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Efficacy comparison of Captopril with Candesartan in hypertension: A systemic review & meta-analysis

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Abstract
Hypertension is one of the most prevalent non-communicable diseases and a major risk factor for cardiovascular morbidity and mortality worldwide. Effective pharmacological management is essential to control blood pressure and prevent complications such as stroke, myocardial infarction, and renal failure. Drugs acting on the renin–angiotensin–aldosterone system (RAAS), including Captopril and Candesartan, are widely used for the treatment of hypertension. However, variations in efficacy, safety, and tolerability between these agents remain a subject of clinical interest. The present study aimed to compare the efficacy and safety of captopril and candesartan in hypertensive patients through a systematic review and meta-analysis of available clinical studies. A comprehensive literature search was conducted using major electronic databases to identify clinical studies comparing captopril and candesartan in patients with hypertension. Eligible studies were screened according to predefined inclusion and exclusion criteria. Data related to systolic blood pressure (SBP), diastolic blood pressure (DBP), treatment duration, and adverse drug reactions were extracted and analyzed. Quantitative synthesis was performed using meta-analysis methods to evaluate pooled treatment effects.
The findings from the included studies indicated that both captopril and candesartan significantly reduced systolic and diastolic blood pressure in hypertensive patients. However, candesartan demonstrated a comparatively better tolerability profile with fewer adverse effects, particularly a lower incidence of cough commonly associated with ACE inhibitors. Both captopril and candesartan are effective antihypertensive agents. Nevertheless, candesartan may provide improved tolerability and patient compliance, making it a suitable alternative for patients who are intolerant to ACE inhibitors.

Keywords: Alzheimer's disease, Vitamin E, Melatonin, Oxidative stress, Meta-analysis, Neuroprotection

Introduction

Worldwide, hypertension ranks high among non-communicable diseases and is a leading cause of cardiovascular disease-related deaths and complications. Problems like coronary artery disease, stroke, heart failure, and chronic renal disease can develop when arterial blood pressure is consistently high [1]. Hypertension is a major public health concern in today's ageing population, especially in underdeveloped nations with inadequate healthcare infrastructure. Global health systems are facing a significant financial and healthcare burden due to the increasing prevalence of hypertension [2]. Over 1.28 billion adults (those aged 30–79) deal with hypertension, with over two-thirds of that number living in low- and middle-income nations, according to a World Health Organization (WHO) report [3]. Regrettably, over half of all hypertension patients do not know they have the illness, nearly half do not receive a proper diagnosis and therapy, and barely a quarter of those patients manage to keep their blood pressure under control enough [3]. So, hypertension is still a major contributor to early mortality worldwide.

Diastolic and/or systolic blood pressure readings that are consistently high are diagnostic criteria for hypertension, a condition that is frequently seen in primary care settings. Blood pressure readings of 130 mm Hg or higher or 80 mm Hg or lower are considered hypertension according to the guidelines set out by the American College of Cardiology and the American Heart Association (ACC/AHA) [4]. When systolic blood pressure (SBP) hits 140 mm Hg or diastolic blood pressure (DBP) reaches 90 mm Hg, pharmaceutical treatment is usually suggested, especially in people with a higher risk of cardiovascular disease [4]. Hypertension can cause myocardial infarction, stroke, renal failure, and higher mortality rates if it is not treated or not controlled properly. Genetic, environmental, and lifestyle variables all play a role in hypertension's complicated pathogenesis. Genome-wide association studies (GWAS) have found multiple genetic loci linked to hypertension risk and blood pressure regulation [5]. Heritability accounts for around 30–50% of the variation in blood pressure, and SNPs have been linked to pathways that control neurohormonal systems, renal sodium management, and vascular tone [6]. Individual variations in therapeutic response, effectiveness, and tolerance may be at least partially explained by genetic variability, according to pharmacogenetic research [7]. This variability may impact the pharmacokinetics and pharmacodynamics of antihypertensive medications. Medications that target the renin-angiotensin-aldosterone system (RAAS) are among the most important pharmacological classes used to control hypertension. The effectiveness of angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) in lowering blood pressure and preventing cardiovascular

