Summary of Product Characteristics for Pharmaceutical Products

1. Name of the medicinal product:

TELGOOD 40 CT 6.25
(Telmisartan 40 mg and Chlorthalidone 6.25 mg Tablets)

2. Qualitative and quantitative composition

Each uncoated tablet contains:
Telmisartan USP.....40mg
Chlorthalidone USP.....6.25mg

Excipients of known effects:
Mannitol

For the full list of excipients, see section 6.1

3. Pharmaceutical form

Uncoated tablets

4. Clinical particulars

4.1 Therapeutic indications

- Hypertension
- Kidney problems
- Control the risk of cardiovascular events

4.2 Posology and method of administration

This tablet form of medicine is an amalgamation of two medicines named Telmisartan which is an angiotensin II receptor antagonist and Chlorthalidone which is a thiazide diuretic or water pill. Telmisartan, an angiotensin receptor antagonist, aids in relaxing and widening blood vessels (arteries). A diuretic called chlorthalidone prevents the body from absorbing more salt, which reduces fluid retention. Together, they reduce blood pressure and lower the risk of heart attack, stroke, and edema(fluid retention) in the future.

Telmisartan and Chlorthalidone mg medicine should only be consumed if prescribed by a medical professional as he/she can advise you better on the dose that is suitable for your health condition. The Telmisartan and Chlorthalidone Tablets should be swallowed as a whole with a glass of water, try not to crush, chew, or break it.
Method of administration
Telmisartan tablets are for once-daily oral administration and should be taken with liquid, with or without food.

4.3 Contraindications

The use of this medication is thought to be quite successful in treating hypertension. Despite the fact that the drug is highly recommended by medical experts, using it may, in extremely rare circumstances, have some

negative effects on the body. The adverse effects of this tablet medication aren't anything to be too concerned about because, as your body adjusts to the medication's treatment, they usually go away on their own. However, if you believe that the adverse effects of the medication are beginning to worsen and you are concerned about them, seek the counsel of a medical practitioner. Some of the most common side effects of Telmisartan and Chlorthalidone Tablets may include:

- Back Pain
- Diarrhea
- Increased Uric Acid Level in the Blood
- Dizziness
- Upper respiratory tract infection
- Tiredness

4.4 Special warnings and precautions for use

Some of the precautions and warnings to keep in mind about Telmisartan and Chlorthalidone Tablets are:

•Telmisartan and chlorthalidone have been recommended for the treatment of high blood pressure in you.

•Dehydration may result from taking chlorthalidone and telmisartan. If you have severe thirst, a very dry mouth, or muscle weakness, make sure to drink lots of fluids and let your doctor know.

•Telmisartan 40mg and Chlorthalidone 12.5mg Tablets can be consumed with or without food.

•The intake of this medicine might make you queasy. Move slowly when getting out of a seated or lying down position.

•It is dangerous to take Telmisartan with Chlorthalidone while pregnant or nursing.

•Additionally, the intake of this tablet medicine might lower the chance of heart attacks and strokes.

•Please refrain from taking Telmisartan and Chlorthalidone medicine if you have anuria (the inability to pass urine) since this is contraindicated and could cause further issues

4.5 Interaction with other medicinal products and other forms of interaction

All drugs interact differently for person to person. You should check all the possible interactions with your doctor before starting any medicine.

Interaction with Alcohol

Description

N/A

Instructions

Consumption of alcohol is not recommended during treatment with Telpres CT 40/12.5 Tablet. Together they may increase the blood-pressure-lowering effects. You may experience weakness, confusion, dizziness, fainting, and blurred vision.

Interaction with Medicine

Cisapride
Dexamethasone
Lithium
Amiodarone
Captopril
Insulin

Nonsteroidal anti-inflammatory medicines

Disease interactions

Congestive heart failure

Congestive heart failure is a condition in which your heart does not pump enough blood to meet the demands of your body. Taking Telpres CT 40/12.5 Tablet in this condition may cause decreased urine output, an increase in blood urea nitrogen (BUN) and serum creatinine levels. It may rarely lead to kidney failure and myocardial ischemia (reduced blood flow to the heart). Your doctor will closely monitor your heart function while you are taking this medicine.

