Not to be sold by retail without the prescription of a Registered Medical Practitioner

Prescribing Information

1. Generic Name

Methylcobalamin, Pyridoxine Hydrochloride, Nicotinamide And Folic Acid Injection

(Brand Name: MEGO®-XL+ Injection)

2. Qualitative and Quantitative Composition

Each 2 ml contains:
Methylcobalamin IP …….... 1000 mcg.
Pyridoxine Hydrochloride IP …….. 100 mg.
Nicotinamide IP ……..... 100 mg.
Folic Acid IP ……... 0.7 mg.
Benzyl Alcohol IP (as preservative) ……. 2% w/v.
Water for Injections IP ……..… q.s.
Overages are added to compensate loss on storage.

3. Dosage Form and Strength

Dosage Form: Solution for injection.
Dosage Strength: Methylcobalamin 1000 mcg, pyridoxine 100 mg, nicotinamide 100 mg, folic acid 0.7 mg in 2 ml ampoule.

4. Clinical Particulars

4.1 Therapeutic Indication

MEGO-XL+ Injection is indicated for the treatment of peripheral neuropathies including diabetic neuropathy in adult patients.

4.2Posology and Method of Administration

Adults: The usual dosage is 1 ampule of MEGO-XL+ Injection daily, administered intramuscular (I.M.) or intravenous (I.V.) infusion 3 times a week. The dosage may be adjusted depending on the patient's age and symptoms. Injectable therapy is usually given for 4 to 8 consecutive weeks; followed by, therapy can be continued with oral preparations.
Or, as prescribed by the physician.

Direction / Handling Conditions

I.M. Administration: Following cautions should be exercised to avoid adverse effects on tissues or nerves. Avoid repeated injection at the same site. Do not inject in densely innervated site. If

insertion of the injection needle causes intense pain or if blood flows back into the syringe, withdraw the needle immediately and inject at a different site.

I.V. Administration: MEGO-XL+ Injection should not be given as a direct, undiluted I.V. injection as it may give rise to dizziness, fainting, and possible tissue irritation. MEGO-XL+ Injection must be diluted prior to I.V. administration with a suitable/compatible diluent such as dextrose, saline or similar I.V. infusion solutions. The solution should be used within 4 hours after dilution. Intravenous infusion may be administered over a period of at least 30 minutes.

Pharmaceutical Precautions

Each ampoule is for single use only. Methylcobalamin is susceptible to photolysis. It should be used promptly after the package is opened, and caution should be taken so as not to expose the ampules to direct light. The unused portion, if any, should be discarded.

Parenteral drug products should be inspected visually for particulate matter and discoloration prior to administration, whenever solution and container permit. Do not use if solution is not clear or has suspended matter. The diluted solution for infusion should not be used if crystals or precipitates are observed.

4.3Contraindications

MEGO-XL+ Injection is contraindicated in the following:

- Patients with known or suspected hypersensitivity to any component of the formulation.
- Not to be used in newly born or premature infants.
- An existing hypervitaminosis.

4.4Special Warnings and Precautions for Use

Test Dose: Before therapy with MEGO-XL+ Injection is instituted, a test dose is recommended to ascertain possibility of hypersensitivity to ingredients of MEGO-XL+ Injection. Serious acute hypersensitivity reactions may require the use of subcutaneous epinephrine and other emergency measures.

Allergic Reactions: Use caution in the management of patients with known anaphylactic reactions to hydroxocobalamin or cyanocobalamin or methylcobalamin. Consideration should be given to use of alternative therapies, if available. Allergic reactions may include anaphylaxis, chest tightness, edema, angioneurotic edema, urticaria, pruritus, dyspnea, and rash.

Immune Response: Antibodies to hydroxocobalamin-transcobalamin II complex have developed during hydroxocobalamin therapy. Arrhythmias secondary to hypokalaemia have occurred at the beginning of parenteral treatment with hydroxocobalamin. This may happen with methylcobalamin therapy also.

Photolysis: Methylcobalamin is susceptible to photolysis. It should be used promptly after the package is opened, and caution should be taken so as not to expose the ampules to direct light.

Renal and Hepatic Impairment: This product has not been studied in hepatic and renal impairment patients. It is recommended to monitor renal and hepatic functions while patient is on this therapy.

4.5Drug Interactions

Methylcobalamin

Oral Contraceptives: Serum concentrations of methylcobalamin may be decreased by use of oral contraceptives.