problems has led to their widespread recommendation as first-line treatments [8]. Reducing vasoconstriction and aldosterone release, angiotensin-converting enzyme (ACE) medications work by blocking this conversion. Nevertheless, side symptoms such angioedema and prolonged cough may occur as a consequence of elevated bradykinin levels caused by ACE inhibition [9]. In addition to its cardioprotective and antihypertensive effects, captopril was the first angiotensin-converting enzyme inhibitor to be utilized in clinical practice. Hypertensive, heart failure, and diabetic nephropathy patients see improved cardiovascular outcomes and peripheral vascular resistance reduction as a result [10]. Although ACE inhibition is successful as a treatment, it may not be suitable for long-term use due to the side effects experienced by certain individuals. Candesartan, on the other hand, blocks the AT1 receptor specifically and decreases angiotensin II's activity without influencing bradykinin metabolism [11]. Candesartan lowers blood pressure by preventing vasoconstriction, reducing aldosterone secretion, and promoting vasodilation through inhibiting AT1 receptors in several tissues, including vascular smooth muscle [11]. Patients who experience ACE inhibitor-induced cough or intolerance may be considered for ARBs as an alternative to ACE inhibitors. Furthermore, ARBs are extensively utilized in the treatment of hypertension, heart failure, and diabetic nephropathy, and they have shown to have excellent safety profiles [12]. The safety and effectiveness of angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) may vary, despite the fact that both are extensively used to treat hypertension. Captopril and candesartan have been the subjects of multiple clinical experiments that have sought to determine their antihypertensive effects; nevertheless, the outcomes have been inconsistent and diverse. Hence, in order to understand the relative therapeutic benefits of each, a thorough review of the existing clinical evidence is required.

Therefore, the purpose of this study is to conduct a comprehensive literature search for clinical trials that compare captopril with candesartan in hypertensive patients, and then to use quantitative methods to draw conclusions about the relative effectiveness and safety of the two medications.

The specific objectives of this study are:

1. To evaluate and compare the antihypertensive efficacy of Captopril and Candesartan in patients with hypertension.
2. To assess the magnitude of blood pressure reduction, particularly changes in systolic blood pressure (SBP) and diastolic blood pressure (DBP), following treatment with these medications.
3. To compare the safety profiles of captopril and candesartan, including the incidence of adverse drug reactions reported in clinical studies.
4. To analyze the overall clinical outcomes and tolerability associated with both drugs in hypertensive patients.

Mathodology

Study Criteria

We have collected data for comparing the Captopril, Candesartan with placebo group for Hypertension.

Inclusion criteria

- In our research, we have conducted randomised control trials on patients receiving Captopril with Candesartan.
- Our study has covered patients with Hypertension disease who were taking these medicines.
- It has been preferable to conduct randomised control trials with diverse age groups.
- Captopril with Candesartan have been used in randomised control studies to treat Hypertension, and exercise tests will be performed for both medicines.
- From the year 2000 through 2022, studies were covered.

Exclusion criteria

- We have rule out anything that isn't a randomised control study.
- Any study that does not meet the inclusion requirements have been disqualified.
- We have rule out studies if the patients' conditions aren't satisfactory.

Source of data

We will use the inclusion and exclusion criteria for finding studies using the following keywords on web data resources like "Elsevier, PubMed, MEDLINE, The Cochrane Library, Science Direct." For systemic review and meta-analysis.

Study procedure

The study involves the following steps:

Study site: The study conducted using the inclusion and exclusion criteria for finding studies using the following keywords on web data resources like "Elsevier, PubMed, MEDLINE, The Cochrane Library, Science Direct." For systemic review and meta-analysis.

Study design

We have used the following Keywords list for finding the studies.

Hypertension
Captopril
Candesartan
Randomized control trials on Captopril.
Randomized control trials on Candesartan.

Study period

An observational study commenced for the duration of six months.

