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This information is intended for use by health professionals Amiodarone 200 mg Tablets Each tablet contains 200 mg of amiodarone hydrochloride. Excipients with known effect: Each tablet contains 125.00 mg lactose monohydrate For the full list of excipients, see section 6.1. Tablet. White to off white flat round bevelled edge uncoated tablets with inscription AZ on one side and scoreline on other side. Treatment should be initiated and normally monitored only under hospital or specialist supervision. Oral amiodarone is indicated only for the treatment of severe rhythm disorders not responding to other therapies or when other treatments cannot be used. Tachyarrhythmias associated with Wolff-Parkinson-White syndrome. Atrial flutter and fibrillation when other drugs cannot be used. All types of tachyarrhythmias of paroxysmal nature including: supraventricular, nodal and ventricular tachycardias, ventricular fibrillation: when other drugs cannot be used. Adults: It is particularly important that the minimum effective dose be used. In all cases the patient's management must be judged on the individual response and well-being. The following dosage regimen is generally effective: Initial stabilisation: Treatment should be started with 200mg, three times a day and may be continued for 1 week. The dosage should then be reduced to 200mg twice daily for a further week. Maintenance: After the initial period the dosage should be reduced to 200mg daily, or less if appropriate. Rarely, the patient may require a higher maintenance dose. The scored 100mg tablet should be used to titrate the minimum dosage required to maintain control of the arrhythmia. The maintenance dose should be regularly reviewed, especially where this exceeds 200mg daily. General considerations Initial dosing: A high dose is needed in order to achieve adequate tissue levels rapidly. Maintenance: Too high a dose during maintenance therapy can cause side effects which are believed to be related to high tissue levels of amiodarone and its metabolites. Amiodarone is strongly protein bound and has an average plasma half-life of 50 days (reported range 20 to 100days). It follows that sufficient time must be allowed for a new distribution equilibrium to be achieved between adjustments of dosage. In patients with potentially lethal arrhythmias the long half-life is a valuable safeguard, as omission of occasional doses does not significantly influence the overall therapeutic effect. It is particularly important that the minimum effective dosage is used and the patient is monitored regularly to detect the clinical features of excess amiodarone dosage. Therapy may then be adjusted accordingly. Dosage reduction/withdrawal Side effects slowly disappear as tissue levels fall. Following drug withdrawal, residual tissue bound amiodarone may protect the patient for up to a month. However, the likelihood of recurrence of arrhythmia during this period should be considered. Paediatric population The safety and efficacy of amiodarone in children has not been established. Currently available data are described in sections 5.1 and 5.2 but no recommendation on posology can be made. Elderly: As with all patients it is important that the minimum effective dose is used. Whilst there is no evidence that dosage requirements are different for this group of patients they may be more susceptible to bradycardia and conduction defects if too high a dose is employed. Particular attention should be paid to monitoring thyroid function. (see sections 4.3,4.4 and 4.8). Amiodarone is for oral administration. Sinus bradycardia and sino-atrial heart block: In patients with severe conduction disturbances (high grade AV block, bifascicular or trifascicular block) or sinus node disease, amiodarone should be used only in conjunction with a pacemaker. Evidence or history of thyroid dysfunction: Thyroid function tests should be performed in all patients prior to therapy. Known hypersensitivity to iodine or to amiodarone (one 100mg tablet contains approximately 37.5mg iodine), or to any of the excipients listed in section 6.1. The combination of amiodarone with drugs which may induce torsades de pointes is contra-indicated (see section 4.5). Pregnancy - except in exceptional circumstances (see section 4.6) Lactation (see section 4.6) Amiodarone can cause serious adverse reactions affecting the eyes, heart, lung, liver, thyroid gland, skin and peripheral nervous system (see section 4.8). Because these reactions may be delayed, patients on long-term treatment should be carefully supervised. As undesirable effects are usually dose-related, the minimum effective maintenance dose should be given. Before surgery, the anaesthetist should be informed that the patient is taking amiodarone (see sections 4.5 and 4.8). Cardiac disorders (see section 4.8): Too high a dosage may lead to severe bradycardia and to conduction disturbances with the appearance of an idioventricular rhythm, particularly in elderly patients or during digitalis therapy. In these circumstances, amiodarone treatment should be withdrawn. If necessary, beta-adrenostimulants or glucagon may be given. Because of the long half-life of amiodarone, if bradycardia is severe and symptomatic the insertion of a pacemaker should be considered. Oral amiodarone is not contra-indicated in patients with latent or manifest heart failure but caution should be exercised as, occasionally, existing heart failure may be worsened. In such cases, amiodarone may be used with other appropriate therapies. The pharmacological action of amiodarone induces ECG changes: QT prolongation (related to prolonged repolarisation) with the possible development of U-waves and deformed T-waves; these changes do not reflect toxicity. In the elderly, heart rate may decrease markedly. Treatment should be discontinued in case of onset of 2nd or 3rd degree A-V block, sino-atrial block or bifascicular block. Amiodarone has a low pro-arrhythmic effect. Onsets of new arrhythmias or worsening of treated arrhythmias, sometimes fatal, have been reported. It is important, but difficult, to differentiate a lack of efficacy of the drug from a proarrhythmic effect, whether or not this is associated with a worsening of the cardiac condition. Proarrhythmic effects generally occur in the context of QT prolonging factors such as drug interactions and/or electrolytic disorders (see sections 4.5, and 4.8). Despite QT interval prolongation, amiodarone exhibits a low torsadogenic activity. Before starting amiodarone, it is recommended to perform an ECG and serum potassium measurement. Monitoring of ECG is recommended during treatment. Amiodarone may increase the defibrillation threshold and/or pacing threshold in patients with an implantable cardioverter defibrillator or a pacemaker, which may adversely affect the efficacy of the device. Regular tests are recommended to ensure the proper function of the device after initiation of treatment or change in posology. Severe Bradycardia and heart block (see section 4.5): Life-threatening cases of bradycardia and heart block have been observed when sofosbuvir-containing regimens are used in combination with amiodarone . Bradycardia has generally occurred within hours to days, but later cases have been mostly observed up to 2 weeks after initiating HCV treatment. Amiodarone should only be used in patients on sofosbuvir- containing regimen when other alternative anti-arrhythmic treatments are not tolerated or are contraindicated. Should concomitant use of amiodarone be considered necessary, it is recommended that patients undergo cardiac monitoring in an in-patient setting for the first 48 hours of coadministration, after which outpatient or self-monitoring of the heart rate should occur on a daily basis through at least the first 2 weeks of treatment. Due to the long half-life of amiodarone, cardiac monitoring as outlined above should also be carried out for patients who have discontinued amiodarone within the past few months and are to be initiated on sofosbuvir- containing regimen. All patients receiving amiodarone in combination with sofosbuvir-containing regimen should be warned of the symptoms of bradycardia and heart block and should be advised to seek medical advice urgently should they experience them. Primary graft dysfunction (PGD) post cardiac transplant: In retrospective studies, amiodarone use in the transplant recipient prior to heart transplant has been associated with an increased risk of PGD. PGD is a life-threatening complication of heart transplantation that presents as left, right or biventricular dysfunction occurring within the first 24 hours of transplant surgery for which there is no identifiable secondary cause (see section 4.8). Severe PGD may be irreversible. For patients who are on the heart transplant waiting list, consideration should be given to use an alternative antiarrhythmic drug as early as possible before transplant. Endocrine disorders (see section 4.8): Amiodarone may induce hypothyroidism or hyperthyroidism, particularly in patients with a personal history of thyroid disorders. Clinical and biological (including ultrasensitive TSH (uTSH)) monitoring should be performed prior to therapy in all patients. Monitoring should be carried out during treatment, at six-monthly intervals, and for several months following its discontinuation. This is particularly important in the elderly. In patients whose history indicates an increased risk of thyroid dysfunction, regular assessments are recommended. Serum uTSH level should be measured when thyroid dysfunction is suspected. Amiodarone contains iodine and thus may interfere with radio-iodine