SUMMARY OF PRODUCT CHARACTERISTICS

1. NAME OF THE MEDICINAL PRODUCT

MINAFEN 120mg / 5ml Syrup

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Active ingredient:

One scale (5 ml) contains 120 mg of paracetamol.

Excipients:

Sorbitol…….1000 mg/5ml

Karmoisin (azorubin)……0,10 mg/5 ml

For other excipients (see section 6.1)

3. PHARMACEUTICAL FORM

Syrup

Clear, pink-red color, fruit flavored, solution

4. CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

It is indicated for the symptomatic treatment of mild to moderate pain and fever in children.

4.2 POSOLOGY AND METHOD OF ADMINISTRATION

Posology / frequency and duration of administration:

It should not be used in higher doses than recommended

It should be used for the shortest possible duration of treatment, at the lowest dose necessary to ensure efficacy.

It is recommended to be 10-15 mg/kg/dose every 6 hours (maximum 500 mg once in children over 30 kg), maximum daily dose of 60 mg/kg (maximum 2 grams daily in

children over 30 kg). The minimum dose interval should be 4 hours and should not be given more than 4 times a day.
Not recommended for children 6 years and older.
Due to the risk of hepatotoxicity in people who drink alcohol, the daily dose of paracetamol should not exceed 2000 milligrams..
It should not be used longer than 3 consecutive days without a docotr's recommendation.

Route of administration

It is administered orally. The thick consistency of MINAFEN prevents the medicine from spilling from the spoon and makes it easier to apply. It should be shaken before use.

Additional information on special populations:

Renal failure:

It should be used with caution in patients with kidney failure, with the recommendation of a doctor. It is contraindicated in patients with severe renal impairment.

Liver failure:

It is contraindicated in patients with severe hepatic impairment (Child Pugh category > 9). It should be used with caution in patients with mild and moderate hepatic impairment, with the recommendation of a doctor.

Pediatric population:

A dose of 2.5 ml (½ cup) is suitable for infants with fever after vaccination at 2 months. It should not be used in infants under two months (see: Posology/administration frequency and duration)..

Geriatric population:

No pharmaceutical data are available for this age group.

Paracetamol can be administered by dosing scheme in adults in active-mobile elderly.

In the fond, sedentary elderly, the dose should be reduced and the dosing interval should be extended.

4.3. Contraindications

PARADISOL,

It is contraindicated in case of hypersensitivity to paracetamol or any of its components, or in severe hepatic (Child Pugh category > 9) and renal impairment.

4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE

MINAFEN contains paracetamol. Do not use with other products containing paracetamol. Concomitant use with other products containing paracetamol may lead to overdose.

Paracetamol overdose can lead to liver transplant or liver failure, which can lead to death.

Cases of liver dysfunction/failure have been reported in patients with reduced glutathione levels (eg, patients with severe malnutrition, anorexic, low body mass index, chronic excessive alcohol use, or patients with sepsis).

he use of paracetamol in patients with reduced glutathione levels (eg, sepsis) may increase the risk of metabolic acidosis

It should be used with caution under the supervision of a doctor in patients with anemia, lung patients, liver and kidney dysfunction. For patients with pre-existing hepatic disease, liver function studies may be required at periodic intervals during high-dose or long-term therapy.

Patients with non-serious arthritis who need to take daily pain relievers are advised to consult a doctor.

Redness of the skin, rash or a skin reaction may occur in the first dose or repeated doses of paracetamol in those who use paracetamol for the first time or those who have a history of use. In this case, the doctor should be contacted, the use of the drug should be discontinued and an alternative treatment should be started. The person who has a skin reaction with paracetamol should not use this drug or any other drug containing paracetamol again. This condition can cause skin reactions, including severe and fatal Steven Johnson Syndrome (SJS), toxic epidermal necrolysis (TEN), and acute generalized exanthematous pustulosis (AGEP).

Dose adjustment should be made and the patient should be monitored continuously.

Causes severe liver toxicity in acute high dose.
May cause liver damage in adults at chronic daily doses.

Because of the risk of hepatotoxicity, paracetamol should not be taken in higher doses or for longer periods than recommended. Patients with mild or moderate hepatic impairment (Child-Pugh category <9) should use paracetamol with caution.

During therapeutic doses of paracetamol, serum alanine aminotransferase (ALT) level may increase.

