

For the use of a Registered Medical Practitioner or a Hospital or a Laboratory or a Specialist only

Aceclofenac, Paracetamol & Serratiopeptidase Tablets

Maxipain SP

Composition:

Each film coated tablet contains:
Aceclofenac IP 100 mg
Paracetamol IP 325 mg
Serratiopeptidase IP 15 mg
(As enteric coated granules eq. to enzyme activity 30,000 units)
Colours : Sunset Yellow FCF & Titanium Dioxide IP

PHARMACEUTICAL FORM

Film Coated Tablet

THERAPEUTIC INDICATION

For acute painful conditions in adults only.
It is indicated for:
Resolution of inflammation and pain due to bone and soft tissue injury.
Resolution of post-operative inflammation, oedema and pain.

DOSAGE AND ADMINISTRATION

The recommended dose is 1 tablet once daily and can be taken twice daily depending upon the severity or as directed by the Physician.
The maximum recommended dose is two tablets daily, taken as one tablet in the morning and one in the evening.

Method of administration: For oral administration only. Patient should be advised not to chew or crush the tablets and should be swallowed whole with a sufficient amount of liquid.

CONTRAINDICATIONS

It is contraindicated in the following situations:
Patients sensitive to aceclofenac, paracetamol, serratiopeptidase or to any of the excipients.
Patients with a history of or active, recurrent peptic ulcer/haemorrhage (two or more distinct episodes of proven ulceration or bleeding).
Patients who have previously shown hypersensitivity reactions (e.g. asthma, rhinitis, angio-oedema or urticaria) in response to ibuprofen, aspirin or other NSAIDs.
Patients with a history of anaphylactic reactions.
Patients with severe heart failure, hypertension, and hepatic or renal impairment should not be prescribed.
During pregnancy, especially during the last trimester of pregnancy, unless there are compelling reasons for doing so. The lowest effective dosage should be used.

SPECIAL WARNINGS AND PRECAUTIONS FOR USE

General
Undesirable effects may be minimized by using the lowest effective dose for the shortest duration necessary to control symptoms. Concomitant use with NSAIDs, including COX-2 selective inhibitors, should be avoided. It should not be combined with other analgesic medications that contain paracetamol and should be given with care to patients with impaired kidney or liver function.
The administration of an NSAID may cause a dose-dependent reduction in prostaglandin formation and precipitate renal failure. Patients at the greatest risk of this reaction are those with impaired renal function, cardiac impairment, liver dysfunction, those taking diuretics and the elderly. Renal function should be monitored in these patients.

Respiratory Disorders

Respiratory is required if administered to patients suffering from, or with a previous history of, bronchial asthma since NSAIDs have been reported to precipitate bronchospasm in such patients.

Hepatic Toxicity

Paracetamol may cause liver damage if more than the recommended dose is taken. Allergic reactions like swelling of the face, mouth and throat, difficulty in breathing, itching or rash may occur due to high doses of paracetamol. Severe liver damage may occur if:

Adult takes more than 4000 mg in 24 hours, which is the maximum daily amount.
Child takes more than 5 doses in 24 hours.

Taken with other drugs containing paracetamol.
Adult has 3 or more alcoholic drinks every day while using this product.

Cardiovascular and Cerebrovascular Effects

Appropriate monitoring and advice are required for patients with a history of hypertension and/or mild-to-moderate congestive heart failure as fluid retention and oedema have been reported in association with NSAID therapy.

Clinical trial and epidemiological data suggest that the use of some NSAIDs (particularly at high doses and in long-term treatment) may be associated with a small increased risk of arterial thrombotic events (e.g., myocardial infarction or stroke). There are insufficient data to exclude such a risk for aceclofenac.

Patients with uncontrolled hypertension, congestive heart failure, established ischaemic heart disease, peripheral arterial disease, and/or cerebrovascular disease should only be treated after careful consideration. Similar consideration should be made before initiating long-term treatment of patients with risk factors for cardiovascular disease (e.g. hypertension, hyperlipidaemia, diabetes mellitus, smoking).

GI Bleeding, Ulceration and Perforation

GI bleeding, ulceration or perforation, which can be fatal, has been reported with all NSAIDs at any time during treatment, with or without warning symptoms or a previous history of serious GI events. Close medical surveillance is imperative in patients with symptoms indicative of GI disorders, with a history suggestive of GI ulceration, with ulcerative colitis or with Crohn's disease, bleeding diathesis or haematological abnormalities.

