstructive sleep apnea, bilateral knee osteoarthritis, and hyperlipidemia. The patient was a regular patient with follow-ups for annual wellness visits.

The patient was on furosemide 40 mg tablets once a day and continuous positive airway pressure for his apnea. He was also started on GLP-1 analogue (Wegovy) 0.5 mg/ 0.5 ml subcutaneously once a week, 30 days with three refills, on June 6th, 2023. An echocardiogram was also performed in 2023, which showed a preserved ejection fraction of 55–60%.

On February 6, 2025, the patient presented to the clinic with a high blood pressure reading of 148/82, and he was prescribed spironolactone 50 mg once a day and losartan potassium 25 mg once a day to manage his high blood pressure and acute diastolic heart failure. Despite the regimen, his blood pressures remained elevated. On June 27, 2025, his blood pressure readings were 156/86 and 146/82 after 10 minutes.

The patient was morbidly obese, with a BMI of 62.3 kg/m² documented on June 3, 2025. He had started a GLP-1 analogue for weight loss management. By June 23, 2025 his blood pressure readings showed a decline to 139/82. The patient consistently showed improved blood pressure control at each monthly follow-up. On July 21, 2025, his blood pressure readings ranged from 118–126 mmHg systolic and 73–78 mmHg diastolic.

On Aug 18, 2025, his blood pressure readings showed a further decline, ranging from 109–116/ 73 mmHg, and he was advised to discontinue his antihypertensive medications. In Nov 2025, his blood pressure remained within the normal ranges with a mean of 110/76 mmHg across two different readings, and his BMI was documented to be 53.99 kg/m². The patient’s BMI further decreased to 47.2 kg/m² by January 2026.

Table 1.1

demonstrates the blood pressure readings over a period of a year. The patient's pressure was recorded at each monthly follow-up visit, with dates mentioned (Table 1.1). The readings were recorded after five minutes of rest and without distractions. Additionally, a total of two recordings were taken at least two minutes apart. A mean blood pressure was calculated and corresponding BMI was recorded at each visit. As shown in Table 1.1, over the course of a year, the patient's BMI significantly decreased from 62.84 kg/m² to 47.7 kg/m², with a significant decrease in mean blood pressure from 139/82 to a mean of 113/74. Despite the discontinuation of his antihypertensive medications on August 18, 2025, the patient consistently showed lowered blood pressures.

| Date | Reading 1 | Reading 2 | Mean BP (mmHg) | BMI (kg/m²) |
|---|---|---|---|---|
| 06/23/2025 | 139/81 | 139/84 | 139/82 | 62.84 |
| 07/21/2025 | 126/78 | 118/73 | 122/75 | 60.50 |
| *08/18/2025 | 116/73 | 109/73 | 112/73 | 60.36 |
| 09/12/2025 | 122/79 | 121/78 | 104/63 | 58.02 |
| 10/10/2025 | 103/68 | 106/58 | 117/78 | 55.20 |
| 11/10/2025 | 114/76 | 121/80 | 110/76 | 53.996 |
| 01/07/2026 | 116/79 | 104/73 | 110/76 | 52.54 |
| 06/22/2026 | 96/67 | 107/73 | 101/70 | 42.5 |
| 06/22/2026 | 111/74 | 115/75 | 113/74 | 47.37 |

Table 1.1. Longitudinal blood pressure readings (mmHg), mean blood pressure (mmHg), and corresponding BMI (kg/m²).

* Discontinuation of antihypertensive medications ( furosemide, spironolactone, and losartan potassium)

# STATISTICAL METHODS:

All longitudinal clinical parameters were calculated using descriptive statistics. Continuous variables were presented as mean ± standard deviation (SD), median with interquartile range (IQR), and absolute and relative changes (Δ) from baseline follow-up to final follow-up visit. The trajectory between body mass index and blood pressure in this single patient was assessed using intra individual bivariate correlation with Pearson's correlation coefficient (r) and Spearman's rank correlation (p). Observed measurements taken from one patient, and are not completely independent, resulting in a possible autocorrelation between observed measurements. All values are exploratory, and do not represent the cause and effect relationship. Furthermore, this data is observed in a single patient, therefore, it can not be generalized to the general population. All parameters are calculated to two decimal points.