Concomitant use of therapeutic doses of paracetamol with drugs that increase hepatic oxidative stress and decrease hepatic glutathione reserve, various conditions such as alcoholism, sepsis or diabetes mellitus may lead to an increased risk of hepatic toxicity.

It should be used with caution in patients with glucose 6 phosphate dehydrogenase deficiency. Rare cases of hemolysis can be seen.

Long-term use of high doses of paracetamol can cause kidney damage.

In general, continued use of paracetamol, especially in combination with other analgesics, may lead to permanent kidney damage and a risk of kidney failure (analgesic nephropathy).

The use of paracetamol by patients with Gilbert's syndrome may cause clinical symptoms such as jaundice and more pronounced hyperbilirubinemia. Therefore, these patients should use paracetamol with caution.

Concomitant intake of paracetamol with moderate alcohol may lead to an increased risk of liver toxicity. It should be used with caution in patients with alcoholic liver disease.

Due to the risk of hepatotoxicity in people who drink alcohol, the daily dose of paracetamol should not exceed 2000 milligrams.

If new symptoms occur or the pain and/or fever do not subside within 3 to 5 days, it is recommended that patients stop using paracetamol and consult a doctor.

Due to its sorbitol content, patients with rare hereditary problems of fructose intolerance should not take this medicine. When used in the maximum daily dose, the amount of sorbitol you take may exceed 10 grams, so it may have a mild laxative effect.

Carmosine (azorubin) in its content may cause allergic reactions due to the dyestuff.

4.5. INTERACTION WITH OTHER MEDICINAL PRODUCTS AND OTHER FORMS OF INTERACTION

Medications that slow gastric emptying, such as propantheline, may cause slow absorption of paracetamol and thus delay the onset of paracetamol's effect.

Drugs that accelerate gastric emptying, such as metoclopramide, may cause the paracetamol to be absorbed more rapidly and therefore the onset of paracetamol's effect faster.

The use of drugs that induce hepatic microsomal enzymes, such as anticonvulsants or oral contraceptives, may increase the extent to which paracetamol is metabolized, resulting in decreased plasma concentrations of the drug and rapid elimination rate.

Concomitant use of some hypnotics and antiepileptic drugs (glutethimide, phenobarbital, phenytoin, carbamazepine, etc.) or drugs that induce hepatic microsomal enzyme induction in the liver, such as rifampicin, with doses of paracetamol, which are harmless when used alone, may cause liver damage. In case of excessive alcohol consumption, taking paracetamol, even in therapeutic doses, can also cause liver damage.

Chronic alcohol intake may have contributed to the acute pancreatitis reported in a patient taking an overdose of paracetamol. Acute alcohol intake may reduce a person's ability to metabolize high doses of paracetamol; the plasma half-life of paracetamol may be prolonged.

The use of paracetamol in combination with chloramphenicol may prolong the half-life of chloramphenicol and therefore increase the risk of toxicity of this drug.

Paracetamol (or its metabolites) interacts with enzymes involved in vitamin K-dependent coagulation factor synthesis. Interactions between paracetamol and warfarin or coumarin derivatives may result in an increase in the "international normalized ratio" (INR) and an increased risk of bleeding. Therefore, patients using oral anticoagulants should not take long-term paracetamol without medical supervision and control.

The 5-hydroxytryptamine (serotonin) type 3 receptor antagonists, tropisetron and granisetron, can completely suppress the analgesic effect of paracetamol by pharmacodynamic interaction.

Simultaneous use of paracetamol and azidothimide (AZT - zidovudine) increases the tendency to neutropenia. Therefore, paracetamol should not be taken with AZT unless there is medical advice.

It is recommended to avoid combination therapy with more than one pain reliever. There is little evidence to suggest that this provides any additional benefit to the patient and generally leads to an increase in adverse effects.

The rate of absorption of paracetamol may be increased by metoclopramide or domperidone and decreased by cholestyramine.

St. John's Wort (Hypericum perforatum - St. John's Wort) may decrease blood levels of paracetamol.

The rate of absorption of paracetamol may be reduced when taken with food.

4.6. PREGNANCY AND LACTATION

General advice

Pregnancy category: B

Women with childbearing potential / Birth control (Contraception)

There is no evidence that paracetamol has an effect on fertility.
Nevertheless, caution should be exercised when administering to women of childbearing potential.