tests (free-T3, free-T4, uTSH) remain interpretable. Amiodarone inhibits peripheral conversion of levothyroxine (T4) to triiodothyronine (T3) and may cause isolated biochemical changes (increase in serum free-T4, free-T3 being slightly decreased or even normal) in clinically euthyroid patients. There is no reason in such cases to discontinue amiodarone treatment if there is no clinical or further biological (uTSH) evidence of thyroid disease. Hypothyroidism: Hypothyroidism should be suspected if the following clinical signs occur: weight gain, cold intolerance, reduced activity, excessive bradycardia. The diagnosis is supported by an increase in serum uTSH and an exaggerated TSH response to TRH. T3 and T4 levels may be low. Euthyroidism is usually obtained within 3 months following the discontinuation of treatment. In life-threatening situations, amiodarone therapy can be continued, in combination with levothyroxine. The dose of levothyroxine is adjusted according to TSH levels. Hyperthyroidism: Hyperthyroidism may occur during amiodarone treatment, or, up to several months after discontinuation. Clinical features, such as weight loss, asthenia, restlessness, increase in heart rate, onset of arrhythmia, angina, congestive heart failure should alert the physician. The diagnosis is supported by a decrease in serum uTSH level, an elevated T3 and a reduced TSH response to thyrotropin releasing hormone. Elevation of reverse T3 (rT3) may also be found. In the case of hyperthyroidism, therapy should be withdrawn. Clinical recovery usually occurs within a few months, although severe cases, sometimes resulting in fatalities, have been reported. Clinical recovery precedes the normalisation of thyroid function tests. Courses of anti-thyroid drugs have been used for severe thyroid hyperactivity; large doses may be required initially. These may not always be effective and concomitant high dose corticosteroid therapy (e.g. 1mg/kg prednisolone) may be required for several weeks. Eye disorders (see section 4.8): If blurred or decreased vision occurs, complete ophthalmologic examination including funduscopy should be promptly performed. Appearance of optic neuropathy and/or optic neuritis requires amiodarone withdrawal due to the potential progression to blindness. Unless blurred or decreased vision occurs, ophthalmologic examination is recommended annually. Hepato-biliary disorders (see section 4.8): Amiodarone may be associated with a variety of hepatic effects, including cirrhosis, hepatitis, jaundice and hepatic failure. Some fatalities have been reported, mainly following long-term therapy, although rarely they have occurred soon after starting treatment particularly after Amiodarone intravenous. It is advisable to monitor liver function particularly transaminases before treatment and six monthly thereafter. Amiodarone dose should be reduced or the treatment discontinued if the transaminases increase exceeds three times the normal range. At the beginning of therapy, elevation of serum transaminases which can be in isolation (1.5 to 3 times normal) may occur. These may return to normal with dose reduction, or sometimes spontaneously. Isolated cases of acute liver disorders with elevated serum transaminases and/or jaundice may occur; in such cases treatment should be discontinued. There have been reports of chronic liver disease. Alteration of laboratory tests which may be minimal (transaminases elevated 1.5 to 5 times normal) or clinical signs (possible hepatomegaly) during treatment for longer than 6 months should suggest this diagnosis. Routine monitoring of liver function tests is therefore advised. Abnormal clinical and laboratory test results usually regress upon cessation of treatment, but fatal cases have been reported. Histological findings may resemble pseudo-alcoholic hepatitis, but they can be variable and include cirrhosis. Although there have been no literature reports on the potentiation of hepatic adverse effects of alcohol, patients should be advised to moderate their alcohol intake while taking Amiodarone tablets. Nervous system disorders (see section 4.8): Amiodarone may induce peripheral neuropathy and/or myopathy. Both these conditions may be severe, although recovery usually occurs within several months after amiodarone withdrawal, but may sometimes be incomplete. Respiratory, thoracic and mediastinal disorders (see section 4.8): Onset of dyspnoea or non-productive cough may be related to pulmonary toxicity (hypersensitivity pneumonitis, alveolar/interstitial pneumonitis or fibrosis, pleuritis, bronchiolitis obliterans organising pneumonitis. Presenting features can include dyspnoea (which may be severe and unexplained by the current cardiac status), non-productive cough and deterioration in general health (fatigue, weight loss and fever). The onset is usually slow but