# RESULTS

The patient's blood pressure improved with significant reduction in the weight and body mass index (BMI). The body mass index decreased from 62.84 kg/m² to 47.37 kg/m², with a total decrease of 15.47 kg/m2 as shown in Fig. 1.1A. The substantial weight loss accounted for a 24.6% decrease in BMI. The average BMI across all visits was 54.81 kg/m² ± 6.68 kg/m², with a median of 55.20 kg/m² and interquartile range (IQR) of 52.54–60.36 kg/m². Starting systolic blood pressure was 139 mmHG while the patient was on antihypertensive medications, which decreased to 113mmHg at the final follow-up visit, resulting in an absolute decrease of 26 mmHg and the 18.7% decrease in the systolic blood pressures, shown in Fig. 1.1A. The mean systolic blood pressure was 114.22 mmHg ± 11.20 mmHg, and the median systolic blood pressure was 112 mmHg with an interquartile range (IQR) of 107–117 mmHg. Additionally, the patient also showed improved diastolic blood pressure starting from 82 mmHg to 74 mmHg at the final visit, with an absolute decrease of 8mmHg, resulting in 9.8% decrease in diastolic blood pressure. The average diastolic blood pressure was 74.11 mmHg ± 5.37 mmHg, with a median of 75 mmHg and interquartile range of 73–76 mmHg. Figure 1.1B demonstrates the strong correlation (r = 0.94) between systolic blood pressure and BMI. As the patient's BMI decreased at each follow-up visit, there was a decrease in systolic blood pressure as well. This graph is plotted using one single patient and is not sufficient to develop a casual relationship between systolic blood pressure and BMI, but it highlights the potential effects of BMI in

decreasing the systolic blood pressure.

# DISCUSSION

The patient showed a noticeable positive correlation between the blood pressure measurements and the use of GLP-1 analogue for weight loss. The patient's blood pressure decreased with the decrease in the BMI. GLP-1 analogues are associated with lower systolic and diastolic blood pressures and total cholesterol. They were also associated with an average weight loss of 2.9 kgs compared to placebo [10]. Obesity has become a global concern as it reaches pandemic level with thrice an increase in prevalence [11]. The graph demonstrates a positive correlation between BMI and systolic and diastolic blood pressure readings. At higher BMI values, the patient had high systolic and diastolic blood pressure despite being on furosemide, losartan potassium, and spironolactone. As patients started losing weight, a decrease in BMI was associated with a downward trend in blood pressure readings. Chronic elevation of leptin in severe obesity leads to high blood pressure, which can be abolished by complete blockade of alpha and beta adrenergic receptors, indicating that high blood pressure may possibly be due to SANS activation [12].

## Anti-inflammatory Effects of GLP-1 Analogues

There are several mechanisms that lead to decreased blood pressure readings as the patient's BMI decreases on a GLP-1 analogue. Obese and overweight individuals have shown disruption in normal

levels of inflammatory markers, such as TNF- alpha (TNF-$\alpha$), interleukin-6 (IL-6), and C reactive protein (CRP) [13]. Substantial weight loss reduced the inflammatory burden by reducing the amount of IL-6 and TNF-alpha (TNF-$\alpha$) factors released. Inflammation leads to formation of reactive oxygen species (ROS). ROS stimulates NF-κB pathway and reduces nitric oxide (NO) bioavailability [14]. Tumor necrosis factor destabilizes the endothelial nitric oxide synthase and reduces the amount of NO, which causes vasoconstriction [15]. Therefore, decrease in inflammatory burden, reduces the oxidative stress and improves nitric oxide bioavailability, leading to vasodilation and subsequently lowering blood pressure.

## Reduced Activation of Sympathetic Nervous System and Renin Angiotensin Aldosterone System

There is decreased activity of the sympathetic nervous system. A lower number of fat cells may reduce the abnormal release of adipokines from the adipose tissue and lead to reduced SANS activation, which results in a lower heart rate, decreased cardiac output, and reduced renal tubular sodium reabsorption. All of these factors contribute to decreased blood pressure. Reduced activation of the sympathetic nervous system also reduces vasoconstriction. SANS and RAAS are activated reciprocally [7]. The reduced SANS activation leads to a decrease in renin release. Inhibition of RAAS results in vasodilation and reduced renal tubular sodium reabsorption, contributing to lower blood pressure. Central obesity, excessive fat around internal organs, is strongly associated with metabolic syndrome. Decrease in BMI also reduces visceral obesity and may decompress the renal parenchyma, contributing to return to a normotensive state.

