SUMMARY OF PRODUCT CHARACTERISTICS

1. NAME OF THE MEDICINAL PRODUCT
BEVIT-B12 film coated tablets

2. QUALITATIVE AND QUANTITATIVE COMPOSITION
Each tablet contains

Active substance(s):

Pyridoxine hydrochloride (vitamin B6)	250 mg
Thiamine hydrochloride (vitamin B1)	250 mg
Cyanocobalamin (vitamin B12)	1 mg

Excipient(s):

Lactose monohydrate	17,42 mg (derived from cow's milk.)
Carmoisine lacquer (E 122)	0,092 mg
Ponso 4R lacquer (E 124)	0,037 mg

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM
Film coated tablets

Pink film-coated, round tablets.

4. CLINICAL PARTICULARS
4.1. Therapeutic indications
- in patients with risk factors for vitamin B1, B6 and B12 deficiency,
- in patients with general preclinical complaints such as mental and physical fatigue, forgetfulness, irritability, weakness, tremor,
- in cases of neuritis, polyneuritis, diabetic neuropathy, neuralgia, shingles, tremor,
- in cases of painful conditions such as arthritis, periarthritis, sciatica pain, lumbalgia, arthralgia, myalgia, cramps and rheumatic pain,
- as an adjunct to the treatment of cardiomyopathy, postoperative vomiting, radiation sickness, febrile rheumatism and chronic intoxications, especially during alcoholism.

4.2. Posology and method of administration
Posology/frequency of administration and duration of the treatment:
For children over 12 years of age and adults, unless recommended otherwise by the doctor: One film-coated tablet per day.
The product is usually prescribed for one to several weeks. In some cases, the doctor may extend the treatment for a few more months.

Method of administration:
Film-coated tablets should be taken orally and swallowed whole with liquid.

Additional information for special populations:
Renal/Hepatic failure:

It is contraindicated in patients with renal or hepatic insufficiency as it contains high doses of B vitamins (see section 4.3).

Pediatric population:
It is contraindicated in children under 12 years of age, as it contains high doses of B vitamins (see section 4.3)

Geriatric population:
There is no specific dosage recommendation.

Other:
It is contraindicated in children under 12 years of age, as it contains high doses of B vitamins (see section 4.3).

4.3. Contraindications

- The use of Bevit-B12 is contraindicated in patients with known hypersensitivity to any of the ingredients of the medicine.
- Since it contains high doses of vitamin B6, the product is contraindicated in the following situations:
  - Pregnancy and lactation
  - Children under the age of 12
  - Patients with renal or hepatic insufficiency

4.4. Special warnings and precautions for use

The recommended dose and duration of treatment should not be exceeded.

Because it contains high levels of vitamin B6 (pyridoxine hydrochloride), the product should not be taken at higher dose levels or for longer than recommended. If vitamin B6 (pyridoxine hydrochloride) is not taken as recommended (risk of overdose, see section "Overdose and treatment"), severe neurotoxicity may occur.

Vitamin B6 accelerates the degradation of levodopa given for therapeutic purposes and reduces its effect. Therefore, in patients treated with levodopa, vitamin B6 should not be used more than a few times the daily dose of 2 mg. This interaction does not occur when the patient is given a peripheral decarboxylase inhibitor or a combination of levodopa and a peripheral decarboxylase inhibitor.

Vitamin B 12 is not recommended to be used in patients with Leber disease as it may increase the risk of optic atrophy.

Hypokalemia, thrombocytosis and sudden death may occur when intensive treatment with vitamin B 12 is used in patients with severe megaloblastic anemia.

In case of decreased vitamin B12 concentration or abnormal decrease in concentration with maximum dose intake, it may cause irreversible neurological damage if inadequate treatment is taken for more than 3 months.

Folate deficiency has not been shown, but may compromise the therapeutic response.

Since this medicinal product contains lactose, patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.
This medicinal product may cause allergic reactions as it contains carmoizine lacquer (E 122) and ponso 4R lacquer (E 124).

4.5. Interaction with other medicinal products and other forms of interaction

Vitamin B1 (thiamine):

- Thiosemicarbazone and 5-fluorouracil inhibit thiamine activity.
- Antacids inhibit thiamine absorption.

Laboratory test interferences

- Thiamine may cause false positive results in the determination of urobilinogen using Ehrlich's reagent.
- High doses of thiamine may interfere with the spectrophotometric determination of serum theophylline concentrations.