may be rapidly progressive. Whilst the majority of cases have been reported with long term therapy, a few have occurred soon after starting treatment. Patients should be carefully evaluated clinically and consideration given to chest X-rays before starting therapy. During treatment, if pulmonary toxicity is suspected, this should be repeated and associated with lung function testing including, where possible, measurement of transfer factor. Initial radiological changes may be difficult to distinguish from pulmonary venous congestion. Pulmonary toxicity has usually been reversible following early withdrawal of amiodarone therapy, with or without corticosteroid therapy. Clinical symptoms often resolve within a few weeks followed by slower radiological and lung function improvement. Some patients can deteriorate despite discontinuing Amiodarone tablets. Skin and subcutaneous tissue disorders (see section 4.8): Patients should be instructed to avoid exposure to sun and to use protective measures during therapy as patients taking Amiodarone tablets can become unduly sensitive to sunlight, which may persist after several months of discontinuation of Amiodarone tablets. In most cases symptoms are limited to tingling, burning and erythema of sun-exposed skin but severe phototoxic reactions with blistering may be seen. Severe bullous reactions: Life-threatening or even fatal cutaneous reactions Stevens-Johnson syndrome (SJS), Toxic Epidermal Necrolysis (TEN) (see section 4.8). If symptoms or signs of SJS, TEN (e.g. progressive skin rash often with blisters or mucosal lesions) are present amiodarone treatment should be discontinued immediately. Drug interactions (see section 4.5): Concomitant use of amiodarone is not recommended with the following drugs: beta-blockers, heart rate lowering calcium channel inhibitors (verapamil, diltiazem), stimulant laxative agents which may cause hypokalaemia. Increased plasma levels of flecainide have been reported with co-administration of amiodarone. The flecainide dose should be reduced accordingly and the patient closely monitored. Amiodarone tablets contains lactose: Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine. Pharmacodynamic interactions • Drugs inducing Torsade de Pointes or prolonging QT - Drugs inducing Torsade de Pointes Combined therapy with the following drugs which prolong the QT interval is contra-indicated (see section 4.3) due to the increased risk of torsades de pointes; for example: • Class Ia anti-arrhythmic drugs e.g. quinidine, procainamide, disopyramide • Class III anti-arrhythmic drugs e.g. sotalol, bretylium • intravenous erythromycin, co-trimoxazole or pentamidine injection • some anti-psychotics e.g. chlorpromazine, thioridazine, fluphenazine, pimozide, haloperidol, amisulpride and sertindole • lithium and tricyclic anti-depressants e.g. doxepin, maprotiline, amitriptyline • certain antihistamines e.g. terfenadine, astemizole, mizolastine • anti-malarials e.g. quinine, mefloquine, chloroquine, halofantrine. • Moxifloxacin - Drugs prolonging QT interval Co-administration of amiodarone with drugs known to prolong the QT interval (such as clarithromycin) must be based on a careful assessment of the potential risks and benefits for each patient since the risk of torsade de pointes may increase and patients should be monitored for QT prolongation. Concomitant use of amiodarone with fluoroquinolones should be avoided (concomitant use with moxifloxacin is contra-indicated). There have been rare reports of QTc interval prolongation, with or without torsades de pointes, in patients taking amiodarone with fluoroquinolones (see section 4.3). • Drugs lowering heart rate or causing automaticity or conduction disorders Combined therapy with the following drugs is not recommended: - Beta blockers and heart rate lowering calcium channel inhibitors (diltiazem, verapamil); potentiation of negative chronotropic properties and conduction slowing effects may occur. • Agents which may induce hypokalaemia Combined therapy with the following drugs is not recommended: - Stimulant laxatives, which may cause hypokalaemia thus increasing the risk of torsades de pointes; other types of laxatives should be used. Caution should be exercised over combined therapy with the following drugs which may also cause hypokalaemia and/or hypomagnesaemia, e.g. diuretics, systemic corticosteroids, tetracosactide, intravenous amphotericin. In cases of hypokalaemia, corrective action should be taken and QT interval monitored. In case of torsades de pointes antiarrhythmic agents should not be given; pacing may be instituted and IV magnesium may be used. • General anaesthesia Caution is advised in patients undergoing general anaesthesia, or receiving high dose oxygen therapy. Potentially severe complications have been reported in patients taking amiodarone undergoing