Chloramphenicol: Chloramphenicol should not be used with methylcobalamin. Parenteral chloramphenicol may attenuate the effect of vitamin B12 in anemia.

Other Drugs: Metformin, H2 receptor antagonists (cimetidine, ranitidine etc.), aminoglycosides, colchicine, aminosalicylic acid, anticonvulsants and alcohol decrease absorption of vitamin B12.

Drug/Laboratory Test Interactions: Persons taking most antibiotics, methotrexate, and pyrimethamine invalidate vitamin B12 diagnostic blood assays.

Pyridoxine

Pyridoxine reduces the effects of levodopa and activity of altretamine. It also decreases serum concentrations of phenobarbital and phenytoin. Pyridoxine may decrease antibiotic activities of erythromycin, kanamycin, streptomycin, doxycycline, and lincomycin. Drugs such as hydralazine, isoniazid, penicillamine, and oral contraceptives may increase the requirements for pyridoxine.

Nicotinamide

Nicotinamide may decrease antibiotic activities of erythromycin, kanamycin, streptomycin, doxycycline, and lincomycin.

Folic Acid

Phenytoin: Folic acid may increase phenytoin metabolism and lower the serum concentration of phenytoin resulting in increased seizure activity. Also, phenytoin may decrease serum folic acid concentrations.

Methotrexate: Folic acid may decrease a patient's response to methotrexate therapy.

Barbiturates: Folate reduces serum barbiturate concentrations.

Other Drugs and Alcohol: Folate deficiency states may be produced by drugs such as antiepileptics, oral contraceptives, anti-tuberculosis drugs, alcohol, and folic acid antagonists such as methotrexate, pyrimethamine, triamterene, trimethoprim, and sulfonamides.

Drug/Laboratory Test Interactions: Persons taking most antibiotics, methotrexate and pyrimethamine invalidate folic acid diagnostic blood assays.

4.6Use in Special Populations

Pregnant Women

Pregnancy Category C. Adequate and well-controlled studies have not been done in pregnant women. MEGO-XL+ Injection can be administered during pregnancy only at the recommendation of the physician.

Lactating Women
Vitamin B12 is known to be excreted in human milk. Caution should be exercised when MEGO-XL+ Injection is administered to a nursing woman. Nursing mothers should not use this preparation unless clearly needed and recommended by physician.

Pediatric Patients
This product has not been studied in children and thus, not indicated for use in pediatric population.

Geriatric Patients
Generally, dose adjustment is not required in the geriatric population with normal body functions (provided there is no severe renal and/or hepatic impairment).

4.7Effect on Ability to Drive and Use Machines

No studies have been performed on effect on the ability to drive and use machines; usually, no specific precautions are necessary. However, patients should be advised not to drive or operate machinery if affected by dizziness.

4.8Undesirable Effects

Methylcobalamin

Anaphylactic Reaction: Anaphylactic reaction such as decrease in blood pressure or dyspnea may occur. Patients should be carefully observed. In the event of such symptoms, treatment should be discontinued immediately and appropriate measures taken.

Other Adverse Reactions: Hypersensitivity, rash, erythema, pruritus, dizziness, agitation, anxiety, headache, hot sensation, diaphoresis, and pain/induration at the site of intramuscular injection. Pulmonary oedema, congestive heart failure (CHF), peripheral vascular thrombosis, polycythemia vera (bone marrow disorder), mild transient diarrhea, itching, transitory exanthema, feeling of swelling of entire body have also been reported with parenteral vitamin B substances.

Pyridoxine

Side effects such as headache, nausea, drowsiness, paresthesia (numbness/tingling of arms/legs) have been reported with pyridoxine when taken in large doses for a long time.

Nicotinamide

Upset stomach, nausea, and diarrhea may occur.

Folic Acid

Gastrointestinal disturbances and allergic sensitization have been reported rarely with folic acid.

4.9Overdose

No overdose has been reported with this product. In the event of overdose, treatment should be symptomatic and supportive. Hemodialysis may be effective in such circumstance.

5. Pharmacological Properties

5.1 Mechanism of Action

Methylcobalamin

Methylcobalamin regulates nerve function and reduces plasma homocysteine levels by following mechanisms:

1. Methylcobalamin promotes myelination (phospholipid synthesis): Methylcobalamin promotes the synthesis of lecithin, the main constituent of medullary sheath lipid and increases myelination of neurons in rat tissue culture more than cobamamide does.