Guidelines followed for systemic review:

Prisma guidelines were used for the systemic review process. The same details will be documented electronically in a special design database using excel sheets, Review manager 5.4, and SPSS v26.

Studies enrolment

Studies that fulfilled the inclusion and exclusion criteria will be enrolled in the study.

Heterogeneity

The Mantel-Haentszel chi-square test and the I^2 test were used to evaluate heterogeneity. For the first technique, heterogeneity in the trial data was defined as $P < 0.10$; for the second method, an I^2 value of less than 30% denotes mild heterogeneity, between 30% and 50% moderate

heterogeneity, and more than 50% severe heterogeneity. When there was considerable heterogeneity, an effort was made to explain the variations using the various participant categories and research design variations.

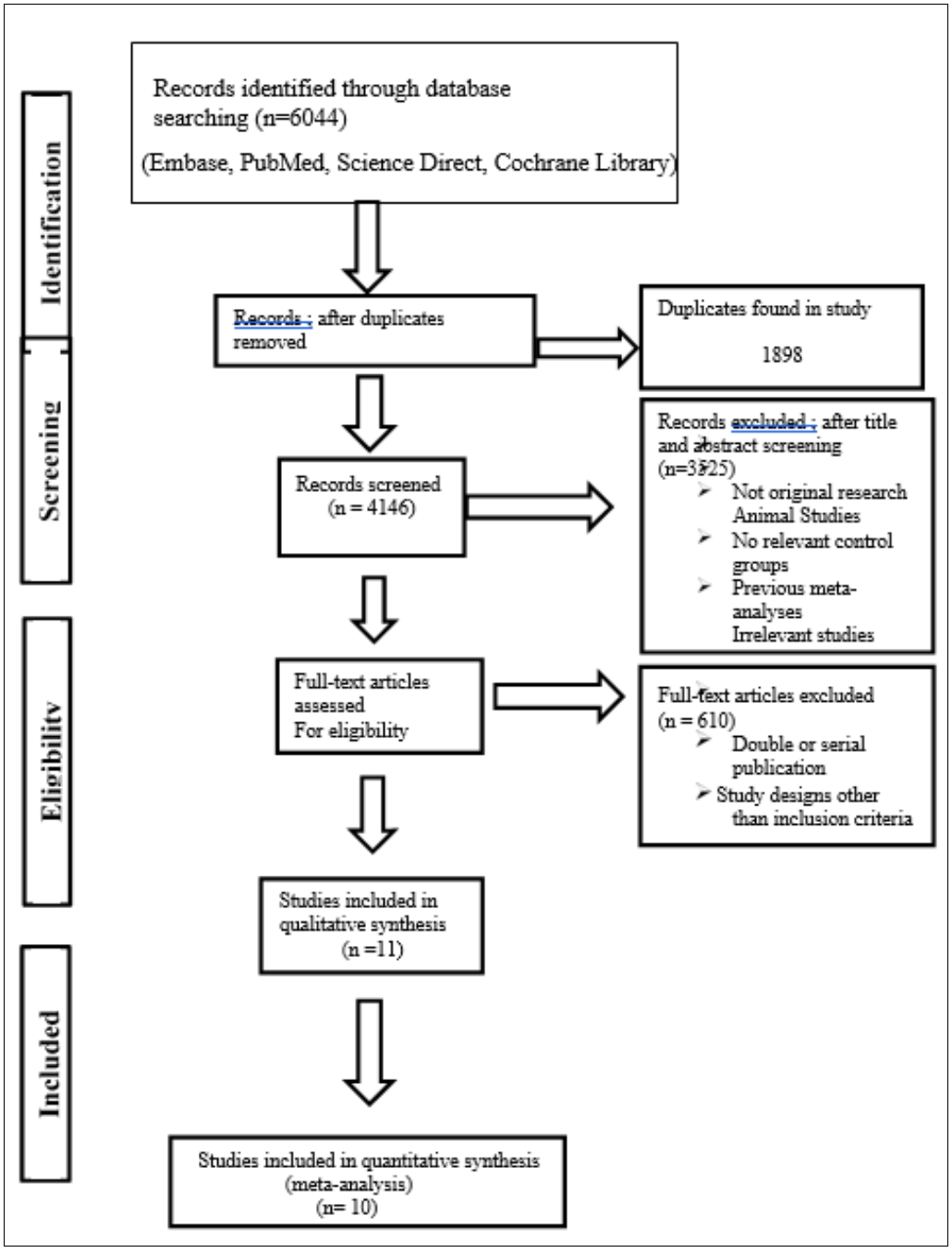

Fig 1: For systematic reviews and meta-analyes (PRISMA) flowchart for study selection

Table 1: Overall study presentation

S. No.	Author	Study Design	Year	Study Site
1	W. Ruzyllo *et al* [13]	A multicentre, double-blind, randomized placebo-controlled trial	2004	Poland
2	Vitale C. *et al* [14]	A randomized double-blind, placebo-controlled trial	2013	Italy
3	Vitale C. *et al* [15]	A randomized double-blind, placebo-controlled trial	2013	Italy
4	Niyazi Güler *et al* [16]	A randomized double-blind, placebo-controlled trial	2003	Turkey
5	Koylan N. *et al* [17]	A multicentre, double-blind comparative study	2004	Turkey
6	Belardinelli R. *et al* [18]	A randomized longitudinal controlled study	2008	Italy