# Role of NF-κB Pathway

NF-κB pathway plays a crucial role in regulating inflammation by releasing pro-inflammatory cytokines, such as interleukin-1 and tumor necrosis factor-alpha. GLP-1 analogues act as anti-inflammatory agents by inhibiting NF-κB pathway, inhibiting release of the pro-inflammatory cytokines [16]. This anti-inflammatory effect of GLP-1 analogues, and reduced inflammation may have an association for reducing blood pressure.

The prevalence of hypertension is more than twice in obese individuals [17]. This case highlights the association between BMI and blood pressure, and possible anti-inflammatory effects of GLP-1 analogues in morbidly obese patients with hypertension and provide a basis for further investigation into the management of hypertension in morbidly obese patients receiving GLP-1 analogues.

## CONCLUSION

In this patient, substantial weight loss, and a reduction of BMI from 62.84 kg/m² to 47.37 kg/m² on GLP-1 analogue reduced the blood pressure significantly from a mean of 139/82 while being on furosemide 40mg, spironolactone 50 mg, and losartan potassium 25 mg to a mean of 113/74 after discontinuation of all three antihypertensives. The reduction in blood pressure after losing significant weight on GLP-1

analogues highlights the potential therapeutic effects of GLP-1 analogues in treating hypertension in morbidly obese patients, however, additional evidence and data is required to develop cause and effect.

## Declarations

The patient consented to participate and publish their clinical case.

## References


1. Ellulu MS, Patimah I, Khaza'ai H, Rahmat A, Abed Y (2017) Obesity and inflammation: the linking mechanism and the complications. Arch Med Sci 13(4):851–863. 10.5114/aoms.2016.58928
2. El Meouchy P, Wahoud M, Allam S, Chedid R, Karam W, Karam S (2022) Hypertension related to obesity: pathogenesis, characteristics and factors for control. Int J Mol Sci 23(20):12305. 10.3390/ijms232012305
3. Banerjee D, Mani A (2025) Obesity's systemic impact: exploring molecular and physiological links to diabetes, cardiovascular disease, and heart failure. Front Endocrinol (Lausanne) 16:1681766. 10.3389/fendo.2025.1681766
4. Ahmed SK, Mohammed RA (2025) Obesity: prevalence, causes, consequences, management, preventive strategies and future research directions. Metabol Open 27:100375. 10.1016/j.metop.2025.100375
5. DeCleene NK, Kahn E, Yuan CW et al (2026) US state-level prevalence of adult obesity by race and ethnicity from 1990 to 2022 and forecasted to 2035. JAMA 335(11):975–985. 10.1001/jama.2025.26817
6. Mills KT, Stefanescu A, He J (2020) The global epidemiology of hypertension. Nat Rev Nephrol 16(4):223–237. 10.1038/s41581-019-0244-2
7. Shariq OA, McKenzie TJ (2020) Obesity-related hypertension: a review of pathophysiology, management, and the role of metabolic surgery. Gland Surg 9(1):80–93. 10.21037/gs.2019.12.03
8. Alharbi SH (2024) Anti-inflammatory role of glucagon-like peptide 1 receptor agonists and its clinical implications. Ther Adv Endocrinol Metab 15:20420188231222367. 10.1177/20420188231222367
9. Scragg J, Koutoukidis DA, Dirksen C, Heitmann BL, Jebb SA (2025) The societal implications of using glucagon-like peptide-1 receptor agonists for the treatment of obesity. Med 6(9):100805. 10.1016/j.medj.2025.100805
10. Collins L, Costello RA (2026) Glucagon-like peptide-1 receptor agonists. In: StatPearls. StatPearls Publishing; Updated February 29, 2024. Accessed August 28, 2026. https://www.ncbi.nlm.nih.gov/books/NBK551568/
11. Parvanova A, Reseghetti E, Abbate M, Ruggenenti P (2024) Mechanisms and treatment of obesity-related hypertension—part 1: mechanisms. Clin Kidney J 17(1):sfad282. 10.1093/ckj/sfad282
12. Hall JE, da Silva AA, do, Carmo JM et al (2010) Obesity-induced hypertension: role of sympathetic nervous system, leptin, and melanocortins. *J Biol Chem*. ;285(23):17271–17276.