Diabetes

Telpres CT 40/12.5 Tabletshould be used with caution if you have Diabetes mellitus because of the increased risk of altered blood sugar levels. Close monitoring of your blood sugar levels is advised while you are taking this medicine.

Hyperuricaemia

Telpres CT 40/12.5 Tablet should be used with caution if you have hyperuricaemia (increased uric acid levels in the blood). This medicine decreases uric acid excretion and increases uric acid in the blood. Your blood uric acid levels will be closely monitored while you are taking this medicine.

Hyperlipidaemia

Telpres CT 40/12.5 Tablet should be used with caution if you have hyperlipidaemia (high levels of fat in the blood) as it may increase your cholesterol and triglyceride levels. Close monitoring of your cholesterol and triglyceride levels is recommended if you are using this medicine.

Food interactions Information not available. **Lab interactions** Information not available.

This is not an exhaustive list of possible drug interactions. You should consult your doctor about all the possible interactions of the drugs you're taking.

4.6 Pregnancy and Lactation

Pregnancy

This tablet is not recommended for use in pregnancy as it may lead to various complications in your foetus. If you are on this medication, consult your doctor before you plan your pregnancy.

Breast-feeding

This tablet is not recommended while breastfeeding as it may pass into your breastmilk. This medicine may decrease milk production and may also dehydrate the infant.

4.7 Effects on ability to drive and use machines

When driving vehicles or operating machinery it should be taken into account that dizziness or drowsiness may occasionally occur when taking antihypertensive therapy such as Telmisartan & Chlorthalidone.

4.8 Undesirable effects

- Stomach pain
- Chills
- Difficult or painful urination
- Fast heartbeat
- Dizziness
- Weakness in arms, hands, legs or feet
- Very low blood pressure
- Constipation
- Diarrhoea
- Nausea
- Vomiting
- Headache
- Blurred vision
- Stomach upset
- Back pain
- Sinus pain

Reporting of suspected adverse reactions: Healthcare professionals are asked to report any suspected adverse reactions via the relevant national regulatory authority pharmacovigilance.

4.9 Overdose

There is limited information available with regard to overdose in humans.

5. Pharmacological properties

5.1 Pharmacodynamic properties

Telmisartan
Pharmacotherapeutic group: Angiotensin II Antagonists, plain,
ATC Code: C09CA07.
Mechanism of action
Telmisartan is an orally active and specific angiotensin II receptor (type AT1) antagonist. Telmisartan displaces angiotensin II with very high affinity from its binding site at the AT1 receptor subtype, which is responsible for the known actions of angiotensin II. Telmisartan does not exhibit any partial agonist activity at the AT1 receptor. Telmisartan selectively binds the AT1

receptor. The binding is long-lasting. Telmisartan does not show affinity for other receptors, including AT2 and other less characterised AT receptors. The functional role of these receptors is not known, nor is the effect of their possible overstimulation by angiotensin II, whose levels are increased by telmisartan. Plasma aldosterone levels are decreased by telmisartan. Telmisartan does not inhibit human plasma renin or block ion channels. Telmisartan does not inhibit angiotensin converting enzyme (kininase II), the enzyme which also degrades bradykinin. Therefore it is not expected to potentiate bradykinin-mediated adverse events.
In human, an 80 mg dose of telmisartan almost completely inhibits the angiotensin II evoked blood pressure increase. The inhibitory effect is maintained over 24 hours and still measurable up to 48 hours.