general anaesthesia: bradycardia unresponsive to atropine, hypotension, disturbances of conduction, decreased cardiac output. A few cases of adult respiratory distress syndrome, sometimes fatal, most often in the period immediately after surgery, have been observed. A possible interaction with a high oxygen concentration may be implicated. Effect of amiodarone on other medicinal products Amiodarone and/or its metabolite, desethylamiodarone, inhibit CYP1A1, CYP1A2, CYP3A4, CYP2C9, CYP2D6 and P-glycoprotein and may increase exposure of their substrates. Due to the long half-life of amiodarone, interactions may be observed for several months after discontinuation of amiodarone • P-gP substrates Amiodarone is a P-gp inhibitor. Co administration with P-gp substrates is expected to result in an increase of their exposure: - Digitalis: administration of Amiodarone to a patient already receiving digoxin will bring about an increase in the plasma digoxin concentration and thus precipitate symptoms and signs associated with high digoxin levels. Clinical, ECG and biological monitoring is recommended and digoxin dosage should be halved. A synergistic effect on heart rate and atrioventricular conduction is also possible. - Dabigatran: caution should be exercised when amiodarone is co administered with dabigatran due to the risk of bleeding. It may be necessary to adjust the dosage of dabigatran as per its label. • CYP 2C9 substrates Amiodarone raises the plasma concentrations of oral anticoagulants (warfarin) and phenytoin by inhibition of CYP 2C9: - Warfarin: the dose of warfarin should be reduced accordingly. More frequent monitoring of prothrombin time both during and after amiodarone treatment is recommended. - Phenytoin: phenytoin dosage should be reduced if signs of overdosage appear (resulting in neurological signs), and plasma levels may be measured. • CYP P450 3A4 substrates When such drugs are co-administered with amiodarone, an inhibitor of CYP 3A4, this may result in a higher level of their plasma concentrations, which may lead to a possible increase in their toxicity. - Ciclosporin: plasma levels of ciclosporin may increase as much as 2-fold when used in combination. A reduction in the dose of ciclosporin may be necessary to maintain the plasma concentration within the therapeutic range. - Statins: the risk of muscular toxicity (e.g.rhabdomyolysis) is increased by concomitant administration of amiodarone with statins metabolised by CYP 3A4 such as simvastatin, atorvastatin and lovastatin. It is recommended to use a statin not metabolised by CYP 3A4 when given with amiodarone. • Other drugs metabolised by cytochrome P450 3A4: examples of such drugs are lidocaine, srolimus, tacrolimus, sildenafil, fentanyl, midazolam, triazolam, dihydroergotamine, ergotamine and colchicine. • CYP 2D6 substrates - Flecainide: given that flecainide is mainly metabolised by CYP 2D6, by inhibiting this isoenzyme, amiodarone may increase flecainide plasma levels; it is advised to reduce the flecainide dose by 50% and to monitor the patient closely for adverse effects. Monitoring of flecainide plasma levels is strongly recommended in such circumstances. Effect of other products on amiodarone CYP3A4 inhibitors and CYP2C8 inhibitors may have a potential to inhibit amiodarone metabolism and to increase its exposure. It is recommended to avoid CYP 3A4 inhibitors during treatment with amiodarone. Grapefruit juice inhibits cytochrome P450 3A4 and may increase the plasma concentration of amiodarone. Grapefruit juice should be avoided during treatment with oral amiodarone. Other drug interactions with amiodarone (see section 4.4) Coadministration of amiodarone with sofosbuvir containing regimens may lead to serious symptomatic bradycardia. If coadministration cannot be avoided, cardiac monitoring is recommended (see section 4.4). Pregnancy: There are insufficient data on the use of amiodarone during pregnancy in humans to judge any possible toxicity. However, in view of its effect on the foetal thyroid gland, amiodarone is contraindicated during pregnancy, except in exceptional circumstances. If, because of the long half-life of amiodarone, discontinuation of the drug is considered prior to planned conception, the real risk of recurrence of life threatening arrhythmias should be weighed against the possible hazard for the foetus. Breast-feeding: Amiodarone is excreted into the breast milk in significant quantities and breast-feeding is contra-indicated. The ability to drive or to operate machinery may be impaired in patients with clinical symptoms of amiodarone-induced eye disorders. The following adverse reactions are classified by system organ class and ranked under heading of frequency using the following convention: Very common (≥1/10), Common (≥1/100 to





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