The risk of GI bleeding, ulceration or perforation is higher with increasing NSAID doses, in patients with a history of ulcers, particularly if complicated with haemorrhage or perforation, and in the elderly. These patients should commence treatment on the lowest dose available. Combination therapy with protective agents (e.g. misoprostol or proton-pump inhibitors) should be considered for these patients, and also for patients requiring concomitant low-dose aspirin, or other drugs likely to increase GI risk.

Patients with a history of GI toxicity, particularly when elderly, should report any unusual abdominal symptoms (especially GI bleeding), particularly in the initial stages of treatment. Caution should be advised in patients receiving concomitant medications that could increase the risk of ulceration or bleeding, such as oral corticosteroids, anticoagulants such as warfarin, selective serotonin-reuptake inhibitors or antiplatelet agents such as aspirin. When GI bleeding or ulceration occurs, the treatment should be withdrawn. NSAIDs should be given with care to patients with a history of GI disease (ulcerative colitis, Crohn's disease) as these conditions may be exacerbated.

SLE and Mixed Connective Tissue Disorders

In patients with systemic lupus erythematosus (SLE) and mixed connective tissue disorders, there may be an increased risk of aseptic meningitis.

Dermatological

Serious skin reactions, some of them fatal, including exfoliative dermatitis, Stevens-Johnson syndrome and toxic epidermal necrolysis, have been reported very rarely in association with the use of NSAIDs. Patients appear to be at the highest risk for these reactions early in the course of therapy, with the onset of the reaction occurring in the majority of cases within the first month of treatment. Discontinuation should be done at the first appearance of skin rash, mucosal lesions or any other sign of hypersensitivity.

Hypersensitivity Reactions

As with other NSAIDs, allergic reactions, including anaphylactic/anaphylactoid reactions, can also occur without earlier exposure to the drug.

Haematological

It may cause reversibly inhibit platelet aggregation.

Long-term Treatment

Individuals receiving long-term treatment should be regularly monitored for renal function tests, liver function tests and blood counts. It is to be used with caution in hepatic porphyria, coagulation disorders, history of peptic ulcers, ulcerative colitis, Crohn's disease, cerebrovascular bleeding, pregnancy and lactation. Caution should be exercised in patients with mild-to-moderate impairment of cardiac, hepatic or renal function and in elderly patients who are more likely to be suffering from these conditions. Caution is also required in patients on diuretic therapy or otherwise at risk of hypovolaemia.

DRUG INTERACTIONS

Aceclofenac

Other Analgesics, Including cox-2 Selective Inhibitors: Avoid concomitant use of two or more NSAIDs (including aspirin) as this may increase the risk of adverse effects.

Antihypertensives: Reduced antihypertensive effect.

Diuretics: Reduced diuretic effect. Diuretics can increase the risk of nephrotoxicity of NSAIDs. Although it was not shown to affect blood pressure control when co-administered with bendrofluzide, interactions with other diuretics cannot be ruled out. When concomitant administration with potassium-sparing diuretics is employed, serum potassium should be monitored.

Cardiac Glycosides: NSAIDs may exacerbate cardiac failure, reduce the glomerular filtration rate (GFR) and increase plasma glycoside levels.

Lithium: Decreased elimination of lithium.

Methotrexate: Decreased elimination of methotrexate. Caution should be exercised if NSAIDs and methotrexate are administered within 24 hours of each other, since NSAIDs may increase plasma levels, resulting in increased toxicity.

Cyclosporin: Increased risk of nephrotoxicity.

Mifepristone: NSAIDs should not be used for 8-12 days after mifepristone administration as NSAIDs can reduce the effect of mifepristone.

Corticosteroids: Increased risk of GI ulceration or bleeding.

Anticoagulants: NSAIDs may enhance the effects of anticoagulants, such as warfarin. Close monitoring of patients on combined anticoagulants and aceclofenac therapy should be undertaken.

Quinolone Antibiotics: Animal data indicate that NSAIDs can increase the risk of convulsions associated with quinolone antibiotics. Patients taking NSAIDs and quinolones may have an increased risk of developing convulsions. Antiplatelet agents and selective serotonin-reuptake inhibitors can lead to increased risk of GI bleeding.