2. Methylcobalamin promotes axonal transport and axonal regeneration: Methylcobalamin normalizes axonal skeletal protein transport in sciatic nerve cells from rat models with streptozotocin-induced diabetes mellitus. It exhibits neuropathologically and electrophysiologically inhibitory effects on nerve degeneration in neuropathies induced by drugs, such as adriamycin, acrylamide, and vincristine (in rats and rabbits), models of axonal degeneration in mice and neuropathies in rats with spontaneous diabetes mellitus.

3. Methylcobalamin is a kind of endogenous coenzyme B12: Methylcobalamin plays an important role in transmethylation as a coenzyme of methionine synthetase in the synthesis of methionine from homocysteine.

4. Methylcobalamin is well transported to nerve cell organelles, and promotes nucleic acid and protein synthesis: Methylcobalamin is better transported to nerve cell organelles than cyanocobalamin in rats. Also, methylcobalamin promotes nucleic acid and protein synthesis in rats more than cobamamide does.

Pyridoxine

Pyridoxine/vitamin B6 is a water soluble vitamin required for amino acid, carbohydrate, and fat metabolism. Pyridoxine have role as a coenzyme in a wide variety of enzymes involved in cell growth and cell division.

High homocysteine level in the blood (hyperhomocysteinemia) is a risk factor for cardiovascular disease, blood clotting abnormalities, myocardial infarction (heart attack), and ischemic stroke. Pyridoxine alone or in combination with folic acid has been shown to be effective for lowering homocysteine levels.

Nicotinamide

Niacin/nicotinamide required for the synthesis of nicotinamide adenine dinucleotide (NAD+) and nicotinamide adenine dinucleotide phosphate (NADP+) enzymes present in the cytosol of most

cell. The nicotinamide nucleotides play a widespread role as coenzymes to many dehydrogenase enzymes occurring both in the cytosol and within the mitochondria. They are therefore key components of many metabolic pathways affecting carbohydrate, lipid, and amino acid metabolism. Generally, NAD+ linked dehydrogenases catalyze oxidoreduction reactions in oxidative pathways, whereas NADP+ linked dehydrogenases or reductases are often found in pathways concerned with reductive syntheses.

Folic Acid

Folic acid is reduced in the body to tetrahydofolate, which is a coenzyme for various metabolic processes including the synthesis of purine and pyrimidine nucleotides, and hence the synthesis of DNA.

5.2Pharmacodynamic Properties

Methylcobalamin

Methylcobalamin is the neurologically active form of vitamin B12. Methylcobalamin is useful to treat or correct various neurological defects such as neuropathies. In many cases, liver does not convert cyanocobalamin, the commonly available form of vitamin B12, into adequate amounts of methylcobalamin. Nutritional inadequacies, enzyme defects, and pathological changes to tissues can all contribute to a reduced ability of the body to accomplish the synthesis of the active forms of vitamin B12 from cyanocobalamin. MEGO-XL+ Injection provides readymade form of vitamin B12 i.e., methylcobalamin to treat various types of neuropathies including diabetic neuropathy.

Pyridoxine

Pyridoxine is essential for cell growth and cell division. Pyridoxine also involves in carbohydrate, protein, and fat metabolism. Pyridoxine also reduces homocysteine levels in the blood.

Nicotinamide

Nicotinamide is key components of many metabolic pathways. It is required for carbohydrate, lipid, and amino acid metabolism.

Folic Acid

Folic acid is used in the treatment and prevention of the folate deficiency state. It is also involved in some amino-acid conversions, and in the formation and utilization of formate.

5.3Pharmacokinetic Properties

Methylcobalamin

Vitamin B12 is extensively bound to specific plasma proteins called transcobalamins; transcobalamin II appears to be involved in the rapid transport of the cobalamins to tissues. Vitamin B12 diffuses across the placenta and also appears in breast milk. Vitamin B12 is stored in the liver, excreted in the bile, and undergoes extensive enterohepatic recycling. Part of a dose is

excreted in the urine, most of it in the first 8 hours; urinary excretion, however, accounts for only a small fraction in the reduction of total body stores acquired by dietary means.

Pyridoxine

Pyridoxine crosses the placenta and is distributed into breast milk. Pyridoxine is stored mainly in the liver where there is oxidation to 4-pyridoxic acid and other inactive metabolites which are excreted in the urine. As the dose increases, proportionally greater amounts are excreted unchanged in the urine.

