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MORGANI-ADAMS-STOKES SYNDROME

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Depending on the type of heart rhythm disturbance, the following 3 clinical and pathogenetic forms of MAS syndrome are distinguished:

1) Bradycardiac (oligiacystolic, adynamic) form, which develops with stop, SU failure, CA-blockade of II degree and complete AV-blockade of III degree with a ventricular contraction rate of 20 per minute or less. i.e., if the patient experiences suppression of the CU, sinoauricular block develops, and at the same time, II and III order automaticity centers are suppressed, MAS attacks appear (lethal outcome is possible due to ventricular asystole).

2) Tachycardiac (tachycardiac, tachysistolic) form, which develops in

- ventricular flutter and flickering
- paroxysmal ventricular tachycardia
- supraventricular tachycardia paroxysm
- atrial flutter - atrial flutter with a ventricular contraction rate exceeding 250 per minute in WPW syndrome.

3) Mixed form.

It develops when tachyarrhythmia (ventricular or atrial) alternates with ventricular asystole periods.

MAS syndrome usually occurs at the moment when the tachyardia suddenly transitions to asystole.

ETIOLOGY AND PATHOGENESIS

Most often, MAS syndrome is caused by various forms of AV blockade. Seizure may occur

- at the moment of incomplete AV blockade transition to complete

- At the moment of occurrence of an incomplete AV block against the background of a sinus rhythm or supraventricular arrhythmia.

In such cases, the development of seizures is associated with a delay in the appearance of ventricular automatism, i.e., when a long pause precedes the onset of stable functioning of the II or III order rhythm driver (pre-automatic pause).

- MAS syndrome can also occur with complete AV blockade in cases of a sharp decrease in impulses from the heterotropic focus of automaticity located in the heart ventricle, in particular, with the development of a blockade of impulse output from this focus.
- Sometimes, a sudden sharp decrease in cardiac output is caused by incomplete AV blockade of a high degree with the passage of every third, fourth, or subsequent atrial impulses to the ventricles.
- Also, a prolonged pre-automatic pause can lead to a sharp and sudden decrease in blood pressure, which precedes the onset of ventricular rhythm in the case of a sudden CA block or complete suppression (stopping) of blood pressure activity.

In most cases, an attack begins if the heart rate becomes less than 30 beats per minute.

- some patients retain consciousness even with a significantly lower heart rate (12-20 beats per minute).
- and, conversely, loss of consciousness in patients with diffuse lesions of brain vessels can develop with relatively frequent heart contractions (35-40 bpm).

The cause of seizures can be not only extremely rare, but also extremely frequent ventricular contractions (usually >200 bpm), which is observed in

- Atrial flutter with each impulse generated in the atria being transmitted to the ventricles (TP 1:1)
- tachysistolic form of BF.

Arrhythmias with such a high heart rate typically occur when there are additional conductive pathways between the atria and ventricles.

Finally, sometimes the development of seizures is accompanied by the complete loss of the contractile function of the heart ventricles due to their fibrillation and asystole.

etc. Causes causing MAS

1) AV blockade

2) transition of an incomplete AV block to a complete one
3) HR < 30 beats per minute.
4) HR > 200 beats per minute (BP 1:1; tachysistolic form of BF)
5) Rhythm disturbance with a sharp decrease in the heart's contractile function (FS, asystole).

CLINICAL PICTURE

Regardless of the pathogenetic form, the clinical picture of MAS syndrome is the same and is determined by the duration of life-threatening rhythm disorders that cause the development of this syndrome.

Clinical symptoms within 3-5 seconds of the onset of life-threatening arrhythmia correspond to the sudden onset of a lipotemic (pre-consciousness) state and are characterized by:

- sudden, sharply expressed general weakness, appearance of dark circles in front of the eyes, tinnitus and ringing, headache, nausea, vomiting;
- disorientation, disruption of coordination;
- pronounced pallor, often sweating;
- heart rhythm disturbance (sharply expressed bradycardia or asystole in bradycardiac form; tachycardia in tachycardiac form)

The exact type of arrhythmia can only be recognized using an ECG!

Within 10-20 seconds of the onset of life-threatening arrhythmia, fainting (syncopal state) develops, the patient is unconscious, and the following signs appear:

- paleness, cyanosis, acrocyanosis
- sharp drop in blood pressure
- significant decrease in muscle tone, however, some patients may experience clonic tremors of the face and torso.
- superficial, barely noticeable breathing
- cardiac arrhythmia persists in accordance with a particular form of MAS syndrome.

- If the seizure is associated with the FJ, a peculiar "buzzing" (Gering's sign) is heard over the xiphoid process in some cases.

For 20-40 seconds after the development of life-threatening arrhythmia, the patient remains unconscious.

- superficial respiration
- Blood pressure is sharply reduced or not even detected
- Generalized epileptiform seizures appear (occur if the duration of the "pre-automatic" pause exceeds 20-25 seconds).
- involuntary urination and defecation are possible.
- heart rhythm disturbance still persists.

Subsequently, if life-threatening arrhythmia persists for 1-5 minutes, clinical death occurs:

- unconscious patient
- pupils are dilated
- corneal reflexes disappear
- intensive cyanosis predominantly of the upper body
- Cheyne-Stokes or Biot respiration, rare or throbbing
- Arterial pulse is absent.

After the restoration of the heart's pump function, the patient quickly regains consciousness, often not remembering the seizure and previous sensations (retrograd amnesia).

The rate of seizure development, its severity, and symptoms can vary greatly.