Clinical efficacy and safety

Treatment of essential hypertension

After the first dose of telmisartan, the antihypertensive activity gradually becomes evident within 3 hours. The maximum reduction in blood pressure is generally attained 4 to 8 weeks after the start of treatment and is sustained during long-term therapy.
The antihypertensive effect persists constantly over 24 hours after dosing and includes the last 4 hours before the next dose as shown by ambulatory blood pressure measurements. This is confirmed by trough to peak ratios consistently above 80 % seen after doses of 80 and 80 mg of telmisartan in placebo controlled clinical studies. There is an apparent trend to a dose relationship to a time to recovery of baseline systolic blood pressure (SBP). In this respect data concerning diastolic blood pressure (DBP) are inconsistent.
In patients with hypertension telmisartan reduces both systolic and diastolic blood pressure without affecting pulse rate. The contribution of the medicinal product's diuretic and natriuretic effect to its hypotensive activity has still to be defined. The antihypertensive efficacy of telmisartan is comparable to that of agents representative of other classes of antihypertensive medicinal products (demonstrated in clinical trials comparing telmisartan to amlodipine, atenolol, enalapril, hydrochlorothiazide, and lisinopril).
Upon abrupt cessation of treatment with telmisartan, blood pressure gradually returns to pre-treatment values over a period of several days without evidence of rebound hypertension.
The incidence of dry cough was significantly lower in patients treated with telmisartan than in those given angiotensin converting enzyme inhibitors in clinical trials directly comparing the two antihypertensive treatments.

Cardiovascular prevention

ONTARGET (ONgoing Telmisartan Alone and in Combination with Ramipril Global Endpoint Trial) compared the effects of telmisartan, ramipril and the combination of telmisartan and ramipril on cardiovascular outcomes in 25620 patients aged 55 years or older with a history of coronary artery disease, stroke, TIA, peripheral arterial disease, or type 2 diabetes mellitus accompanied by evidence of end-organ damage (e.g. retinopathy, left ventricular hypertrophy, macro- or microalbuminuria), which is a population at risk for cardiovascular events.
Patients were randomized to one of the three following treatment groups: telmisartan 80 mg (n = 8542), ramipril 10 mg (n = 8576), or the combination

of telmisartan 80 mg plus ramipril 10 mg (n = 8502), and followed for a mean observation time of 4.5 years.
Telmisartan showed a similar effect to ramipril in reducing the primary composite endpoint of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, or hospitalization for congestive heart failure. The incidence of the primary endpoint was similar in the telmisartan (16.7 %) and ramipril (16.5 %) groups. The hazard ratio for telmisartan vs. ramipril was 1.01 (97.5 % CI 0.93 - 1.10, p (non-inferiority) = 0.0019 at a margin of 1.13). The all-cause mortality rate was 11.6 % and 11.8 % among telmisartan and ramipril treated patients, respectively.
Telmisartan was found to be similarly effective to ramipril in the pre-specified secondary endpoint of cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke [0.99 (97.5 % CI 0.90 - 1.08), p (non-inferiority) = 0.0004], the primary endpoint in the reference study HOPE (The Heart Outcomes Prevention Evaluation Study), which had investigated the effect of ramipril vs. placebo.
TRANSCEND randomized ACE-I intolerant patients with otherwise similar inclusion criteria as ONTARGET to telmisartan 80 mg (n=2954) or placebo (n=2972), both given on top of standard care.
The mean duration of follow up was 4 years and 8 months. No statistically significant difference in the incidence of the primary composite endpoint (cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, or hospitalization for congestive heart failure) was found [15.7 % in the telmisartan and 17.0 % in the placebo groups with a hazard ratio of
0.92 (95 % CI 0.81 - 1.05, p = 0.22)]. There was evidence for a benefit of telmisartan compared to placebo in the pre-specified secondary composite endpoint of cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke [0.87 (95 % CI 0.76 - 1.00, p = 0.048)]. There was no evidence for benefit on cardiovascular mortality (hazard ratio 1.03, 95
% CI 0.85 - 1.24).
Cough and angioedema were less frequently reported in patients treated with telmisartan than in patients treated with ramipril, whereas hypotension was more frequently reported with telmisartan.
Combining telmisartan with ramipril did not add further benefit over ramipril or telmisartan alone. CV mortality and all cause mortality were numerically higher with the combination. In addition, there was a significantly higher incidence of hyperkalaemia, renal failure, hypotension and syncope in the combination arm. Therefore the use of a combination of telmisartan and ramipril is not recommended in this population.
In the "Prevention Regimen For Effectively avoiding Second Strokes" (PRoFESS) trial in patients 50 years and older, who recently experienced stroke, an increased incidence of sepsis was noted for telmisartan compared with placebo, 0.70 % vs. 0.49 % [RR 1.43 (95 % confidence interval 1.00 - 2.06)]; the incidence of fatal sepsis cases was increased for patients taking telmisartan (0.33 %) vs. patients taking placebo (0.16 %) [RR 2.07 (95 % confidence interval 1.14 - 3.76)]. The observed increased occurrence rate of sepsis associated with the use of telmisartan may be either a chance finding or related to a mechanism not currently known.