Nicotinamide

Nicotinamide and nicotinic acid are widely distributed in the body tissues. Nicotinic acid appears in breast milk. The main route of metabolism is their conversion to N-methylnicotinamide and the 2-pyridone and 4-pyridone derivatives; nicotinuric acid is also formed. Small amounts of nicotinic acid and nicotinamide are excreted unchanged in urine after therapeutic doses; however the amount excreted unchanged is increased with larger doses.

Folic Acid

Folic acid is converted to the metabolically active form 5-methyltetrahydrofolate in the plasma and liver. Folate is distributed into breast milk. The principal storage site of folate is the liver; it is also actively concentrated in the CSF. Folate undergoes enterohepatic circulation. Folate metabolites are eliminated in the urine and folate in excess of body requirements is excreted unchanged in the urine.

6. Nonclinical Properties

6.1 Animal Toxicology

Methylcobalamin

Repeated dose toxicity: A total of 48 Sprague-Dawley rats were randomly assigned to receive, intravenously, 1, 5, 25 or 100 mg/kg of cyanocobalamin (6 males and 6 females in each group) three times per week until completion of the study at 182 days (26 weeks). The animals were weekly examined and blood samples were taken at days 1, 85 and 182 for cyanocobalamin determination. Finally, all animals survived throughout the study period and had similar growth rates. No evidence of toxicity was detected by a detailed weekly examination of animals during the study period. Therefore the no-observed-adverse-effect-level (NOAEL) can be established at least at 100 mg/kg under the test conditions.

Carcinogenicity: Vitamin B12 (as cyanocobalamin or hydroxocobalamin) has a long history of safe use even at high doses. A tumour promoting effect of vitamin B12 has been reported in one study in rats. Rats kept on a methionine deficient diet supplemented with 5 µg/100 g vitamin B12 and treated with the carcinogen p-dimethylaminobenzene (DAB) had a higher incidence of

hepatomas compared to the group without supplemental vitamin B12. A control group receiving the supplemented diet without DAB showed no hepatic tumours. In another study, the effect of methylcobalamin and cyanocobalamin on the growth of Walker's carcinosarcoma and on the longevity of rats with implanted Zajdela ascites hepatoma cells has been studied. Study reported reduced survival of rats upon treatment with both compounds.

Pyridoxine

Acute oral toxicity: The acute oral toxicity of ten batches of pyridoxine hydrochloride was tested in mice. After 10 days observation period, the LD50 was found to be 6994 mg/kg body weight.

Repeated dose toxicity - oral: Rats and mice were orally exposed to pyridoxine hydrochloride for 10 days. Only at the lowest test doses (2000 mg/kg/day (mice) and 500 mg/kg/day (rats)) no mortality was seen. Rats and mice dosed at the highest dose (16000 mg/ kg/day) died on the first day. At 8000 mg/kg/day, exposed mice and rats died after second dosing. Pyridoxine influenced body weight gain of mice and rats in a dose-dependent way.

Mutagenicity: An Ames test was performed with pyridoxine. All bacterial strains showed negative responses up to 5000 ug/plate, i.e. no significant dose-related increase in the number of revertants with or without metabolic activation was seen. No cytotoxicity and/or precipitation of the test substance was observed. The negative and strain-specific positive control values were within the laboratory historical control data ranges indicating that the test conditions were adequate and that the metabolic activation system functioned properly. Based on the results of this study it is concluded that pyridoxine is not mutagenic in the *Salmonella typhimurium* reverse mutation assay and in the *Escherichia coli* reverse mutation assay with or without metabolic activation.

Nicotinamide

Acute oral toxicity: Two acute oral studies in rats were available yielding slightly different results. In the first study an LD50 value of about 3.5 g/kg was reported for both male and female animals. Effects were tremor and convulsions, sedation, and coma. In the other study a value of 7.1 g/kg was found for males and 5.5 g/kg for females. Clinical symptoms included ruffled coat, lethargy and coma. The oral LD50 in mice reported in a study was 3.1 g/kg. Loss of activity was observed in high dose animals within 60 minutes after dosing. Survivors were asymptomatic within 24 hours. Other data from the literature for nicotinamide administrated orally to mice and rats indicated LD50 values between 2.0 and 3.0 g/kg.

Acute dermal toxicity: Acute dermal toxicity was established in rabbits. When applied via this route an LD50 of >2000 mg/kg was found for nicotinamide.