10.1074/jbc.R110.113175

13. Khanna D, Khanna S, Khanna P, Kahar P, Patel BM (2022) Obesity: a chronic low-grade inflammation and its markers. Cureus 14(2):e22711. 10.7759/cureus.22711
14. Xiao L, Harrison DG (2020) Inflammation in hypertension. Can J Cardiol 36(5):635–647. 10.1016/j.cjca.2020.01.013
15. McMaster WG, Kirabo A, Madhur MS, Harrison DG (2015) Inflammation, immunity, and hypertensive end-organ damage. Circ Res 116(6):1022–1033. 10.1161/CIRCRESAHA.116.303697
16. The nuclear factor NF-kappaB pathway in inflammation (2009) Cold Spring Harb Perspect Biol 1(6):a001651. 10.1101/cshperspect.a001651
17. Lam BCC, Lim AYL, Chan SL, Yum MPS, Koh NSY, Finkelstein EA (2023) The impact of obesity: a narrative review. Singap Med J 64(3):163–171. 10.4103/singaporemedj.SMJ-2022-232

# Figures

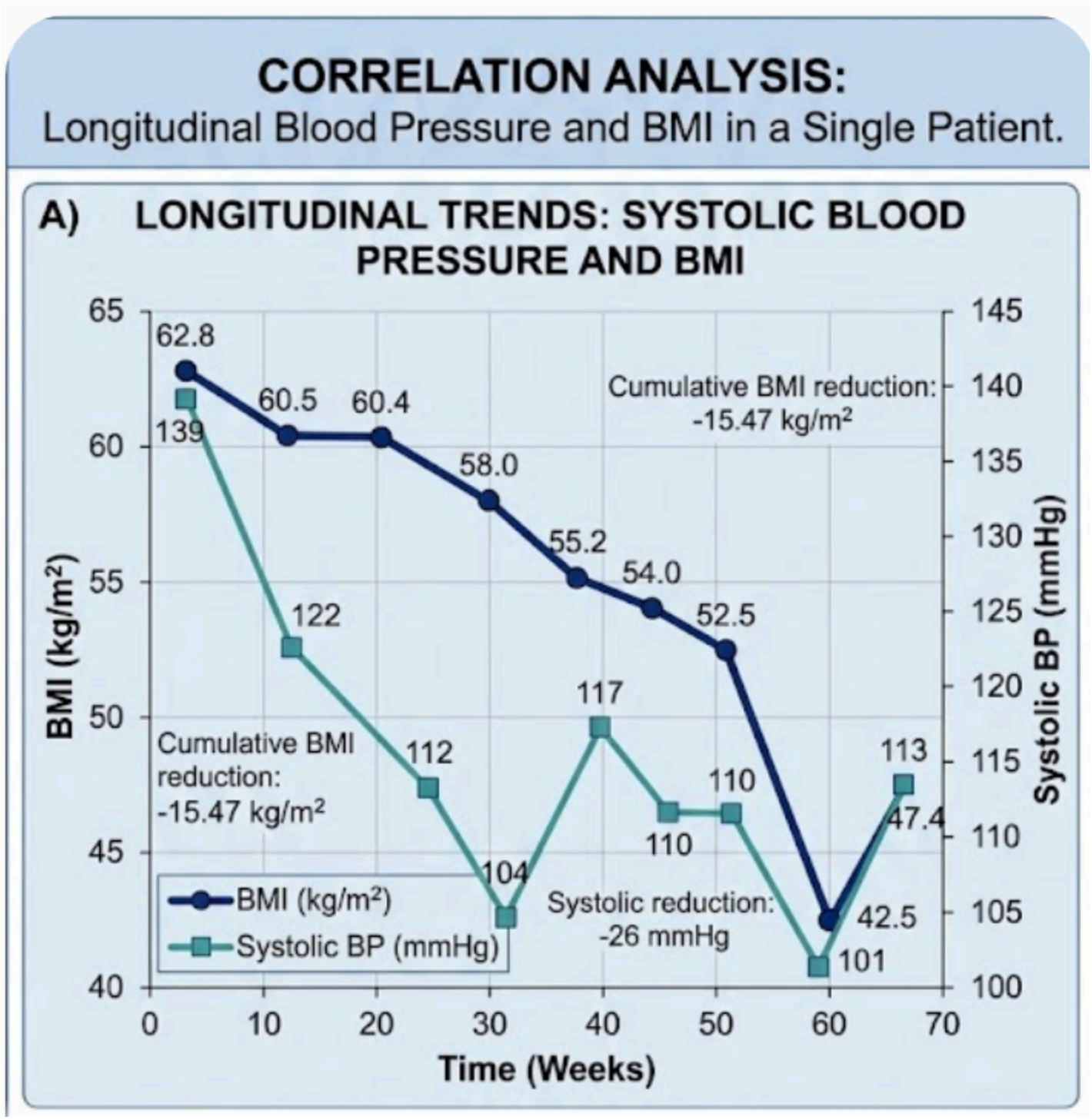



**Figure 1**

Figure 1.1A: Longitudinal trends in systolic blood pressure and BMI

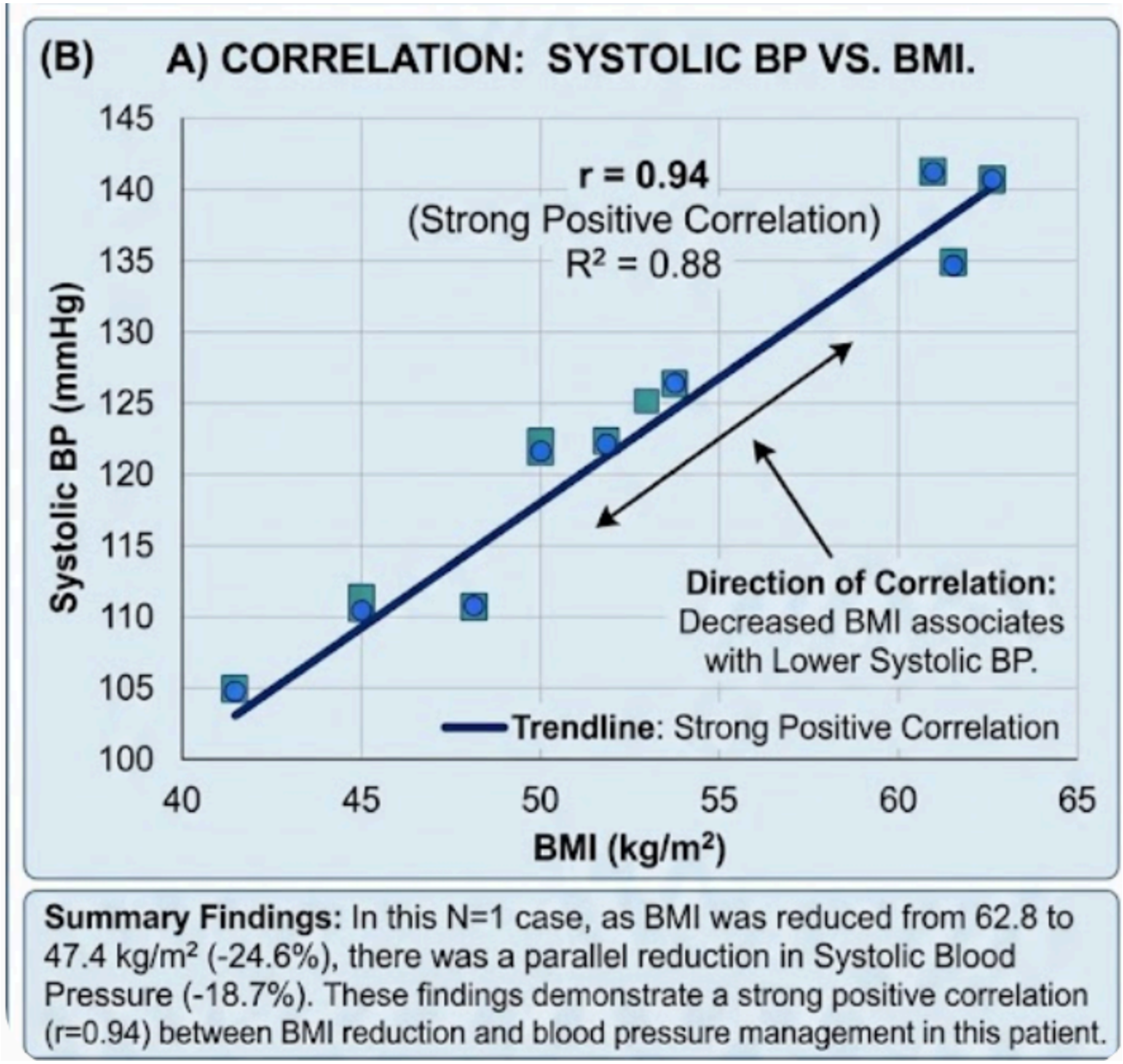



**Figure 2**

Figure 1.1B: Linear regression analysis between systolic blood pressure and BMI