Gebelik dönemi

As with all drug use during pregnancy, pregnant women should consult their doctor before taking paracetamol. The lowest effective dose and shortest duration of treatment should be considered. For paracetamol, adequate clinical data on pregnancies are not available.
Animal studies do not indicate direct or indirect harmful effects with respect to pregnancy / embryonal / fetal development / parturition or postnatal development.

Lactation period:

Paracetamol is passes into breast milk, but the amount passed is not clinically significant at recommended doses. There are no contraindications for breastfeeding in the published literature.

Reproductive ability / Fertility

In chronic toxicity studies in animals, paracetamol has been reported to cause testicular atrophy and inhibit spermatogenesis. There are no studies investigating the effect on human fertility.

4.7. EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

In some patients, dizziness or somnolence may occur due to paracetamol use. Patients taking paracetamol should be careful during activities that require them to stay awake.

4.8. UNDESIRABLE EFFECTS

Adverse effects reported in clinical trials and post-marketing research are listed according to the following frequency degrees.

Very common (≥1 / 10); common (≥1 / 100 to <1/10); uncommon (≥1 / 1,000 to <1/100); infrequently (≥1 / 10,000 to <1 / 1,000); Very rare (<1 / 10,000), unknown (cannot be estimated from available data)

Infections and infestations

Common: Infection (2.9%)

Blood and lymphatic diseases

Very rare: Agranulocytosis, thrombocytopenia (isolated reports)

Bağışıklık sistemi hastalıkları

Rare: Eruption, urticaria, angioedema

Very rare: Anaphylactic shock, allergy test positive**, immune thrombocytopenia***

Diseases of the nervous system

Common: Headache (5.1%), dizziness (3.58%), drowsiness (6.97%), paresthesia (5.4%)

Ear and inner ear diseases

Uncommon: Impaired balance (1%)

Vascular diseases

Very sparse: Purpura

Respiratory, chest disorders and mediastinal diseases

Common: Upper respiratory tract infection (2.7%)

Very rare: bronchospasm*

Gastrointestinal diseases

Common: Nausea (2.3%), diarrhea (4.7%), dyspepsia (2.3%), flatulence (2.3%), abdominal pain (3.9%), constipation (3.9%), vomiting (7.8%)

Uncommon: Gastrointestinal bleeding (0.13%)

Hepatobiliyer hastalıklar

Very common: Above the upper limit of ALT (17.4%)

Common: 1.5 times the lower upper limit (4.2%)

Very rare: Hepatic dysfunction

Skin and subcutaneous tissue diseases

Rarely: Skin rash, pruritus, urticaria, allergic edema and angioedema, acute generalized exanthematous pustulosis, erythema multiform, Stevens-Johnson syndrome and toxic epidermal necrolysis (including fatal outcomes)

Kidney and urinary tract diseases

Unknown: Nephrotoxic effects of paracetamol following therapeutic doses are not common.

Papillary necrosis has been reported in long-term administration.

General disorders and application area diseases

Common: Facial edema (4.5%)

Uncommon: Peripheral edema (1%)

Very rare: Fever, asthenia

Surgical and medical procedures

Uncommon: Post-tonsillectomy hemorrhage (0.5%)

Common: Post-extraction bleeding (3.3%).

* Bronchospasm: Acetylsalicylic acid susceptible to asthma in 20% of patients

** Oral provocation test with paracetamol: It is positive in 15.5% of patients with paracetamol-related allergic symptoms (eruption, urticaria, anaphylaxis).

***Immune thrombocytopenia: In the presence of paracetamol and paracetamol sulfate, antibodies bind to GPIIb/IIIa and GPIb/IX/V receptors of platelets. Discontinuation of paracetamol treatment In a literature review of 2000 patients comparing paracetamol with placebo and nonsteroidal anti-inflammatory drugs, no difference was found between paracetamol and placebo in terms of frequency of undesirable effects and discontinuation of treatment. In a second literature review involving 2100 patients comparing paracetamol with nonsteroidal anti-inflammatory drugs, discontinuation of treatment was observed more frequently in the paracetamol group due to insufficient effect of the drug. One out of every 10 patients receiving paracetamol treatment discontinued the treatment, and one out of every 15 patients discontinued the treatment because they found the drug's effect insufficient. Compared to NSAIDs, the rate of discontinuation due to undesirable effects is lower. In clinical laboratory evaluations, the undesirable effects and changes in laboratory values of paracetamol used at therapeutic doses in clinical trials were found to be indistinguishable from those of placebo. Changes in biochemical values related to liver function indicate that the drug is taken in toxic doses. If the drug has been taken in toxic doses, aspartate aminotransferase (AST) and alanine aminotrasferase (ALT) begin to rise within 24 hours and peak after 72 hours. Any increase above 1000 units is descriptive for hepatotoxicity. In addition, bilirubin and creatinine rise, glucose falls. A decrease in arterial pH below 7.30, creatinine above 3.4 mg/dL, prolongation of prothrombin time for more than 100 seconds, and serum lactate level above 3.5 millimol/L are signs of poor prognosis. Differences in sensitivity to the adverse and toxic effects of paracetamol based on gender, race, height, weight, body structure, lifestyle and location have not been reported. Apart from these, risk factors that increase sensitivity to the toxic effects of paracetamol are included in the drug interactions section (See Section 4.5).

