Ejercicios de distribucion normal

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Professional Reference articles are designed for health professionals to use. They are written by UK doctors and based on research evidence, UK and European Guidelines. You may find the Abnormal Heart Rhythms (Arrhythmias) article more useful, or one of our other health articles. Treatment of almost all medical conditions has been affected by the COVID-19 pandemic. NICE has issued rapid update guidelines in relation to many of these. This guidance is changing frequently. Please visit to see if there is temporary guidance issued by NICE in relation to the management of this condition, which may vary from the information given below.Torsades de pointes is a distinctive polymorphic ventricular tachycardia in which the QRS amplitude varies and the QRS complexes appear to twist around the baseline. Torsades de pointes is associated with a prolonged QT interval, which may be congenital or acquired.[1, 2]Torsades de pointes is usually not sustained and terminates spontaneously but frequently recurs unless the underlying cause is corrected. Torsades de pointes may degenerate into sustained ventricular tachycardia or ventricular fibrillation. Torsades is a life-threatening arrhythmia and may present as sudden cardiac death in patients with structurally normal hearts.The corrected QT interval is longer in the white population than in the black population, and longer in females than males. Therefore, torsades de pointes is more common in white races and in females.[4]Torsades occurs at any age. If it occurs at an early age, the cause is usually due to congenital long QT syndrome. In later years, the cause is usually due to acquired long QT syndrome.Congenital long QT syndromes - eg, Jervell and Lange-Nielsen syndrome, Romano-Ward syndrome.Acquired long QT syndromes:Acute myocardial infarction.Drugs - eg, antiarrhythmic agents of classes Ia and III, erythromycin, ketoconazole, tricyclic antidepressants, methadone, antipsychotics.[5, 6]Electrolyte disturbances; hypokalaemia, hypomagnesaemia, hypocalcaemia.Acute kidney injury, liver failure.Metabolic; hypothyroidism, anorexia nervosa, malnutrition.Bradycardia; sinoatrial disease, atrioventricular (AV) block.Toxins; heavy metals, insecticides.Episodes of torsades in patients with congenital long QT syndromes may be triggered by stress, fear or physical exertion.Patients with torsades usually present with recurrent episodes of palpitations, dizziness, and syncope.[7] Sudden cardiac death can occur with the first episode.Nausea, pallor, cold sweats, shortness of breath and chest pain may occur.A history of congenital deafness or a family history of sudden death may indicate a long QT syndrome.Physical findings depend on the rate and duration of tachycardia and the degree of cerebral hypoperfusion. Findings include rapid pulse, low or normal blood pressure, and transient or prolonged loss of consciousness.Other physical signs depend on the cause - eg, features of a congenital disorder.ECG:[8]Paroxysms of 5-20 beats, with a heart rate faster than 200 beats per minute. Sustained episodes are occasionally seen.Progressive change in polarity of QRS about the isoelectric line occurs with complete 180° twist of QRS complexes in 10-12 beats.Usually, a prolonged QT interval and pathological U waves are present. The most consistent indicator of QT prolongation is a QT of 0.60 seconds or longer or a QTc (corrected for heart rate) of 0.45 seconds or longer. QTc = QT interval divided by the square root of the interval (in seconds) between the onset of each QRS complex (Bazett's formula).A short-long-short sequence between the R-R interval occurs before the trigger response.Electrolytes; hypokalaemia, hypomagnesaemia and hypocalcaemia.Cardiac enzymes; assessment for myocardial ischaemia.CXR and echocardiography, to rule out structural heart disease.ResuscitationDefibrillation:Although torsades is often self-terminating, it may develop into ventricular fibrillation, which requires defibrillation.[9]In an otherwise stable patient, direct current (DC) cardioversion is usually a last resort because torsades is paroxysmal in nature and frequently recurs after cardioversion.Discontinuation of any offending agent (stop all QT-prolonging drugs) and correction of any underlying cause such as hypokalaemia, hypomagnesaemia and bradycardia.Intravenous magnesium is the drug of choice for torsades de pointes. Magnesium is effective even in patients with normal magnesium levels.Acceleration of the heart rate can be achieved by using beta 1-adrenergic agonists such as isoprenaline or overdrive electrical pacing.Isoprenaline is used as an interim treatment until overdrive pacing can be started:Isoprenaline accelerates AV conduction and decreases the QT interval.It can be used in bradycardia-dependent torsades that is usually associated with acquired long QT syndrome.Isoprenaline is given as a continuous intravenous infusion to keep the heart rate faster than 90 beats per minute.Beta-adrenergic agonists are contra-indicated in the congenital form of long QT syndrome.Temporary transvenous pacing:Pacing can be effective in terminating torsades by increasing the heart rate and so reducing the QT interval.Atrial pacing is the preferred mode because it preserves the atrial contribution to ventricular filling. In patients with AV block, ventricular pacing can be used to suppress torsades.Patients without syncope, ventricular tachyarrhythmia or a family history of sudden cardiac death can be observed without starting any treatment.Congenital long QT syndrome:Beta-adrenergic antagonists are used as a first-line long-term therapy in congenital long QT syndrome. Propranolol is has been the most extensively used.Beta-blockers are contra-indicated in acquired cases because bradycardia produced by these agents can precipitate torsades. They should also be avoided in those congenital cases in which bradycardia is a prominent feature.Permanent pacing benefits patients who remain symptomatic despite receiving the maximally tolerated dose of beta-blockers and can be used in addition to beta-blockers.High left thoracic sympathectomy is effective in patients who remain refractory to beta-blockade and pacing.Implantable cardioverter-defibrillators (ICDs) are useful in rare instances when torsades still continues despite all of these treatments. Beta-blockers should be used along with ICDs because shock can further precipitate torsades by adrenergic stimulation.Acquired long QT syndrome:Long-term treatment in acquired cases is usually not required because the QT interval returns to normal once the predisposing factor has been corrected.Pacemaker implantation is effective in cases that are associated with heart block or bradycardia.ICDs are indicated in cases that cannot be managed by avoidance of any specific precipitating factor.Ventricular tachycardiaVentricular fibrillationSudden cardiac deathPatients may revert spontaneously or convert to a non-polymorphic ventricular tachycardia or ventricular fibrillation.[9]Torsades is a life-threatening arrhythmia and may present as sudden cardiac death in patients with structurally normal hearts.In acquired long QT syndrome, the prognosis is excellent once any precipitating factor has been removed.Avoid offending drugs that prolong the QT interval.Prevent predisposing conditions such as hypokalaemia, hypomagnesaemia, and hypocalcaemia, especially in patients shown to have long QT interval.Screen families of patients with torsades for whom the cause for prolonged QT is suggested to be congenital.Kaye AD, Volpi-Abadie J, Bensler JM, et al; QT interval abnormalities: risk factors and perioperative management in long QT syndromes and Torsades de Pointes. 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