Repeated dose toxicity - oral: In a 4-week oral toxicity study 5 rats received 0, 215 and 1000 mg nicotinamide/kg/day by gavage. Two additional groups of 5 rats, treated with 0 and 1000

mg/kg/day, were included in the study design and were allowed to recover for a 6 week period. In treated males body weight gain and food consumption were significantly decreased during part of the treatment period. Liver weight was increased in all treated animals. This finding was accompanied histopathologically by mild liver centrilobular hypertrophy. These effects were considered to be an adaptive response to nicotinamide treatment in males. In females at the high dose group extramedullary haematopoiesis of the spleen was reported. The NOAEL derived from this study is 215 mg/kg/day. In a dietary study administration of nicotinamide (35, 70 and 140 mg/kg/day) to male rats led to an enhanced growth at 70 mg/kg/day and growth inhibition at the highest dose level. No effects on weight of the adrenal glands and the kidney were seen. Relative liver weight was significantly decreased at 70 mg/kg/day only.

Mutagenicity: Nicotinamide was negative in an Ames test performed with Salmonella strains TA98, TA100, TA1535, TA1537 and TA1538 both with and without metabolic activation (rat S-9). Other tests using Salmonella strains and liver S-9-mixes from rat, mouse or monkey showed a similar result. One Ames test using TA97a and TA102 showed a weak, questionable response in the strain TA102 in absence of metabolic activation. Nicotinamide was not mutagenic in Saccharomyces stain D4. Based on the results of this study it is concluded that nicotinamide is not mutagenic in bacteria and it did not induce clastogenic effects both *in vitro* and *in vivo*.

Carcinogenicity: In a lifetime carcinogenicity study in Swiss mice receiving 1% nicotinamide in the diet, no increase of tumour incidence was observed. In a few studies where nicotinamide was given in combination with known carcinogens, both promoting and antitumorigenic effects were reported. Nicotinamide appeared to have a promoting effect in rats on pancreatic islet tumours when combined with streptzotocin and on renal tumours in rats that were pre-treated with diethylnitrosamine. Urethane initiated lung tumorgenesis in mice was significantly inhibited by post-treatment with nicotinamide in the diet (1 and 2.5%). The induction of pancreatic ductular adenomas and carcinomas induced by N-nitrosobis(2-oxoprolylamine) in hamster was completely inhibited by nicotinamide given intraperitoneally at 350 mg/kg.

Teratogenicity: A study on potential teratogenic effects is available with nicotinic acid but not with nicotinamide. Pregnant rats were exposed orally to 0, 40, 200 and 1000 mg/kg nicotinic acid during day 6-15 of gestation. They were sacrificed on day 20 and their reproductive tract was examined. Body weight gain of the dams in the highest dose group was slightly decreased. Placental weight was significantly decreased at this dose level. Fetuses did not show any adverse effects, except for a significantly lower body weight in male offspring of females treated at 1000 mg/kg/day. There was no teratogenic effect up to the maximum dose of 1000 mg/kg/day. The NOAEL for maternal toxicity and fetal effects was 200 mg/kg/day. Effects at the higher dose level were related to maternal toxicity.

Folic Acid

Animal studies have shown that folic acid can be a neurotoxin, and can cause convulsions in laboratory animals. This evidence is in part based upon in vitro tissue and cell culture studies, and/or using very high dose levels (i.v. dosages 60-90 mg). There is however no clear evidence for a folic acid-induced neurotoxicity in humans.

Toxicity in different strains of mice showed that toxic doses of folic acid may lead to convulsions, ataxia and weakness. Histopathological studies in some strains of mice showed acute renal tubular necrosis.

In another study with experimentally (diet) induced vitamin B12 deficiency in rhesus monkeys, three of the nine monkeys received 5 mg/week of supplemental folic acid intramuscularly, followed by 5 mg in the drinking water (5 days/week). Five animals developed visual impairment and optic atrophy, including the 3 monkeys that received supplemental folic acid. Apparently, the optical nerve lesions occurred earlier (by 10-11months) in the folic acid-treated animals. It should be noted that the visual lesions observed in these monkeys are only rarely noted in human disease. Spastic paralysis of hind legs and tail was found in 3 animals, including 2 animals receiving folic acid. Other lesions in cranial and peripheral nerves and in the white matter of the spinal cord were observed in some animals, but were apparently not affected by supplemental folic acid.

7. Description

MEGO-XL+ Injection contain dark red coloured clear solution.

Each 2 ml ampoule of MEGO-XL+ Injection contains 1000 mcg of methylcobalamin, 100 mg of pyridoxine, 100 mg of nicotinamide, and 0.7 mg of folic acid for I.M. or I.V. infusion use.