Children under 6 years of age are less susceptible to the toxic effects of paracetamol. It has been suggested that glutathione reserves and the high rate of detoxification play a role in this.
Chronic hepatic necrosis has been reported in a patient receiving daily therapeutic doses of paracetamol for approximately one year, and liver injury has been reported with daily intake of excessive amounts for shorter periods. Evaluation in a group of patients with chronic active hepatitis did not reveal differences in liver function abnormalities in long-term paracetamol users, and there was also no improvement in disease control after paracetamol was discontinued.

4.9. OVERDOSE AND TREATMENT

Experience with conditions following paracetamol overdose indicates that clinical signs of liver damage usually occur after 24 to 48 hours, with a peak of 4 to 6 days.

Paracetamol overdose can lead to liver transplant or liver failure, which can lead to death. Acute pancreatitis has generally been observed with hepatic dysfunction and liver toxicity.

There is a possibility of development of toxicity if used over 10 grams in adults. If risk factors are present (see below), taking 5 grams or more of paracetamol can cause liver damage.

Risk factors:
if you are sick,
- Carbamazepine, phenobarbitone, phenytoin, primidone, rifampicin, St. are on long-term treatment with St. John's Wort or other drugs that induce liver enzymes
Or
- If he regularly consumes ethanol well above the recommended doses
Or
- If there is a possibility of glutathione depletion (eg malnutrition, cystic fibrosis, HIV infection, starvation, cachexia)

The harm of overdose is greater in those with non-cirrhotic alcoholic liver disease. Hepatic injury following overdose in children is relatively rare. In paracetamol overdosage with hepatic cell damage, the half-life of paracetamol, which is around 2 hours in normal adults, is usually prolonged to 4 hours or longer. A decrease in $^{14}CO2$ excretion has been reported after ^{14}C-aminopyrine. This better demonstrates the relationship between paracetamol overdose and liver cell damage than measurements of plasma paracetamol concentration or half-life or conventional liver function test.

Renal failure may occur due to acute tubular necrosis following fulminant hepatic failure due to paracetamol. However, its incidence is not more frequent in this group of patients compared with patients with fulminant hepatic failure for other reasons. Infrequently, renal tubular necrosis may occur 2-10 days after ingestion, despite only minimal liver toxicity. It has been reported that chronic alcohol intake contributes to the development of acute pancreatitis in a patient who has taken an overdose of paracetamol. In addition to acute overdose, liver damage and nephrotoxic effects have been reported after daily ingestion of excessive amounts of paracetamol.

Signs and symptoms:

Pallor, anorexia, nausea and vomiting are common early symptoms of paracetamol overdose.. Hepatic necrosis is a dose-related complication of paracetamol overdose.

Hepatic enzymes may be elevated and prothrombin time is prolonged within 12 to 48 hours, but clinical symptoms may not be evident within 1 to 6 days following ingestion of the drug. This condition may include hepatomegaly, liver tenderness, jaundice, acute liver failure, and hepatic necrosis. Abnormalities in glucose metabolism and metabolic acidosis may occur. There may be an increase in blood bilirubin, hepatic enzymes, INR, prothrombin time, blood phosphate and blood lactate values. Severe poisoning may progress to liver failure, encephalopathy, hemorrhage, hypoglycemia, cerebral edema and death. Acute renal failure with acute tubular necrosis, marked by inguinal pain, hematuria, and proteinuria, may develop without liver damage. Cardiac arrhythmias and pancreatitis have been reported.

Treatment:

Paracetamol overdose should be treated promptly, even if symptoms of overdose are not present to protect the patient against delayed hepatotoxicity. For this, it may be necessary to give intravenous N-acetylcysteine or oral methionine.