✓ With a very short duration, the seizures are often reduced in nature, limiting themselves to

- short-term dizziness
- weakness
- short-term vision impairment

✓ **The syncopal state** sometimes lasts for several seconds and is not accompanied by the development of seizures or other manifestations of a widespread seizure.

✓ Sometimes loss of consciousness does not occur even with a very high heart rate (about 300 beats per minute), the symptoms are limited to sharp weakness and inhibition. Such seizures are more often observed in young people with good myocardial contractility and intact cerebral vessels.

✓ In cases of pronounced diffuse (usually atherosclerotic) lesions of the cerebral vessels, symptoms, on the contrary, develop rapidly.

MAS attacks in cardiac blockages can be triggered

- rapid transition from a horizontal position to a vertical one, sudden rise from bed (vegetative disorders).
- mental over-excitement (stress, fear, anxiety, waiting, etc.)

DIAGNOSIS

✓ In typical cases, it's not difficult, but sometimes presents certain difficulties, as it can be difficult.

✓ abortive seizures, manifested only by dizziness, weakness, darkening of vision, brief clouding of consciousness, pallor, are often encountered in various pathological conditions (including chronic cerebrovascular insufficiency).

✓ in the developed picture of MAS syndrome, differential diagnosis is most often carried out with epilepsy, less often with hysteria.

In epileptic seizures

- The patient's face is hyperemic.
- tonic seizures are replaced by clonic seizures.
- Aura (vegetative, motor, sensory, speech, mental) precedes a seizure.

ECG

During the MAS attack and immediately after it, giant negative expanded T waves of large amplitude are often recorded on the ECG. They are usually best seen in the V2-V4 branches. Giant deformed negative T waves are usually accompanied by a significant expansion of the QT interval (ventricular electrical systole). Such T on

the ECG in most cases clearly indicates a recent onset of loss of consciousness, which is a very characteristic sign of MAS.

Daily ECG monitoring (Holter)

This method allows you to "catch" incoming cardiac blockage, against the background of which loss of consciousness occurs.

HEALING

Treatment of patients with MAS syndrome consists of:

- measures aimed at suppressing attacks and
- measures aimed at preventing recurrent seizures.

In the case of a first-time identified syndrome, even if this diagnosis is presumptive, it is indicated to be hospitalized in a medical institution of cardiological profile to clarify the diagnosis and choose therapy.

Emergency care

During a widespread seizure, the patient receives the same immediate assistance on the spot as during cardiac arrest (ventricular asystole), as the direct cause of the seizure cannot be immediately established.

1. A punch to the lower third of the sternum (induces a reflex reaction of the heart). With no effect
2. Indirect cardiac massage
3. IVL (when breathing stops)
4. Electrical defibrillation (it suppresses the heart rate, but can also "start" the heart during asystole).
5. Emergency electroacridiostimulation in asystole
6. Intrakardial or endotracheal administration of 0.5 - 1ml of 0.1% adrenaline solution in 10 ml of isotonic sodium chloride solution. This procedure is performed when cardiac stimulation is impossible or delayed.

The measures are continued until the attack subsides or signs of biological death appear.Drug prophylaxis of attacks is possible only if they are caused by paroxysms of tachycardia or tachyarrhythmia. Constant use of various antiarrhythmic agents is

prescribed. In all forms of AV blockade, MAS attacks serve as absolute indications for surgical treatment - implantation of electrostimulants.

The stimulant model is selected depending on the form of blockade. So, in complete atrioventricular block, asynchronous, constantly acting cardiac pacemakers are implanted.If a critical decrease in heart rhythm against the background of incomplete atrioventricular block occurs periodically, cardiac stimulators are implanted, which are activated "on demand" ("demand" mode).The electrode of the implanted pacemaker is usually inserted through the vein into the cavity of the right ventricle of the heart, where it is fixed in the intertrabecular space using various devices. Less frequently (with pronounced sinoauricular block or periodic cessation of the sinus node), the electrode is fixed in the wall of the right atrium. The stimulant body is usually placed in the vagina of the rectus abdominis muscle, in women - in the retromammary space. Previously used electrode implantation on an open heart was practically abandoned due to the trauma of the operation. The operating time of electric cardiac stimulators is determined by the capacity of their power sources and the parameters of the impulses generated by the device. The work capacity of stimulants is monitored once every 3-4 months using special extracorporeal devices. Separate successful attempts to implant stimulators that generate programmed ("paired," "coupled," etc.) impulses, allowing for the suppression of a tachycardia attack, as well as miniature defibrillators, the working electrode of which can be implanted into the myocardium of the atria or ventricles, have been described. These devices are equipped with systems for automatic analysis of electrocardiographic information and activate when certain heart rhythm disturbances occur. In a number of cases, destruction (cryosurgical, laser, chemical, or mechanical) of additional atrioventricular conduction pathways is indicated, for example, the Kent bundle in patients with atrioventricular premature excitation syndrome complicated by atrial fibrillation.

The prognosis is determined by the severity and duration of the seizure. If severe cerebral hypoxia continues for more than 4 minutes, irreversible brain damage develops. However, if possible, early initiated simple resuscitation measures (indirect cardiac massage, artificial respiration) allow maintaining the body's vital activity at a sufficient level for several hours. The long-term prognosis depends on

the frequency and duration of seizures, the rate of progression of the pathological condition, the disruption of the rhythm or conductivity underlying the seizures, the states of the myocardial contractile function, and the presence and severity of diffuse lesions of the cerebral arteries. Timely surgical treatment significantly improves the prognosis.

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