Methylcobalamin

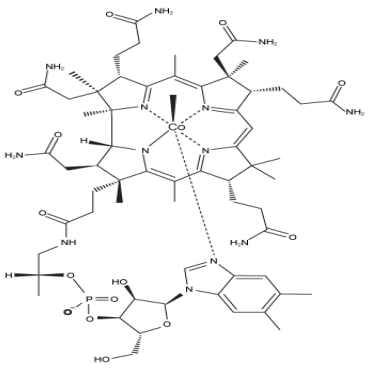
Methylcobalamin appears as dark red crystals or crystalline powder. It is sparingly soluble in water, slightly soluble in ethanol and practically insoluble in acetonitrile.

Molecular Weight: 1344.38 g/mol.

Molecular Formula: C63H91CoN13O14P.

Chemical Name: Methyl-5, 6-dimethylbenzimidazolylcobalamin.

Structural Formula:



Pyridoxine

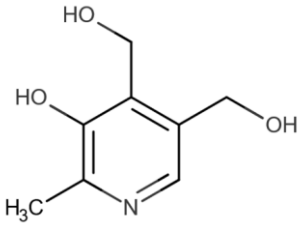
Pyridoxine hydrochloride is a white or practically white crystals or crystalline powder, soluble in water and insoluble in ether.

Molecular Weight: 169.18 g/mol.

Molecular Formula: C8H11NO3.

Chemical Name: 4,5-bis(hydroxymethyl)-2-methylpyridin-3-ol.

Structural Formula:



Nicotinamide

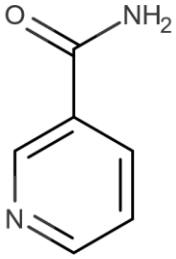
Nicotinamide is white or almost white, crystalline powder or colourless crystals, freely soluble in water and in dehydrated alcohol.

Molecular Weight: 122.12 g/mol.

Molecular Formula: C6H6N2O.

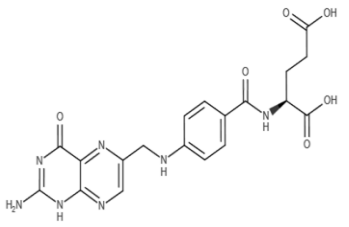
Chemical Name: Pyridine-3-carboxamide.

Structural Formula:



Folic Acid

Folic acid is a yellow, yellow-brownish, or yellowish orange, odourless crystalline powder. Very slightly soluble in water; insoluble in alcohol, in acetone, in chloroform, and in ether.
Molecular Weight: 441.40 g/mol.
Molecular Formula: C19H19N7O6.
Chemical Name: (2S)-2-[(4-{[(2-amino-4-oxo-1,4-dihydropteridin-6-yl)methyl]amino} phenyl) formamido] pentanedioic acid.
Structural Formula:



Inactive ingredients (excipients) of MEGO-XL+ Injection contain benzyl alcohol, phenol, EDTA di-sodium, and sodium hydroxide.

8. Pharmaceutical Particulars

8.1 Incompatibilities

MEGO-XL+ Injection should not be mixed with any calcium containing preparation as folic acid content of this formulation is unstable in the presence of calcium salts such as calcium gluconate.
MEGO-XL+ Injection should not be mixed with any other solution/injection for which physical and chemical compatibility has not been established.

8.2 Shelf-life

18 months.

8.3 Packaging Information

2 ml glass ampoule.

8.4 Storage and Handling Instructions

Store at a temperature not exceeding 25°C. Protect from light.
Keep out of reach of children.

9. Patient Counseling Information

Administration Instructions to Patients

- Instruct patient not to remove medication from its original packaging. Also, not to expose the ampules to direct light because methylcobalamin is susceptible to photolysis; this leads to degradation of the methylcobalamin.

- Instruct patients not to change their medication dose or schedule without consulting doctor or pharmacist. Do not exceed the recommended dose or duration of treatment.
- Instruct patient not to take injection at the same site. The injection site must be changed for each dose.
- Instruct patients not to share their medication with others even though it has been prescribed for same disease/condition. Also, not to use medication prescribed for others.

10.Details of Manufacturer

M/s. Nitin Lifesciences Ltd.,
Rampur Road, Paonta Sahib,
Dist. Sirmour – 173025, Himachal Pradesh, India.

11. Details of Permission or License Number with Date

DCG (I) NOC Date: 17th July 2015.
Manufacturing License No. MB/05/209. Date of Product permission - 26th April 2011.

12. Date of Revision

January 2021.

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