ARCHY FOLIC ACID TABLET 20mg (Folic Acid) SMPC

1. Name of the medicinal product

ARCHY FOLIC ACID 5mg TABLET
Folic Acid 5mg Tablet

2. Qualitative and quantitative composition

Each tablet contains Folic acid BP 5mg

3. Pharmaceutical form

TABLET
A Yellow round flat shaped tablet with FCD on one side and plain on the otherside

4. Clinical Particulars

4.1 Therapeutic Indications

Folic acid is indicated for treatment of folate-deficient megaloblastic anemia resulting for example from malnutrition, pregnancy or as a result of treatment with antiepileptics. It is also used prophylactically to prevent development of folate-deficient megaloblastic anemia in malnutrition, pregnancy, in some cases of renal dialysis and in chronic hemolytic states such as thalassaemia major or sickle cell anemia.

4.2 Posology and method of administration

Route of administration: ORAL.

The standard dosage in treatment of megaloblastic anemia is 5mg daily for a period of 4 months. In some cases complicated by malabsorption up to 15mg daily may be required.

The prophylactic dose in chronic hemolytic cases such as thalassaemia major or sickle-cell anaemia and renal dialysis is 5mg daily or even weekly depending on the diet and the rate of haemolysis

The prophylactic dose for megaloblastic anaemia in pregnancy is 200 -500mcg daily. In proven cases of folate deficiency a dosage of 0.25 -1.0mg daily should be given until haemopoetic response is obtained.

4.3 Contraindications

Long-term folate therapy is contraindicated in any patient with untreated cobalamin deficiency. This can be untreated pernicious anaemia or other cause of cobalamin deficiency, including lifelong vegetarians. In elderly people, a cobalamin absorption test should be done before long-term folate therapy. Folate given to such patients for 3 months or longer has precipitated cobalamin neuropathy. No harm results from short courses of folate.

Folic acid should never be given alone in the treatment of Addisonian pernicious anaemia anc other vitamin B_{12} deficiency states because it may precipitate the onset of subacute combined degeneration of the spinal cord.

Folic acid should not be used in malignant disease unless megaloblastic anaemia owing tc folate deficiency is an important complication.

Known hypersensitivity tc folic acid or any of the excipients

4.4 Special Warnings and Precautions for Use

Patients with vitamin B_{12} deficiency should not be treated with folic acid unless administered with adequate amounts of hydroxocobalamin, as it can mask the condition but the subacute irreversible damage to the nervous system will continue. The deficiency can be due tc undiagnosed megaloblastic anaemia including in infancy, pernicious anaemia or macrocytic anaemia of unknown aethiology or other cause of cobalamin deficiency, including lifelong vegetarians.

Caution should be exercised when administering folic acid tc patients who may have folate dependent tumours.

This product is not intended for healthy pregnant women where lower doses are recommended, but for pregnant women with folic acid deficiency or women at risk for the reoccurrence of neural tube defect.

Folic Acid Tablets contain lactose. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.

4.5 Interaction with other medicinal products and other forms of Interaction

There is a specific interaction between phenytoin and folate such that chronic phenytoin use produces folate deficiency. Correction of the folate deficiency reduces plasma phenytoin with potential loss of seizure control. Similar but less marked relationship exist with all anti-

convulsant treatments including sodium valproate, carbamazepine and the barbiturates. Sulphasalazine and triamterene also inhibit absorption.

Antibacterial, chloramphenicol and co-trimoxazole, may interfere with folate metabolism.

Folate supplements enhance the efficacy of lithium therapy. Methotrexate and trimethoprim are specific anti-folates and the folate deficiency caused by their prolonged use cannot be treated by Folic Acid Tablets. Folinic acid should be used. Nitrous oxide anaesthesia may cause an acute folic acid deficiency. Both ethanol and aspirin increase folic elimination.

4.6 Pregnancy and Lactation

Pregnancy

There are no known hazards tc the use of folic acid in pregnancy, supplements of folic acid are often beneficial.

folate deficiency induced by drugs such as anticonvulsants and some antineoplastics early in pregnancy results in congenital anomalies. Lack of the vitamin or its metabolites may also be responsible for some cases of spontaneous abortion and intrauterine growth retardation.

Lactation

Folic acid is actively excreted in human breast milk. Accumulation of folate in milk takes precedence over maternal folate needs. Levels of folic acid are relatively low in colostrum but as lactation proceeds, concentrations of the vitamin rise. Nc adverse effects have been observed in breast fed infants whose mothers were receiving folic acid

4.7 Effects on Ability to Drive and Use Machines

None unknown

4.8 Undesirable Effects

Rarely anorexia, nausea, abdominal distension and flatulence, allergic reactions, comprising erythema, rash, pruritus, urticaria, dyspnea, shock and anaphylactic reaction occurs.

4.9 Overdose and treatment

There are no specific symptoms of overdosage and similarly nc emergency treatment or antidotes, metabolisation and excretion can be rapid

5. Pharmacological properties

5.1 Pharmacodynamic properties

ATC Code: B03B B01 folic acid and derivatives.

Folic acid is a member of the vitamin B group, which is reduced in the body to tetrahydrofolate, a co-enzyme active in several metabolic processes and produces a haemopoietic response in nutritional megaloblastic anaemias

Folic acid is rapidly absorbed and widely distributed in body tissues.

5.2 Pharmacokinetic properties

Absorption – folic acid is rapidly absorbed from the gastrointestinal tract, mainly from the proximal part of the small intestine. Dietary folates are stated to have about half the bioavailability of crystalline folic acid. The naturally occurring folate polyglutamates are largely deconjugated and reduced by dihydrofolate reductase in the intestine to form 5-methyltetrahydrofolate (5MTHF). Folic acid given therapeutically enters the portal circulation largely unchanged, since it is a poor substrate for reduction by dihydrofolate reductases.

Distribution – via portal circulation. 5MTHF from naturally occurring folate is extensively plasma bound. The principal storage site of folate is in the liver; it is also actively concentrated in the CSF. Folate is distributed into breast milk.

Metabolism – therapeutically given folic acid is converted intc the metabolically active form 5MTHF in the plasma and liver. There is an enterohepatic circulation for folate.

Elimination – Folate not be used in malignant disease unless megaloblastic anaemia owing tc deficiency is an important complication.

Folic acid should

5.3 Preclinical safety data

There is no pre-clinical data of relevance tc a prescriber, which is additional to that already included in other sections of the SPC.

6. Pharmaceutical Particulars

6.1 List of Excipients

Lactose monohydrate
White Corn Starch
White Corn Starch For Paste
Gelatin
Sodium Benzoate
Tartrazine yellow Soluble Colour
Sodium crosscarmellose
Magnesium Stearate
Purified Talc
Sodium Lauryl Sulphate
Purified Water

6.2 Incompatibilities

None.

6.3 Shelf Life

36 months

6.4 Special Precautions for Storage

Store below 30°C in a dry place.

6.5 Nature and Contents of Container

1000's packed in polythene bags contained in HDPE containers with a literature insert.

Special precaution for disposal and other handling

No special requirements.

6.6 Marketing Authorization Holder and Manufacturing Site Addresses

Marketing Authorization Holder:

7 ARCHY PHARAMCEUTICAL LIMITED

Plot 30, Win Funke Street, Off Access bank junction Ahmadiyya, Lagos Abeokuta Expressway, Ojokoro Lagos State