7	Luiz A. *et al* [19]	A randomized double-blind, placebo-controlled trial	2007	Brazil
8	H. Szwed *et al* [20]	A randomized, multicentre, double-blind, placebo-controlled parallel-group study	2001	Poland
9	Roberto Ferrari *et al* [21]	A randomized, double-blind, placebo-controlled, event-driven trial	2020	Italy
10	Stone G. W. *et al* [22]	A randomized double-blind, placebo-controlled trial	2006	Russia

Blood pressure comparison at the peak of exercise in different studies concerning Captopril with Candesartan

For Blood pressure at the peak of exercise data, the following studies were relevant from included studies and given as follows:

Table 2

Studies included in Blood pressure at the peak of exercise Comparison Group 1 Captopril vs Candesartan	
Studies included Captopril	Study included Candesartan
4,5,6,7	1
2	1
3	1

Table 3

Studies included in heart rate at the peak of exercise Comparison Group 2 Candesartan vs placebo	
Studies included Captopril	Study included Candesartan
No data available for comparison	

Table 4

Studies included in heart rate at the peak of exercise Comparison Group 3 Captopril vs Placebo	
Studies included Captopril	Study included Candesartan
4,5,6,7	11
2	11
3	11

Table 5

Studies included in heart rate at the peak of exercise Comparison Group 4 Captopril with Candesartan vs Captopril	
Studies included Captopril	Study included Candesartan
No data available for comparison	

Table 6

Studies included in heart rate at the peak of exercise Comparison Group5 Captopril with Candesartan vs Placebo	
Studies included Captopril	Study included Candesartan
4,5,6,7	1
2	1
3	1