Vitamin B6 (pyridoxine):

Various medicines interact with pyridoxine and may result in lower levels of pyridoxine. These medicines include:

- Cycloserine
- Hydralazines
- Isoniazid
- Desoxypiridoxine
- D-penicillamine
- Oral contraceptives
- Alcohol

High doses of vitamin B6 prevent the effect of levodopa (See section Special warnings and precautions for use).

Vitamin B12:

Excessive alcohol intake for more than two weeks or its combination with aminosalicylates or colchicine, especially aminoglycosides, histamine (H2) receptor antagonists, metformin and related biguanides may reduce vitamin B12 absorption from the gastrointestinal tract. The need for vitamin B12 increases in patients who receive these treatments.

Antibiotics may affect the microbiological measurement method of serum and red blood cell vitamin B12 concentration and cause false low results.

High and sustained folic acid doses can reduce the concentration of vitamin B12 in the blood.

Ascorbic acid can deplete vitamin B12. Large amounts of vitamin C should be avoided 1 hour after oral intake of vitamin B12.

Additional information for special populations

Pediatric population

No interaction studies have been conducted with B12. The interactions listed above are based on bibliographic data.

BEVIT-B12 is also contraindicated for children under 12 years of age.

4.6. Fertility, pregnancy and lactation

General advice:

Pregnancy Category: X

Women of childbearing potential/Birth control (contraception):

It is contraindicated during pregnancy. Women of childbearing potential must use effective contraception during treatment.

Vitamins B1, B6 and / or B12 have no effect on hormonal birth control methods. However, there is no study on other control methods.

Pregnancy:
This medicinal product is contraindicated for use during pregnancy as it contains high doses of vitamin B6, which greatly exceeds the Recommended Daily Intake.

Breast-feeding:
This medicinal product is contraindicated for use during pregnancy as it contains high doses of vitamin B6, which greatly exceeds the Recommended Daily Intake.

Reproductive ability / fertility:
There is no fertility study.

4.7. Effects on ability to drive and use machines
There is no reported effect on the ability to drive and use machines.

4.8. Undesirable effects

Evaluation of undesirable effects is made on the basis of the following frequencies:

Very common (≥1/10); common (≥1/100 to <1/10); not common (≥1/1.000 to <1/100); rare (≥1/10.000 to <1/1.000); very rare (<1/10.000), not known (can not be estimated based on available data).

The undesirable effects listed are based on spontaneous reports. Therefore, it is not possible to provide frequency information for each.

Immune system disorders
Not known: Allergic reactions may include urticaria, facial edema, wheezing, erythema, rashes and blisters.

Nervous system disorders
Not known: dizziness, headache, peripheral neuropathy, somnolence, paresthesia

Respiratory, thoracic and mediastinal disorders *
Not known: wheezing
***** As an allergic reaction only

Gastrointestinal disorders
Not known: Diarrhea, dyspepsia, nausea, abdominal pain

Skin and subcutaneous tissue disorders
Not known: Rash, erythema

Renal and urinary disorders
Not known: Abnormal urine odor

Investigations
Not known: Aspartate aminotransferase increased, blood folate levels decreased

Reporting of suspected adverse reactions

Reporting suspected adverse reactions after authorization of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via Turkish Pharmacovigilance Center (TÜFAM). (www.titck.gov.tr; e-mail: tufam@titck.gov.tr; tel.: 0 800 314 00 08; fax: 0 312 218 35 99)

4.9. Overdose and treatment

There is no evidence that this product can cause an overdose if used as recommended.

Overdose causes sensory neuropathy and neuropathy syndromes, nausea, headache, paresthesia, somnolence, increased serum AST (SGOT) levels and decreased serum folic acid concentrations. These effects resolve when treatment is stopped.

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: VITAMIN-B COMPLEX (PLAIN)
ATC code: A11DB

Active substances in BEVIT-B12 are vital in cellular energy production, protein and nucleic acid metabolism.

Vitamin B1

Thiamine pyrophosphate (TPP), the coenzyme form of vitamin B1, is involved in two main types of metabolic reactions: decarboxylation of α-keto acids (eg pyruvate, α-ketoglutarate and branched-chain keto acids) and transketolation (eg between hexose and pentose phosphates). Thus, the major physiological role of vitamin B1 is that it serves as a coenzyme in carbohydrate metabolism where TPP is required for several steps in the degradation of glucose to provide energy.