If overdose occurs within 1 hour, treatment with activated charcoal should be considered. Plasma paracetamol concentration should be measured at or after 4 hours following oral administration (earlier concentrations are unreliable).

Treatment with N-acetylcysteine can be used for up to 24 hours after oral ingestion of paracetamol; however, the maximum protective effect is achieved up to 8 hours after oral ingestion. The efficacy of the antidote declines steeply after this time. If needed, the patient should be given intravenous N-acetylcysteine in accordance with the commonly used dosage schedule. If vomiting is not a problem, oral methionine may be a suitable alternative in remote areas outside the hospital. Control of patients presenting with severe hepatic dysfunction beyond 24 hours after oral ingestion should be discussed with the NPIS.

5. PHARMACOLOGICAL PROPERTIES

5.1 PHARMACODYNAMIC PROPERTIES

Pharmacotherapeutic group: Other analgesics and antipyretics, Anilides

ATC code: N02BE01

Paracetamol is an analgesic and antipyretic agent. The therapeutic effects of paracetamol are thought to be due to inhibition of prostaglandin synthesis as a result of inhibition of the cyclooxygenase enzyme. There is evidence that paracetamol is a more potent inhibitor of central cyclooxygenase than peripheral cyclooxygenase. Paracetamol has analgesic and antipyretic properties but only weak anti-inflammatory properties. This can be explained by the fact that inflammatory tissues contain higher levels of cellular peroxides than other tissues and that these cellular peroxides inhibit cyclooxygenase inhibition of paracetamol.

5.2 PHARMACOKINETIC PROPERTIES

Absorption:

Absorption of paracetamol is mainly achieved by passive transfer from the small intestine. Gastric emptying is a rate-limiting step for oral paracetamol absorption. Peak plasma paracetamol concentration usually occurs between 30 and 90 minutes after oral administration, depending on the formulation. Since paracetamol undergoes first pass metabolism at a variable rate, it is not fully present in the systemic circulation after oral administration. Oral bioavailability in adults seems to depend on the amount of paracetamol administered. Its oral bioavailability is 63% after a 500 mg dose, but increases to about 90% after 1 or 2 g (tablet form).

Distribution:

Paracetamol is evenly dispersed in many body fluids; The estimated volume of distribution is 0.95 l / kg. Following therapeutic doses, paracetamol does not significantly bind to plasma proteins.

The distribution kinetics (Vd / F) in children are similar to those in adults..

Metabolism:

The plasma half-life of paracetamol after therapeutic doses is 1.5-2.5 hours. Paracetamol is metabolized in the liver. The major urinary excretion is glucuronide and sulfate conjugate. Up to 10% of the administered paracetamol is converted into acetamidokinone, a reactive metabolite with a cytochrome P-450 mixed function oxidase system (mainly CYP2E1 and CYP3A4) in a minor route. This metabolite is conjugated with rapidly reduced glutathione and excreted as cysteine and mercapturic acid conjugates. When large amounts of paracetamol are taken, hepatic glutathione may decrease and lead to excessive accumulation of hepatocyte acetamidokinone covalently bound to vital hepatocellular macromolecules. This leads to hepatic necrosis, which can be seen in case of overdose.

The main metabolite of paracetamol in children (3-10 years) and newborns (0-2 days) is paracetamol sulfate.

Elimination:

Following a single dose (1000 mg i.v.), total body clearance of paracetamol is about 5 ml / min / kg. The renal clearance of paracetamol depends on urine flow rate, but not on pH. Less than 4% of the administered drug is excreted as unchanged paracetamol. In healthy individuals, approximately 85-95% of the therapeutic dose is excreted in the urine within 24 hours.

There was no age-related difference in the total elimination rate of paracetamol between children and adults.

Linearity and Nonlinear Status:

Binding of reactive paracetamol metabolites to liver cell proteins causes hepatocellular damage. At therapeutic doses, these metabolites are bound by glutathione and form non-toxic conjugates. However, in the case of massive overdose, the liver depletes the SH-donors store (which facilitates and promotes glutathione formation); toxic metabolites of the drug accumulate in the liver and develop liver cell necrosis, which leads to deterioration of liver function and progressively to hepatic coma.

The pharmacokinetics are linear when used in accordance with the posology.