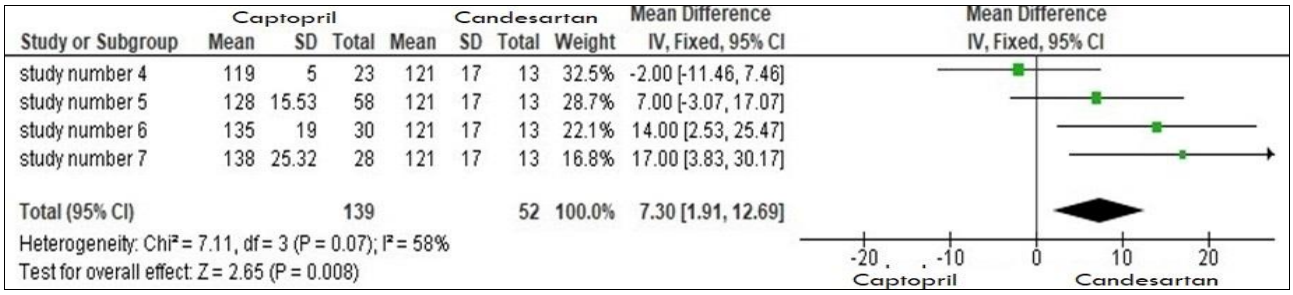

Fig 2: Forest plot Blood Pressure comparison at the peak of exercise group 1

For blood pressure comparison at the peak of exercisegroup 1, the total of 4 studies was enrolled; after adding data, the mean difference found to be 7.30 [1.91,12.69] at 95% confidence interval with $\chi 2$ value 7.11 at the degree of freedom 3 at P-value 0.07 and the test for overall effect Z value 2.65at P-value 0.008.(Fig. 2)

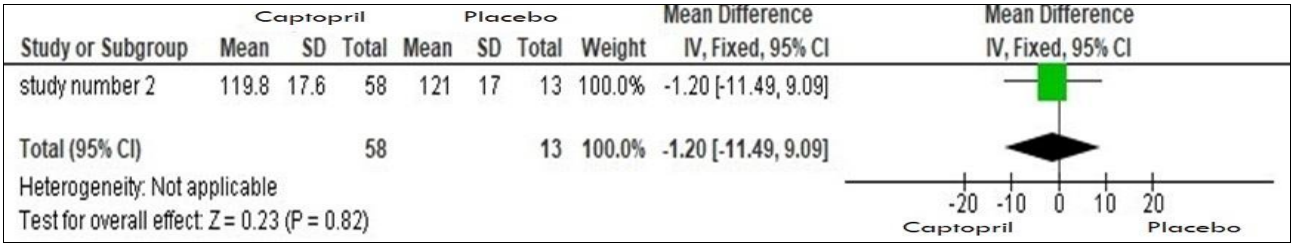

Fig 3: Forest plot Blood pressure comparison at the peak of exercise group 2

For blood pressure comparison at the peak of group 2, the total of 1 study was enrolled; after adding data the mean difference found to be -1.20 [-11.49, 9.09] at 95% confidence interval with the test for overall effect Z value0.23 at P-value 0.82.(Fig. 3)

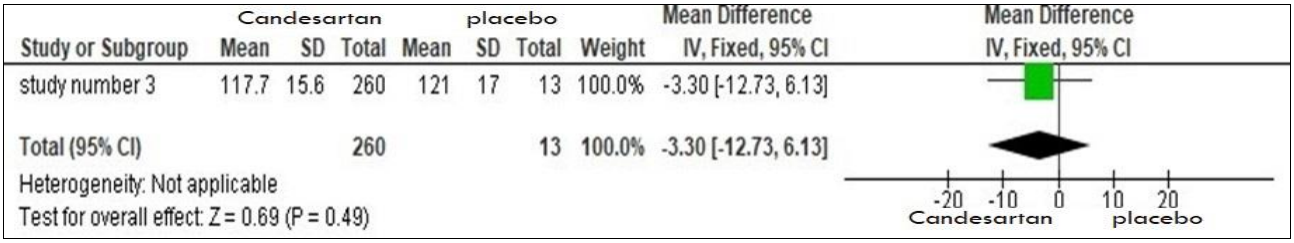

Fig 4: Forest plot Blood pressure comparison at the peak of exercise group 3

For blood pressure comparison at the peak of exercise group 3, the total of 1 study was enrolled; after adding data the mean difference found to be -3.30[-12.73,6.13] at 95% confidence interval with the test for overall effect Z value 0.69 at P-value 0.49. (Fig. 4)