In addition to the metabolic role it plays as a coenzyme, vitamin B1 also plays a role in neurotransmitter function and nerve conduction.

Vitamin B1, in high doses, and especially in combination with vitamins B6 and B12, suppresses neural stimuli transmission and thus can exhibit an analgesic effect.

Early stages of vitamin B1 deficiency may be accompanied by nonspecific symptoms that may be overlooked or easily misinterpreted. Clinical signs of deficiency are anorexia; weight loss; mental changes such as apathy, short-term memory loss, confusion and irritability; muscle weakness; cardiovascular effects such as heart enlargement.

Conditions that frequently accompany marginal vitamin B1 deficiency and require supplementation are regular alcohol consumption, high carbohydrate intake, and intense physical exertion.

The functional consequences of severe vitamin B1 deficiency are cardiac failure, muscle weakness, and peripheral and central neuropathy. Symptoms of clinical beriberi (severe vitamin B1 deficiency) change with age. Dry (paralytic or nervous), wet (cardiac), or cerebral (Wernicke-Korsakoff syndrome) forms of beriberi can be seen in adults. These conditions should be treated immediately with vitamin B1. Severe cases of vitamin B1 deficiency in industrialized countries are likely to be associated with high alcohol consumption in addition to limited food consumption. In such cases, renal and cardiovascular complications are life-threatening.

Vitamin B6

Vitamin B6 is a coenzyme for more than 100 enzymes involved in amino acid and protein metabolism, including aminotransferases, decarboxylases, racemases, and dehydratases. It is a coenzyme both for δ-aminolevulinate synthase, which catalyzes the first step in its biosynthesis, and for cystathionine ß-synthase and cystathioninase enzymes that play a role in the transsulfuration pathway from homocysteine to cysteine. Most of the total vitamin B6 in the body is in the phosphorylase muscle ligament.

The high rate of amino acid production and degradation is an important parameter for the central nervous system to function effectively and efficiently; therefore, a sufficient and appropriate supply of vitamin B6 is required. It plays an important role in the synthesis of biogenic amines and neurotransmitters in the brain. Vitamin B6 is required as a coenzyme for the conversion of glutamic acid to GABA (an inhibitory neurotransmitter in the central nervous system). Vitamin B6 is also required for the conversion of tyrosine to dopamine and noradrenaline, tryptophan to 5-hydroxytryptamine, and histidine to histamine.

The classic clinical symptoms of vitamin B6 deficiency are:

Skin and mucosal lesions such as seborrheic dermatitis, glossitis and buccal erosions. Peripheral neuritis with nerve degeneration causing sensory disturbances, polyneuropathies.

Cerebral convulsions with electroencephalographic abnormalities.

Hypochromic anemia with microcytosis.

Impairment of lymphocyte proliferation and maturation, antibody production and T-cell activities.

Depression and confusion.

Impairment in platelet function and coagulation mechanisms.

Vitamin B6 deficiency is also observed, as many drugs act as pyridoxine antagonists. These drugs include:
-Cycloserine, an antibiotic
-Hydralazines
-Isoniazid, a tuberculostatic
-Desoxipyridoxine, an antimetabolite
-D-penicillamine, a copper-binding agent
-Oral contraceptives
-Alcohol

Other factors that cause a decrease in vitamin B6 are diseases and pathological conditions such as asthma, diabetes, renal disorders, heart disease and breast cancer. A number of conditions are treated with higher doses of vitamin B6 (premenstrual syndrome, carpal tunnel syndrome, depressions and diabetic neuropathy).

Vitamin B6 in concentrations between 30-100 mg / day is often prescribed as first-line treatment of nausea and vomiting in pregnancy.

Appropriate doses of vitamin B6 also protect patients against the side effects of radiation therapy.

Vitamin B12 (cobalamin):

Vitamin B12 is a cofactor for two enzymes: Methionine synthase (an essential reaction for tetrahydrofolic acid regeneration) and L-methylmalonyl-CoA mutase. Methionine synthase

requires methylcobalamine as a cofactor for methyl transfer from methyltetrahydrofolate to homocysteine, which ultimately consists of methionine and tetrahydrofolate. L-Methylmalonyl-CoA mutase requires adenosylcobalamin to eventually convert L-methylmalonyl-CoA to succinyl-CoA in an isomerization reaction. In B12 deficiency, folate may accumulate in the serum as a result of the B12-dependent methyltransferase slowdown. It is important to provide a suitable and sufficient supply of B12 for normal blood formation and neurological function. Vitamin B12 is a cofactor for catechol-O-methyl transferase, which plays an important role in the breakdown of catecholamines, namely noradrenaline and dopamine, in the synaptic space.