Two large randomised, controlled trials (ONTARGET (ONgoing Telmisartan Alone and in combination with Ramipril Global Endpoint Trial) and VA NEPHRON-D (The Veterans Affairs Nephropathy in Diabetes)) have examined the use of the combination of an ACE-inhibitor with an angiotensin II receptor blocker.
ONTARGET was a study conducted in patients with a history of cardiovascular or cerebrovascular disease, or type 2 diabetes mellitus accompanied by evidence of end-organ damage. For more detailed information see above under the heading “Cardiovascular prevention”.VA NEPHRON-D was a study in patients with type 2 diabetes mellitus and diabetic nephropathy.
These studies have shown no significant beneficial effect on renal and/or cardiovascular outcomes and mortality, while an increased risk of hyperkalaemia, acute kidney injury and/or hypotension as compared to monotherapy was observed. Given their similar pharmacodynamic properties, these results are also relevant for other ACE-inhibitors and angiotensin II receptor blockers.
ACE-inhibitors and angiotensin II receptor blockers should therefore not be used concomitantly in patients with diabetic nephropathy.
ALTITUDE (Aliskiren Trial in Type 2 Diabetes Using Cardiovascular and Renal Disease Endpoints) was a study designed to test the benefit of adding aliskiren to a standard therapy of an ACE-inhibitor or an angiotensin II receptor blocker in patients with type 2 diabetes mellitus and chronic kidney disease, cardiovascular disease, or both. The study was terminated early because of an increased risk of adverse outcomes. Cardiovascular death and stroke were both numerically more frequent in the aliskiren group than in the placebo group and adverse events and serious adverse events of interest (hyperkalaemia, hypotension and renal dysfunction) were more frequently reported in the aliskiren group than in the placebo group.

Paediatric population

The safety and efficacy of telmisartan in children and adolescents aged below 18 years have not been established.
The blood pressure lowering effects of two doses of telmisartan were assessed in 76 hypertensive, largely overweight patients aged 6 to < 18 years (body weight ≥ 20 kg and ≤ 120 kg, mean 74.6 kg), after taking telmisartan 1 mg/kg (n =29 treated) or 2 mg/kg (n = 31 treated) over a four-week treatment period. By inclusion the presence of secondary hypertension was not investigated. In some of the investigated patients the doses used were higher than those recommended in the treatment of hypertension in the adult population, reaching a daily dose comparable to 160 mg, which was tested in adults. After adjustment for age group effects mean SBP changes from baseline (primary objective) were -14.5 (1.7) mm Hg in the telmisartan 2 mg/kg group, -9.7 (1.7) mm Hg in the telmisartan 1 mg/kg group, and
-6.0 (2.4) in the placebo group. The adjusted DBP changes from baseline were -8.4 (1.5) mm Hg, -4.5 (1.6) mm Hg and -3.5 (2.1) mm Hg respectively. The change was dose dependent. The safety data from this study in patients aged 6 to < 18 years appeared generally similar to that observed in adults. The safety of long term treatment of telmisartan in children and adolescents was not evaluated.