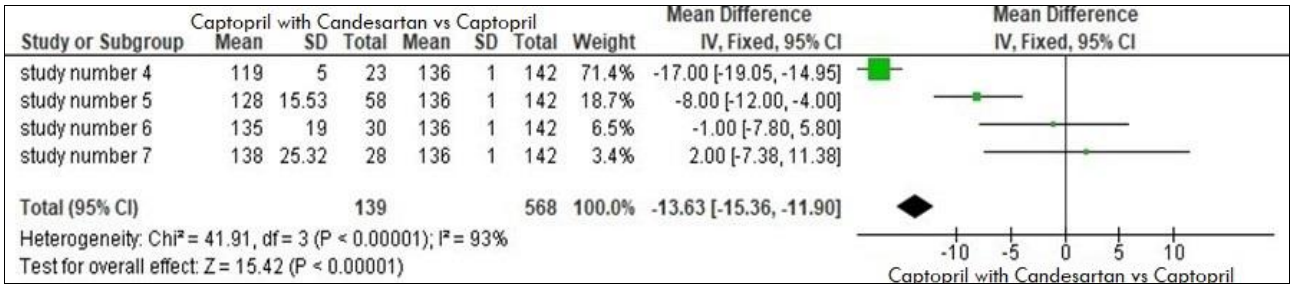

Fig 5: Forest plot Blood pressure comparison at the peak of exercise group 3

For blood pressure comparison at the peak of exercise group 4, a total of 4 studies were enrolled; after adding data, the mean difference was found to be-13.63[-15.36,-11.90] at95% confidence interval with χ2value 41.91 at the degree of freedom 3 at P-value< 0.0001 and the test for overall effect Z value15.42 at P-value<0.0001. (Fig. 5)

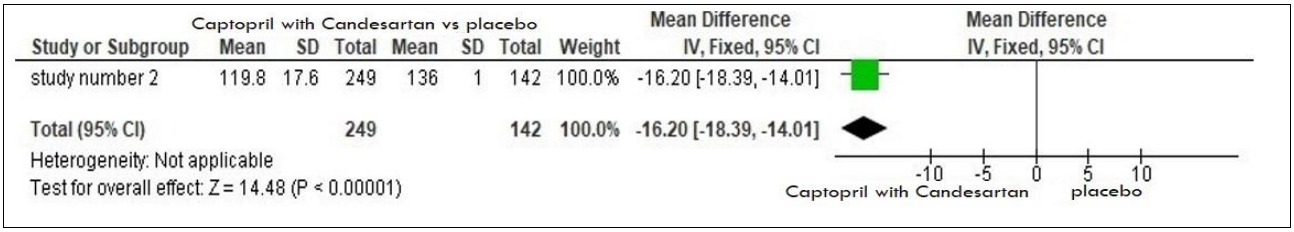

Fig 6: Forest plot Blood pressure comparison at the peak of exercise group 5

For blood pressure comparison at the peak of exercise group 5, the total of 1 study was enrolled; after adding data the mean difference found to be -16.20 [-18.39,-14.01] at 95% confidence interval with the test for overall effect Z value14.48at P-value<0.00001.(Fig. 6)

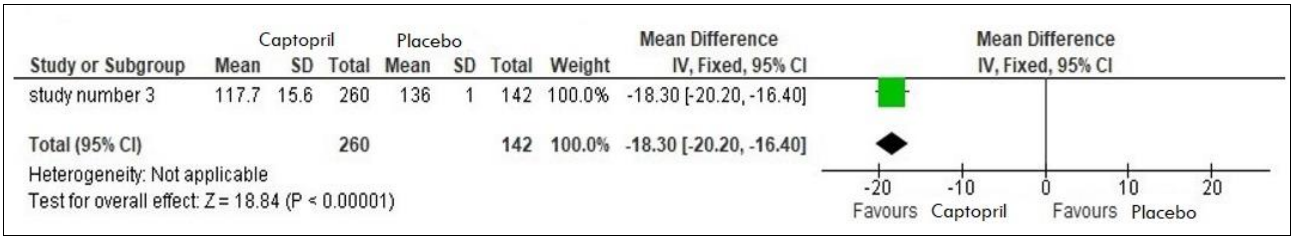

Fig 7: Forest plot of adverse reactions of Captopril vs placebo

For comparing Captopril vs placebo for adverse effects, the total of 1 study was enrolled; after adding data, the mean difference found to be -18.30[-20.20,-16.40] at 95% confidence interval with the test for overall effect Z value 18.84atP-value<0.00001. (Fig. 7)