Characteristics features of patients

Pharmacokinetics in renal insufficiency: The mean plasma half-life between 2-8 hours is the same in normal and renal failure patients, but elimination rate decreases in renal insufficiency between 8-24 hours. Significant accumulation of glucuronide and sulfate conjugates occurs in chronic renal failure. With the limited regeneration of the parent compound, some extra elimination may occur in paracetamol conjugates accumulated in patients with chronic renal failure. In chronic renal failure, it is recommended to extend the dose ranges of paracetamol. Since paracetamol plasma levels may decrease in hemodialysis, additional doses of paracetamol may be required to maintain therapeutic blood levels.

Pharmacokinetics in hepatic insufficiency: The mean plasma half-life in patients with mild hepatic disease is similar to that in normal individuals, but is significantly prolonged (approximately 75%) in severe hepatic impairment. However, the clinical significance of prolonging the half-life is unclear; because drug accumulation and hepatotoxicity have not been proven in patients with liver disease and glutathione

conjugation is not reduced. Administration of 4 g of paracetamol daily for 13 days to 20 patients with chronic stable liver disease did not cause deterioration in liver function. Paracetamol has not been proven to be harmful when taken at recommended doses in mild liver disease. However, in severe liver disease, the plasma paracetamol half-life is significantly prolonged.

Pharmacokinetics in the elderly: Differences observed in pharmacokinetic parameters between young and old healthy subjects are not considered clinically important. However, there is evidence suggesting that the serum paracetamol half-life is significantly increased (approximately 84%) and that paracetamol clearance is reduced in weak, immobile, and elderly patients compared to healthy young individuals (approximately 47%).

Pharmacokinetics in children: Studies have shown that paracetamol sulfate is the major metabolite of paracetamol in neonates aged 0-2 days and in children aged 3-10 years. Data in adults and children aged 12 years and older have shown that the major metabolite is glucoronide conjugate. However, there are no significant age-related differences in the overall elimination rate of paracetamol or the total amount of medication that passes into the urine.

5.3. PRECLINICAL SAFETY DATA

Non-clinical safety data obtained for paracetamol did not indicate significant findings in terms of recommended dosage and product use.

Mild toxicity was observed after oral administration of paracetamol in adult rats. It was found to be more toxic in rats due to the immaturity of the hepatic enzyme system. Acute toxicity symptoms caused vomiting. In chronic administration, effects such as reduced weight gain, diuresis, aciduria and dehydration and susceptibility to infection have been observed.

Potential genotoxicity was observed in rats at the hepatotoxic dose level and this finding was explained not as a direct DNA damage but as an indirect consequence of hepatotoxicity/myelotoxicity.

No increase in embryotoxic or teratogenic risk has been observed after extensive use in humans. Paracetamol is also frequently taken during pregnancy, and no adverse effects were observed on the course of pregnancy or on the unborn child.

In chronic toxicity studies in animals, it has been reported that paracetamol causes testicular atrophy and inhibits spermatogenesis.

6. PHARMACEUTICAL PARTICULARS

6.1. LIST OF EXCIPIENTS

Sorbitol (70%)
Glycerin 99.5%
Polyethylene glycol 400
Carboxymethylcellulose sodium
Strawberry aroma
Tutti frutti aroma
Citric acid monohydrate
Carmoisine (azorubine)
Sucralose
Sodium benzoate
Sodium citrate dihydrate
Purified water

6.2. INCOMPATIBILITIES

Not valid.

6.3. SHELF LİFE

36 ay

6.4. SPECIAL PRECAUTIONS FOR STORAGE

Store at room temperature below 30°C, protected from light. Do not store in refrigerator.
Shake well before use.
MINAFEN Syrup is used without dilution.

6.5 NATURE AND CONTENTS OF CONTAINER

100 and 150 mL amber colored glass bottles, in carton box

6.6. DISPOSAL OF OTHER MEDICINAL PRODUCTS AND OTHER SPECIAL PRECAUTIONS

Unused products or waste materials must be disposed of in accordance with the "Medical Waste Control Regulation" and the ve Packaging and Packaging Waste Control Regulations ".

7. MARKETING AUTHORISATION HOLDER

Drogsan Pharmaceuticals San. ve Tic. Inc.

Oğuzlar Mah. 1370. Sok. 7/3

06520 Balgat-ANKARA

8. MARKETING AUTHORISATION NUMBER(S) (IN TURKEY)

170/47

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORIZATION (IN TURKEY)

Date of first authorisation : 22.08.1994

Renewal of the authorization :

10. DATE OF REVISION OF THE TEXT

18/12/2019