An increase in eosinophils reported in this patient population has not been recorded in adults. Its clinical significance and relevance is unknown.
These clinical data do not allow to make conclusions on the efficacy and safety of telmisartan in hypertensive paediatric population.

Chlorthalidone
Pharmacotherapeutic group: Thiazide-like diuretic
ATC Code: C03BA04
Mechanism of action
Chlorthalidone exerts its therapeutic action by antagonizing sodium-chloride symporter in the distal convoluted tubule of the nephron. It is similar to a thiazide diuretic in its mechanism of action, although it has a mildly altered chemical structure. Both thiazide and thiazide-like diuretics contain a sulfonamide group that also works to inhibit carbonic anhydrase and its antagonistic action at the distal convoluted tubule.
Chlorthalidone inhibits sodium reabsorption at the level of the distal convoluted tubule and thus chloride via inhibition of the Na/Cl symporter. By removing sodium reabsorption at this location, the distal convoluted tubule of the nephron retains a higher sodium content. This lack of reabsorption alters the osmotic gradient and shifts fluid distribution from the outside of the tubule to the inside of the tubule. The increased osmotic load from its increased sodium concentration leads to elevated intratubular volume, thus promoting its diuretic effect. The increased excretion of sodium and extracellular fluid decreases intravascular water and solute concentration. By lowering the intravascular volume and osmotic gradient, the patient has reduced hydrostatic pressure leading to a clinical reduction in blood pressure.

5.2 Pharmacokinetic properties

Telmisartan
Absorption
Absorption of telmisartan is rapid although the amount absorbed varies. The mean absolute bioavailability for telmisartan is about 50 %. When telmisartan is taken with food, the reduction in the area under the plasma concentration-time curve (AUC0-∞) of telmisartan varies from approximately 6 % (80 mg dose) to approximately 19 % (160 mg dose). By 3 hours after administration, plasma concentrations are similar whether telmisartan is taken fasting or with food.
Linearity/non-linearity
The small reduction in AUC is not expected to cause a reduction in the therapeutic efficacy. There is no linear relationship between doses and plasma levels. Cmax and to a lesser extent AUC increase disproportionately at doses above 80 mg.
Distribution
Telmisartan is largely bound to plasma protein (>99.5 %), mainly albumin and alpha-1 acid glycoprotein. The mean steady state apparent volume of distribution (Vdss) is approximately 500 l.
Biotransformation

Telmisartan is metabolised by conjugation to the glucuronide of the parent compound. No pharmacological activity has been shown for the conjugate.

Elimination

Telmisartan is characterised by biexponential decay pharmacokinetics with a terminal elimination half-life of >20 hours. The maximum plasma concentration (Cmax) and, to a smaller extent, the area under the plasma concentration-time curve (AUC), increase disproportionately with dose. There is no evidence of clinically relevant accumulation of telmisartan taken at the recommended dose. Plasma concentrations were higher in females than in males, without relevant influence on efficacy.

After oral (and intravenous) administration, telmisartan is nearly exclusively excreted with the faeces, mainly as unchanged compound. Cumulative urinary excretion is <1 % of dose. Total plasma clearance (Cltot) is high (approximately 1,000 ml/min) compared with hepatic blood flow (about 1,500 ml/min).

Special Populations Paediatric population

The pharmacokinetics of two doses of telmisartan were assessed as a secondary objective in hypertensive patients (n = 57) aged 6 to < 18 years after taking telmisartan 1 mg/kg or 2 mg/kg over a four-week treatment period. Pharmacokinetic objectives included the determination of the steady-state of telmisartan in children and adolescents, and investigation of age related differences. Although the study was too small for a meaningful assessment of the pharmacokinetics of children under 12 years of age, the results are generally consistent with the findings in adults and confirm the non-linearity of telmisartan, particularly for Cmax.