Tacrolimus: Possible increased risk of nephrotoxicity when NSAIDs are given with tacrolimus.

Zidovudine: Increased risk of haematological toxicity when NSAIDs are given with zidovudine. There is evidence of an increased risk of haemarthroses and haematoma in HIV(+) haemophiliacs receiving concurrent treatment with zidovudine and ibuprofen.

Antidiabetic Agents: Clinical studies have shown that diclofenac can be given together with oral antidiabetic agents without influencing their clinical effect. However, there have been isolated reports of hypoglycaemic and hyperglycaemic effects. Thus, with aceclofenac, consideration should be given to adjustment of the dosage of hypoglycaemic agents.

Other NSAIDs: Concomitant therapy with aspirin or other NSAIDs may increase the frequency of adverse reactions, including the risk of GI bleeding.

Paracetamol

Drugs that induce hepatic microsomal enzymes, such as alcohol, barbiturates and other anticonvulsants, may increase the hepatotoxicity of paracetamol, particularly after overdosage. The anticoagulant effect of warfarin and other coumarins may be enhanced by prolonged regular use of paracetamol, with increased risk of bleeding. The effect appears to increase as the dose of paracetamol is increased, but can occur with doses as low as 1.5-2 g paracetamol per day for at least 5-7 days. Occasional doses have no significant effect.

Probenecid inhibits the glucuronidation of paracetamol which can affect the clearance of paracetamol. This should be considered when these medicines are administered concomitantly.

Paracetamol may affect the pharmacokinetics of chloramphenicol. This interaction should be considered when these medications are administered concomitantly, especially in malnourished patients.

Enzyme-inducing medicines, such as some antiepileptic drugs (phenytoin, phenobarbital, carbamazepine) have been shown in pharmacokinetic studies to reduce the plasma AUC of paracetamol to approximately 60%. Other substances with enzyme-inducing properties, e.g. rifampicin and St John's wort (Hypericum perforatum) are also suspected of causing lowered concentrations of paracetamol. In addition, the risk of liver damage during treatment with the maximum recommended doses of paracetamol will be higher in patients being treated with enzyme-inducing agents.

Serratiopeptidase

Serratiopeptidase may interact with medications that slow blood clotting (anticoagulant/antiplatelet drugs). Serratiopeptidase might decrease blood clotting. Therefore, taking serratiopeptidase along with medications that also slow clotting might increase the chances of bruising and bleeding. Some medications that slow blood clotting include aspirin, clopidogrel, diclofenac, ibuprofen, naproxen, dalteparin, enoxaparin, heparin, warfarin and others.

USE IN SPECIAL POPULATION

Pregnancy

Congenital abnormalities have been reported in association with NSAID administration in humans; however, these are low in frequency and do not appear to follow any discernible pattern. In view of the known effects of NSAIDs on the fetal cardiovascular system (risk of closure of the ductus arteriosus) and on the possible risk of persistent pulmonary hypertension of the newborn, use in the last trimester of pregnancy is contraindicated. The regular use of NSAIDs during the last trimester of pregnancy may decrease uterine tone and contraction. The onset of labour may be delayed and the duration increased, with an increased bleeding tendency in both mother and child. NSAIDs should not be used during the first two trimesters of pregnancy or labour unless the potential benefit to the patient outweighs the potential risk to the foetus. The drug is not recommended in pregnant women.

Lactation

In limited studies so far available, NSAIDs can appear in breast milk in very low concentrations. NSAIDs should, if possible, be avoided when breastfeeding.

The use of aceclofenac should be avoided in pregnancy and lactation unless the potential benefits to the other outweigh the possible risks to the foetus. The drug is not recommended in breastfeeding women.

Geriatric

As with other NSAIDs and combinations, caution is advised in elderly patients who are more likely to have concomitant renal, hepatic or cardiovascular impairment or receiving concurrent medication. The elderly have an increased frequency of adverse reactions to NSAIDs, especially GI bleeding and perforation, which may be fatal.