High doses of vitamin B12 (in combination with vitamins B1 and B6) have an analgesic effect.

The major cause of clinically observable B12 deficiency is pernicious anemia. The haematological effects of B12 are cold skin associated with a gradual onset of common anemia symptoms such as decreased energy and exercise tolerance, fatigue, shortness of breath, and palpitations. The underlying mechanism of anemia is an interference with normal deoxyribonucleic acid (DNA) synthesis. Hematological complications resolve completely with treatment with B12 supplements.

75-90% of patients with clinically observable B12 deficiency have neurological complications, and only about 25% of cases may be clinical manifestations of B12 deficiency. Sensory disturbances (tingling and numbness) in the extremities (more in the lower extremities) are included in the neurological symptoms. Vibration and position senses are particularly affected. Motor impairments occur, including gait abnormalities. Cognitive changes can occur, with or without mood changes, ranging from loss of concentration to memory loss, disorientation, and distinct dementia. In addition, visual disturbances, insomnia, impotence, and impaired bowel and bladder control may occur. Although the progression of neurological symptoms varies, it usually occurs gradually. Whether neurological complications improve after treatment depends on the duration of these complications.

Caution should be exercised in patients at risk of vitamin B12 deficiency:

- Older people
- Vegans and vegetarians
- HIV positive patients
- Patients with gastrointestinal diseases
- Patients with autoimmunity or a family history of pernicious anemia

Bevit-B12 film-coated tablet, which is a combination of vitamins B1, B6 and B12 in pharmacological doses, shows analgic, antinuritic, detoxifying and antianemic properties. These vitamins, which are found together in nature, complement each other in terms of their functions on the metabolism of cells, especially the cells of the nervous system. Consequently, Bevit-B12 shows an effect much more than the effect that can be achieved by using these vitamins individually.

5.2 Pharmacokinetic properties

General characteristics

Vitamin B1 (Thiamine):

Absorption:

Vitamin B1 is rapidly absorbed in humans, mostly in the proximal small intestine. There are two mechanisms, one through carrier-based transport at low physiological concentrations (<2

μm), and the other through passive diffusion at higher concentrations. Absorption is generally high, but intestinal absorption in humans is rate-limited.

The need for vitamin B1 is directly related to the intake of carbohydrates: 0.5 mg per 1,000 calories. High calorie and especially high carbohydrate food intake increases thiamine requirement.

Distribution:

The total average amount of vitamin B1 in adults is about 30 mg. It is most commonly found in the heart (0.28-0.79 mg per 100 g), followed by kidney (0.24-0.58), liver (0.20-0.76), and brain (0.14-0.44). The level of vitamin B1 in the spinal cord and brain is approximately twice that in the peripheral nerves. Vitamin B1 in complete blood ranges from 5 - 12 μg / 100 mL, and 90% is found in red blood cells and leukocytes. The concentration found in leukocytes is 10 times higher than that in red blood cells. The rate of production and breakdown of vitamin B1 in the body is relatively higher and is never stored in large amounts in tissues. Therefore, it must be continuously supplied. Insufficient intake can cause signs of biochemical and subsequent clinical deficiency. When intake of vitamin B1 is about 60 μg per 100 g of body weight (or 42 mg per 70 kg) and when total body vitamin B1 is 2 μg / g (or 140 mg per 70 kg), a plateau level is reached in most tissues.

There are two other mechanisms involved in the transfer of vitamin B1 within the blood-brain barrier. However, the saturable mechanism in the blood-brain barrier is different from the energy-dependent mechanism in the intestine and the active transport system seen in cerebral cortex cells, which is based on membrane-bound phosphatases.

Biotransformation:
Oral (or parenteral) thiamine rapidly converts in tissues to dysphosphate and, to a lesser extent, triphosphate esters. All vitamin B1 in excess of tissue needs, binding and storage capacity is rapidly excreted in the urine. In rats, the parenteral intake of thiamine of 10 μg / 100 mg body weight (or 7 mg per 70 kg) has been proven to be sufficient for proliferation but less than normal tissue levels. Stimulation of the nerves causes the release of thiamine or monophosphate with a simultaneous decrease in tri- and diphosphatases.