Gender

Differences in plasma concentrations were observed, with Cmax and AUC being approximately 3- and 2-fold higher, respectively, in females compared to males.

Elderly

The pharmacokinetics of telmisartan do not differ between the elderly and those younger than 65 years.

Renal impairment

In patients with mild to moderate and severe renal impairment, doubling of plasma concentrations was observed. However, lower plasma concentrations were observed in patients with renal insufficiency undergoing dialysis. Telmisartan is highly bound to plasma protein in renal-insufficient patients and cannot be removed by dialysis. The elimination half-life is not changed in patients with renal impairment.

Hepatic impairment

Pharmacokinetic studies in patients with hepatic impairment showed an increase in absolute bioavailability up to nearly 100 %. The elimination half-life is not changed in patients with hepatic impairment.

Chlorthalidone

Absorption

Not Available

Volume of distribution

Chlorthalidone has been shown to rapidly concentrate within erythrocytes and subsequently equilibrate via a slow diffusion back into the serum compartment, resulting in a large volume of distribution.

Protein binding

Approximately 75 percent of the drug is bound to plasma proteins, 58 percent of the drug being bound to albumin. This is caused by an increased affinity of the drug to erythrocyte carbonic anhydrase.

Metabolism Liver

Route of elimination

Approximately 50% of the administered dose is excreted unmetabolized through the kidney, and excretion is characterized by biphasic elimination with a rapid phase followed by a slow secretory phase.

5.3 Preclinical safety data

In preclinical safety studies, doses producing exposure comparable to that in the clinical therapeutic range caused reduced red cell parameters (erythrocytes, haemoglobin, haematocrit), changes in renal haemodynamics (increased blood urea nitrogen and creatinine), as well as increased serum potassium in normotensive animals. In dogs, renal tubular dilation and atrophy were observed. Gastric mucosal injury (erosion, ulcers or inflammation) also was noted in rats and dogs. These pharmacologically-mediated undesirable effects, known from preclinical studies with both angiotensin converting enzyme inhibitors and angiotensin II receptor antagonists, were prevented by oral sodium chloride solution supplementation.

In both species, increased plasma renin activity and hypertrophy/hyperplasia of the renal juxtaglomerular cells were observed. These changes, also a class effect of angiotensin converting enzyme inhibitors and other angiotensin II receptor antagonists, do not appear to have clinical significance.

No clear evidence of a teratogenic effect was observed, however at toxic dose levels of telmisartan an effect on the postnatal development of the offsprings such as lower body weight and delayed eye opening was observed.

There was no evidence of mutagenicity and relevant clastogenic activity in in vitro studies and no evidence of carcinogenicity in rats and mice.

6. Pharmaceutical Particulars

6.1 List of Excipients

Ingredient	Specification
Mannitol DC	**BP**
Microcrystalline Cellulose	**BP**
Meglumine	**BP**

Pvp-K 30	**BP**
Cross Povidone	**BP**
Aspartame	**BP**
Magnesium Stearate	**BP**

6.2 Incompatibilities
None

6.3 Shelf-Life
36 months

6.4 Special Precautions for storage
Store in Cool and Dry Place. Protect from direct sunlight. Store below 30°C.

6.5 Nature and Content of container
10 tablets pack in an alu blister such 3-blister pack in 1 mono carton with insert.

6.6 Special precautions for disposal and other handling
As the product contains benzalkonium chloride, soft contact lenses should not be worn during treatment.

7. Marketing Authorization Holder
ZAIN PHARMA LTD.
Plot No: 209/13741, Colchester Park,
Go-Down No.1, 2, 3, Off Mombasa Road, Behind Nice And Lovely House,
P.O. Box: 100167-00101, Nairobi, Kenya

8. Marketing Authorization Number

H2025/CTD11876/25331

9. Date of first authorization/renewal of the authorization

2025 September 15

10. Date of revision of the text

2025 September 15