Patient with Renal Impairment

It should be avoided in patients with moderate and severe renal impairment. The importance of prostaglandins in maintaining renal blood flow should be taken into account in patients with impaired cardiac or renal function, those being treated with diuretics or recovering from major surgery. Effects on renal function are usually reversible on withdrawal.

Patient with Hepatic Impairment

In patients with hepatic impairment, dosage reductions are recommended. If abnormal liver function tests persist or worsen, clinical signs or symptoms consistent with liver disease develop or if other manifestations occur (eosinophilia, rash), it should be discontinued. Close medical surveillance is necessary in patients suffering from mild to moderate impairment of hepatic function. Hepatitis may occur without prodromal symptoms. Use in patients with hepatic porphyria may trigger an attack.

EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Undesirable effects such as dizziness, vertigo, drowsiness, fatigue, visual disturbances or other central nervous system disorders are possible after taking NSAIDs. If affected, patients should not drive or operate machinery.

UNDESIRABLE EFFECTS

Fixed drug eruption (FDE) has been reported with Paracetamol

Commonly-observed Adverse Events

GI Effects: The most commonly-observed adverse events are GI in nature. Peptic ulcers, perforation or GI bleeding, sometimes fatal, particularly in the elderly, may occur. Nausea, vomiting, diarrhoea, flatulence, constipation, dyspepsia, abdominal pain, melaena, haematemesis, ulcerative stomatitis, exacerbation of colitis and Crohn's disease have been reported following administration. Less frequently, gastritis has been observed. Pancreatitis has been reported very rarely.

Hypersensitivity: Hypersensitivity reactions have been reported following treatment with NSAIDs. These may consist of (a) non-specific allergic reactions and anaphylaxis (b) respiratory tract reactivity comprising asthma, aggravated purpura, bronchospasm or dyspnoea, or (c) assorted skin disorders, including rashes of various types, pruritus, urticaria, angioedema, angio-oedema and, more rarely, exfoliative and bullous dermatoses (including epidermal necrolysis and erythema multiforme).

Cardiovascular: Oedema, hypertension and cardiac failure have been reported in association with NSAID treatment. Clinical trial and epidemiological data suggest that use of some NSAIDs (particularly at high doses and in long-term treatment) may be associated with an increased risk of arterial thrombotic events (e.g. myocardial infarction or stroke). Vasculitis has been reported rarely.

Other Adverse Reactions Reported Less Commonly

Renal: Nephrotoxicity in various forms, including interstitial nephritis, nephritic syndrome and renal failure.
Hepatic: Abnormal liver function, hepatitis and jaundice.

Neurological and Special Senses: Visual disturbances, optic neuritis, headaches, dizziness, vertigo, paraesthesia, tinnitus (especially in patients with existing autoimmune disorders, such as SLE and mixed connective tissue disorders) and with symptoms such as stiff neck, headache, nausea, vomiting, fever or disorientation, depression, confusion, hallucinations, tinnitus, dizziness, malaise, fatigue and drowsiness.

Dermatological: Thrombocytopenia, neutropenia, agranulocytosis, aplastic anaemia and haemolytic anaemia.
Haematological: Bullous reactions, including Stevens-Johnson syndrome and toxic epidermal necrolysis (very rare). Photosensitivity.

Within the system organ classes, undesirable effects are listed under headings of frequency, using the following categories: very common (1/10); common (1/100 to <1/10); uncommon (1/1,000 to <1/100); rare (1/10,000 to <1/1,000); very rare (<1/10,000), and not known (cannot be estimated from the available data). Within each frequency grouping, undesirable effects are presented in order of decreasing seriousness.

Common: Nausea, dizziness, drowsiness, dyspepsia, abdominal pain, diarrhoea, redness of the rectal mucous membranes and hepatic enzyme increased.

Uncommon: Flatulence, gastritis, constipation, vomiting, mouth ulceration, pruritus, rash, dermatitis, urticaria.

Rare: Anaemia, anaphylactic reaction (including shock), hypersensitivity, allergic reaction, visual disturbance, dyspnoea, melaena, face oedema, exanthema, urticaria.