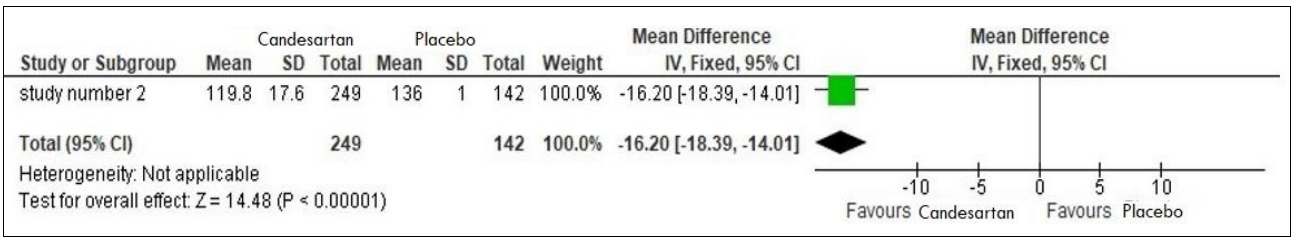

Fig 8: Forest plot of adverse effects of Candesartan vs placebo

For blood pressure comparison of adverse effects of Candesartan vs placebo, the total of 1 study was enrolled; after adding data, the mean difference found to be -16.20[-18.39,-14.01] at 95% confidence interval with the test for overall effect Z value 14.48 at P-value<0.00001. (Fig. 8)

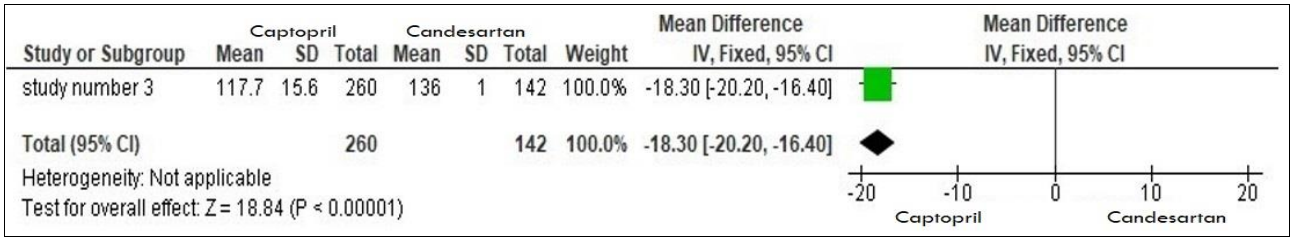

Fig 9: Forest plot of adverse reaction of Captopril vs Candesartan

For comparing of Adverse reaction of Captopril vs Candesartan, the total of 1 study was enrolled; after adding data, the mean difference found to be -18.30[-2020,16.40] at 95% confidence interval with the test for overall effect Z value 18.84 at P-value<0.00001. (Fig.9)

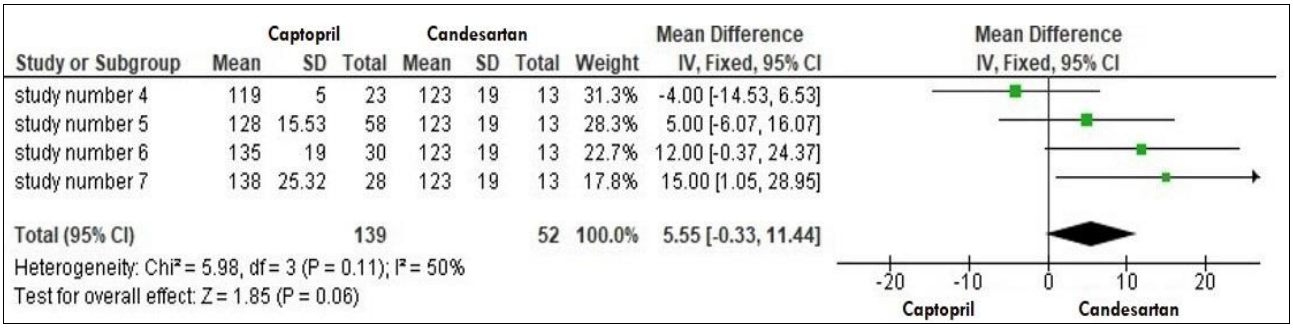

Fig 10: Forest plot of effect of Captopril vs Candesartan in reduction of High Blood Pressure