Elimination:
Vitamin B1 is excreted in the urine. In humans, when oral doses higher than 2.5 mg are administered, there is a small increase in urinary vitamin B1 excretion. The half-life of vitamin B1 in the body is 10-20 days. In addition to free vitamin B1 and small amounts of thiamine diphosphate, thiocram, and thiamine disulfide, approximately 20 or more vitamin B1 metabolites have been reported in the urine of rats and humans, but only six of them were actually determined and identified. The ratio of metabolites compared to vitamin B1 excreted increases with decreasing vitamin B1 intake.

Vitamin B6 (pyridoxine):
Absorption:
Various dietary forms of vitamin B6 are absorbed by intestinal mucosal cells through passive diffusion-dependent phosphorylation, especially in the jejenum and ileum (intestine), that is, their capacity is high.

Distribution:
The B6 forms are transformed into pyridoxal phosphate (PLP) and pyridoxamine phosphate (PMP) in liver, erythrocytes, and other tissues. These compounds are distributed in animal tissues, but none are stored. Most of the vitamin B6 in the body is found in phosphorylase, the

enzyme that converts glycogen into glucose-1-phosphate. About half of the vitamin B6 in the body can reflect the phosphorylase of skeletal muscle. PLP can be found in plasma as a PLP-albumin complex and in erythrocytes in conjunction with hemaglobin. The concentration of PL in the erythrocyte is four to five times the concentration in plasma.

Biotransformation:
PLP and PMP, in particular, act as coenzymes in transamination reactions. In particular, PLP acts as a cofactor for many enzymes involved in the synthesis or catabolism of amino acids. PLP is also involved in the decarboxylation and racemization of A-amino acids, other metabolic transformations of amino acids, and the metabolism of lipid and nucleic acids. It is also the basic coenzyme for glycogen phosphorylase. (IOM Vitamin B6, 1998). Pyridoxal phosphate is also required for the synthesis of δ-aminolevulinic acid, which is a precursor of heme.

Elimination:
Normally, the major excretion product is 4-pyridoxic acid; this corresponds to about half of the B6 compounds in urine. With higher doses of vitamin B6, the proportion of other forms of vitamin B6 also increases. At very high pyridoxine doses, most of the dose is excreted unchanged in the urine. B6 is probably also excreted in the faeces in limited quantities, but is difficult to quantify due to microbial synthesis of B6 in the gut.

Vitamin B12 (cobalamin):
Absorption:
As mentioned above, the terms vitamin B12 and cobalamin refer to all members of a group of high cobalt-containing compounds (corrinoids). Corrinoids can be converted to two cobalamin coenzymes active in human metabolism. Cyanocobalamin is the commercially available form of vitamin B12.

Cobalamins are absorbed by two different mechanisms: an active mechanism (protein-based) and a diffusion-type mechanism. A small amount of vitamin B12 is absorbed through the active process that requires a healthy stomach, intrinsic factor (a glycoprotein secreted by the parietal cells of the stomach after stimulation with food), pancreatic competence and terminal ileum with normal functioning. In the stomach, food-bound B12 is broken down from proteins due to acid and pepsin. The released B12 then binds to R proteins (haptocorrins) secreted through the salivary glands and gastric mucosa. In the small intestine, pancreatic proteases partially degrade R proteins and released B12 binds to the intrinsic factor. The resulting intrinsic factor and B12 complex bind to specific receptors in the ileal mucosa; after internalization of the complex, B12 enters the enterocyte. After about 3 to 4 hours, B12 is recirculated. At dose levels up to 10 µg, the efficiency of this mechanism is approximately 50%. At dose levels higher than 10 µg, the efficiency and effectiveness of absorption decrease. The second absorption mechanism occurs by diffusion at a very low rate and shows an efficiency and efficacy corresponding to about 1% and only provides a quantitatively statistically significant result at oral doses exceeding 100 µg.

B12 malabsorption occurs if there is a deficiency in the intrinsic factor (the condition in pernicious anemia). If this condition is not treated, potentially irreversible neurological damage and life-threatening anemia occurs.