Very rare: Granulocytopenia, thrombocytopenia, neutropenia, haemolytic anaemia, hyperkalemia, hypoglycaemia, depression, abnormal dreams, insomnia, paraesthesia, tremor, somnolence, headache, dysgeusia (abnormal taste), vertigo, palpitations, flushing, hot flush, bronchospasm, stridor, stomatitis, haematemesis, gi haemorrhage, leg ulcer, pancreatitis, hepatitis, jaundice, liver damage, purpura, dermatitis, bullous exanthema, angioedema, cramps in the leg, renal impairment, nephrotic syndrome, oedema, fatigue, blood alkaline phosphatase increased, weight increase, increased serum potassium.

Most of the adverse events are minor and reversible with treatment discontinuation. As with other NSAIDs, severe mucocutaneous skin reactions may also occur.

Reporting of side effects or suspected adverse reaction: If you get or experience any side effects, talk to your doctor or pharmacist or report to india.drugsafety@akums.in. You can also report side effects directly via the National Pharmacovigilance Program of India by calling on 1800 180 3024. You should report side effects, you can help provide more information on the safety of this product.

OVERDOSE

Liver damage is possible in adults who have taken 10 g or more of paracetamol. Ingestion of 5 g or more of paracetamol may lead to liver damage if the patient has risk factors.

Risk factors:

If the patient:
is on long term treatment with carbamazepine, phenobarbitone, phenytoin, primidone, rifampicin, St John's wort or other drugs that induce liver enzymes, or
is likely to be glutathione depleted e.g. eating disorders, cystic fibrosis, HIV infection, starvation, cachexia.

Immediate treatment is essential in the management of overdosage. Despite a lack of significant early symptoms, patients should be referred to a hospital urgently for immediate medical attention.

Symptoms include headache, pallor, nausea, vomiting, epigastric pain, GI irritation, GI bleeding, rarely diarrhoea, disorientation, excitation, coma, drowsiness, dizziness, tinnitus, hypotension, respiratory depression, fainting, occasionally convulsions, anorexia and abdominal pain. In cases of significant poisoning, acute renal failure and liver damage are possible. Abnormalities of glucose metabolism and metabolic acidosis may occur.

In severe poisoning, hepatic failure may progress to encephalopathy, coma and death. Acute renal failure with acute tubular necrosis may develop even in the absence of severe liver damage. Cardiac arrhythmias and pancreatitis have been reported. Patients should be treated symptomatically as required. Within 1 hour of ingestion of a potentially toxic amount, activated charcoal should be considered. Alternatively, in adults, gastric lavage should be considered within 1 hour of ingestion of a potentially life-threatening overdose. Administration of oral methionine or intravenous N-acetylcysteine, which may have a beneficial effect up to at least 48 hours after the overdose, may be required. General supportive measures must be available.

Specific therapies such as dialysis or haemoperfusion are probably of no help in eliminating NSAIDs due to their high rate of protein binding and extensive metabolism. Good urine output should be ensured. Renal and liver function should be closely monitored. Patients should be observed for at least 4 hours after ingestion of potentially toxic amount. In case of frequent or prolonged convulsions, patients should be treated with intravenous diazepam.

Other measures may be indicated by the patient's clinical condition. Management of acute poisoning with NSAIDs essentially consists of supportive and symptomatic measures. Management of acute poisoning with NSAIDs essentially consists of supportive and symptomatic measures.

PHARMACODYNAMIC PROPERTIES

Pharmacodynamic Properties

Aceclofenac

Aceclofenac is a non-steroidal agent with anti-inflammatory and analgesic properties. The mode of action of aceclofenac is largely based on the inhibition to prostaglandin synthesis. Aceclofenac is a potent inhibitor of the enzyme, COX, which is involved in the production of prostaglandins. Aceclofenac relieves pain and inflammation through a variety of mechanisms and, in addition, exerts stimulatory effects on cartilage matrix synthesis.

It inhibits various mediators of pain and inflammation, including the following:

PGE2 via COX inhibition (COX-1 and COX-2) after intracellular metabolism to 4-hydroxyaceclofenac and diclofenac in human rheumatoid synovial cells and other inflammatory cells.

IL-1beta, IL-6 and tumour necrosis factor-alpha in human osteoarthritic synovial cells and human articular chondrocytes.

Reactive oxygen species (which plays a role in joint damage) has also been observed in patients with OA of the knees.

Expression of cell adhesion molecules (which is implicated in cell migration and inflammation) has also been shown in human neutrophils.