The effect of Captopril vs Candesartan in reduction of high blood pressure, the total of 4 studies was enrolled after adding data the mean difference found to be 5.55[-0.33,11.44] with χ2value 5.98 at the degree of freedom three at P-value 0.11 at 95% confidence interval and the test for overall effect Z value1.855 at P-value0.06. (Fig. 10)

Discussion & Conclusion

Hypertension remains one of the most significant modifiable risk factors for cardiovascular morbidity and mortality worldwide. Effective control of blood pressure is essential to reduce the risk of complications such as stroke, myocardial infarction, and renal impairment. Pharmacological agents targeting the renin–angiotensin–aldosterone system (RAAS), particularly angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs), are widely used as first-line therapies for the management of hypertension.

The present systematic review and meta-analysis evaluated the comparative efficacy and safety of Captopril and Candesartan in hypertensive patients. The findings of the included studies suggest that both drugs are effective in lowering systolic and diastolic blood pressure. However, differences were observed in their safety profiles and tolerability. Captopril, as an ACE inhibitor, reduces blood pressure by inhibiting the conversion of angiotensin I to angiotensin II, thereby decreasing vasoconstriction and aldosterone secretion. Several clinical trials have demonstrated its effectiveness in reducing blood pressure and improving cardiovascular outcomes in hypertensive patients. Nevertheless, ACE inhibitors are associated with certain adverse effects such as persistent cough and, in rare cases, angioedema due to the accumulation of bradykinin.

In contrast, candesartan, an ARB, selectively blocks angiotensin II type-1 (AT1) receptors, preventing the vasoconstrictive and aldosterone-secreting effects of angiotensin II without affecting bradykinin metabolism. Because of this mechanism, ARBs generally exhibit a better tolerability profile and a lower incidence of cough compared with ACE inhibitors. The studies included in this review indicate that candesartan provides comparable or slightly superior blood pressure reduction with improved tolerability in certain patient populations.

The results of the meta-analysis support the hypothesis that both medications are effective antihypertensive agents; however, candesartan may offer advantages in terms of safety and patient compliance. Improved tolerability may contribute to better long-term adherence to therapy, which is a critical factor in achieving optimal blood pressure control. Despite these findings, several limitations should be considered. The included studies varied in sample size, treatment duration, and study design, which may introduce heterogeneity in the results. In addition, differences in patient characteristics, baseline blood pressure levels, and concomitant medications could influence treatment outcomes. Therefore, further large-scale randomized controlled trials with standardized methodologies are necessary to provide more definitive comparative evidence. Overall, the present systematic review highlights the importance of individualized antihypertensive therapy, taking into account both the efficacy and safety profiles of available medications. In conclusion, the findings of this systematic review and meta-analysis demonstrate that both Captopril and Candesartan are effective in the management of hypertension. Both agents significantly reduce systolic and diastolic blood pressure and play an important role in

preventing cardiovascular complications associated with uncontrolled hypertension.

However, candesartan appears to have a more favorable safety and tolerability profile compared with captopril, particularly with regard to adverse effects such as cough that are commonly associated with ACE inhibitors. This improved tolerability may enhance patient adherence and contribute to better long-term blood pressure control. Therefore, while both drugs remain valuable therapeutic options, candesartan may be considered a preferable alternative in patients who experience intolerance to ACE inhibitors. Further well-designed randomized clinical trials and long-term studies are required to strengthen the evidence regarding the comparative effectiveness and safety of these two antihypertensive agents.

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