Distribution:
Its predominant forms in plasma and tissue are methylcobalamin, adenosylcobalamin and hydroxocobalamin. Methylcobalamin accounts for 60% to 80% of the total plasma cobalamin. In normal humans, cobalamins are found mainly in the liver, where the average amount is 1.5

mg. Each of the kidneys, heart, spleen and brain contain approximately 20-30 µg. Mean values for the total body content calculated for adult humans are 2-5 mg. The pituitary gland is the tissue with the highest concentration per gram of organs / tissues. Adenosylcobalamin is the major cobalamin among all cellular tissues, accounting for about 60-70% in the liver and about 50% in other organs.

Biotransformation:
As vitamin B12 passes through the intestinal mucosa, it is transferred to the plasma transporter protein transcobalamin II, which delivers the vitamin to the cells. The specific biochemical reactions in which cobamide coenzymes play a role are of two types: (1) reaction involving 5-deoxyadenosine covalently attached to the cobalt atom (adenosylcobalamin) and (2) reaction with a methyl group attached to the central cobalt atom (methylcobalamine). Coenzyme methylcobalamin catalyzes a transmethylation from a folic acid cofactor to homocysteine and forms methionine. This reaction releases the unmethylated folate cofactor for other single-carbon transfer reactions which are important for nucleic acid synthesis. The other cobalamin coenzyme, deoxyadenosylcobalamin, catalyzes the conversion of methylmalonyl-coenzyme A to succinyl-coenzyme A, a reaction in the degradation pathway of certain amino acids and single-chain fatty acids.

Vitamin B12 deficiency causes macrocytic, megaloblastic anemia, neurological symptoms due to demyelination of the spinal cord, brain, optic and peripheral nerves, and other less specific symptoms (e.g. tongue pain, weakness). In the absence of anemia, and especially in the elderly, neuropsychiatric symptoms of vitamin B12 deficiency are observed.

Elimination
Urinary, biliary and fecal pathways are the main pathways of excretion. Only unbound plasma cobalamin is available for urinary excretion, and thus urinary excretion through glomerular filtration of free cobalamin is minimal: i.e. varying at levels up to 0.25 µg per day. About 0.5-5 µg of cobalamin per day is secreted into the digestive system, mainly bile; at least 65-75% is reabsorbed in the ileum via the intrinsic factor mechanism. The reversal of this effective B12 enterohepatic circulation, which occurs in the bile and other intestinal secretion channels, does not function in the presence of pernicious anemia due to insufficient intrinsic factor activity. Total loss in the body ranges from 2 to 5 µg per day. Thus, the daily loss of vitamin B12 corresponds to approximately 0.1% of the body pool (in the range of 0.05-0.2%), regardless of the total size.

5.3 Preclinical safety data
There are no specific studies conducted with this product, the preclinical safety of its individual components has been widely documented.

6. PHARMACEUTICAL PARTICULARS
6.1. List of excipients
Tablet:
Lactose monohydrate (Produced from cow's milk.)
Microcrystalline cellulose PH102
Povidone K30
Talc

Coating material:
Hydroxypropyl methylcellulose
Copovidon

Polydextrose
Polyethylene glycol
Medium chain triglycerides
Titanium dioxide (E171)
Carmoisine lacquer (E122)
Ponso 4R lacquer (E124)

6.2. Incompatibilities
Not reported.

6.3. Shelf life
24 months

6.4. Special precautions for storage
Store at room temperature below 25 ° C and in a dry place. Keep out of reach of children and in its package.

6.5. Nature and contents of container
Bevit-B12 Film Tablet is marketed as 30 and 60 tablets in PVC / PE / PVDC / Al blisters.

6.6. Special precautions for disposal and other handling
Any unused product or waste material should be disposed of in accordance with "Directive on Control of Medical Waste" and "Directive on the Control of Packaging and Packaging Waste.

7. MARKETING AUTHORISATION HOLDER:
Berko İlaç ve Kimya San. A.Ş.
Yenişehir Mah. Özgür Sok. No: 16-18 Ataşehir/İstanbul-Turkey
+90 216 456 65 70 (Pbx)
+90 216 456 65 79 (Fax)
info@berko.com.tr

8. MARKETING AUTHORISATION NUMBER(S)
236/41

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION
Date of first authorisation: 01.11.2011
Date of latest renewal: 13.04.2017

10. KÜB'ÜN YENİLENME TARİHİ