Stimulatory Effects on Cartilage Matrix Synthesis: Aceclofenac stimulates glycosaminoglycan synthesis in human osteoarthritic cartilage by inhibition of IL-1beta and suppresses cartilage degeneration by inhibiting IL-1beta-mediated pro-matrix metalloproteinase production and proteoglycan release.

Paracetamol

Paracetamol is an aniline derivative with analgesic and antipyretic actions similar to those of aspirin, but with no demonstrable anti-inflammatory activity. Paracetamol is less irritant to the stomach than aspirin. It does not affect thrombocyte aggregation or bleeding time. Paracetamol is generally well tolerated by patients hypersensitive to acetylsalicylic acid. The central analgesic action of paracetamol resembles that of aspirin. It produces analgesia by raising the pain threshold. The antipyretic effect of paracetamol is attributed to its ability to inhibit COX in the brain where the peroxide tone is low. Recent evidence suggests inhibition of COX-3 (believed to be a splice variant product of the COX-1 gene) and could represent a primary central mechanism by which paracetamol decreases pain and, possibly, fever.

Serratiopeptidase

Serratiopeptidase binds to alpha-2-macroglobulin in the blood in the ratio of 1:1, which helps to mask its antigenicity, but retain its enzymatic activity. Levels of serratiopeptidase are slowly transferred to the exudate at the site of inflammation and gradually, the blood level declines. By hydrolysing bradykinin, histamine and serotonin, it indirectly reduces dilatation of blood capillaries and controls permeability. Serratiopeptidase blocks plasmin inhibitors, thus helping the fibrinolytic activity of plasmin. Degradation of extra-fibrin to small fragment prevents the clogging of microcapillaries, helps clearance of exudates, reduces swelling and improves microcirculation.

Pharmacokinetic properties

Aceclofenac

Absorption: After oral administration, aceclofenac is rapidly and completely absorbed as unchanged drug. Peak plasma concentrations are reached approximately 1.25-3.00 hours following ingestion.

Distribution: Aceclofenac penetrates into the synovial fluid, where the concentrations reach approximately 57% of those in plasma. The volume of distribution is approximately 25 L. Aceclofenac is highly protein-bound (>99%). Aceclofenac circulates mainly as unchanged drug.

Metabolism: 4-hydroxyaceclofenac is the main metabolite detected in plasma.

Elimination: The mean plasma elimination half-life is around 4 hours. Approximately two-thirds of the administered dose is excreted via the urine, mainly as hydroxymetabolites.

Paracetamol

Absorption: Paracetamol is well absorbed by the oral route. The plasma half-life is about 2 hours.

Distribution: Plasma protein binding is negligible at the usual therapeutic concentration, but increases with increasing concentrations. Acetaminophen is, relatively, uniformly distributed throughout most body fluids. The plasma half-life is (t½) 2-3 hours and the effect after an oral dose lasts for 3-5 hours.

Metabolism: Paracetamol is primarily metabolized in the liver by conjugation to glucuronide and sulphate. A small amount (about 3-10% of a therapeutic dose) is metabolized by oxidation and the reactive intermediate metabolite thus formed is bound preferentially to the liver glutathione and excreted as cysteine and mercapturic acid conjugates.

Elimination: Excretion occurs via the kidneys. Of a therapeutic dose, 2-3% is excreted unchanged, 80-90% as glucuronide and sulphate, and a smaller amount as cysteine and mercapturic acid derivatives.

Serratiopeptidase

Absorption: After oral administration, serratiopeptidase is almost totally absorbed from the gastrointestinal (GI) tract.

Distribution: Serratiopeptidase binds to alpha-2 macroglobulin in the blood and produces an enzyme activity in the blood circulation. It shows a steep rise in concentration at the site of injury and inflammation.

Metabolism: Metabolism of serratiopeptidase takes place in the liver.

Excretion: The metabolites of Serratiopeptidase are excreted through the urine and faeces.

INCOMPATIBILITY

Not known.

Shelf Life

Refer on carton

Packing Information

Refer on carton

STORAGE CONDITIONS

Store protected from light & moisture, at a temperature not exceeding 25°C.

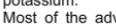
Keep all medicines out of reach of children.

Warning : Not for veterinary use.

Manufactured by :
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(A subsidiary of
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