

Case report

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CARDIOVASCULAR CHANGES IN 18-MONTH BOY WITH PURULENT MENINGOENCEPHALITIS

SUMMARY

It is well- known that acute cerebrovascular accidents could provoke changes in hemodynamics, function and electrophysiology of the heart.

The paper presents the case of a child without previous heart disease, who developed left ventricular dysfunction as well as characteristic ECG changes in the form of the rhythm of wondering pacemaker and and a giant T wave, coinciding with an acute meningoencephailitis.

Key words: meningoencephalitis, ECG, heart

CASE REPORT

An 18-month boy was admitted to hospital on 17th of October in 2002 for the evaluation of anorexia, somnolence, high fever, vomitting and nuchal rigidity. A week ago he had a cold and was not treated for that.

Physical examination revealed febrile T=38,8°C, pale and somnolent male toddler, with mild pharynx inflammation and slight nuchal rigidity.

His respitory rate was 32/min.

On cardiac examination, there were normal heart sounds without murmurs, slow and irregular pulse minimal rate 56/min, and increased blood pressure (TA=100/75 mmHg).

Laboratory analyses showed:

- Normal chest roentgenogram and normal fundus examination.
- Normal values of: urea, creatinine, glucose, electrolytes, creatinin-kinase, lactat-dehidrogenase, transaminases, as well as normal arterial blood gases.

- Blood count showed leukocitosis Le=17,6 with 90,3% of neutrophils.
- Obtained Cerebrospinal fluid (CSF) contained two thousand cells per cubic millimeter, with predominance of polymorhonuclears.
- Biochemical analyzes showed proteinorachia = 1, 83 g/l and glucorahia = 2,09 mmol/l
- CSF culture five days later revealed H. influenza.

First ECG obtained after admission showed the giant T wave and the rhythm of wandering pacemaker (figure 1).

Echocardiographic examination revealed no morphologic and hemodynamic changes except slightly depressed myocardial contractile function with shortening fraction – FS=28%.

Electroencephalogram performed 6h later showed signs of diffuse delta dysfunction (1–2 c/sek) without ictal changes.

ECG were normalized on the second day, as well as myocardial contractility (figure 2).

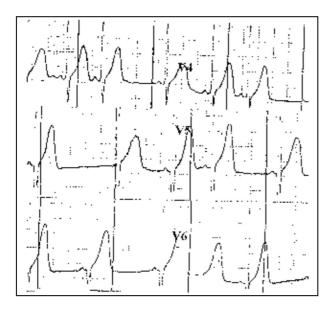


Figure 1. The rhythm of wondering pacemaker and giant T wave in lateral precordial leads

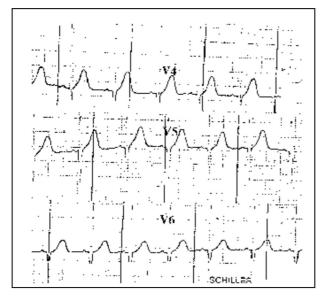


Figure 2. Normal electrocardiogram on the second day in the same boy

The similar patient's condition without significant changes lasted next 4 days. After that, the patient showed signs of improvements.

All these days the patient was somnolent, without neurologic, cardiorespiratory and other complication.

Acid-base, fluid and electrolyte values which were frequently controlled, were also within physiologic range.

The boy was treated with ceftriaxon (14 days, during) and manitol with dexamethasone in doses of 0,4-mg/kg bids, during the first and second day of hospitalization.

DISCUSSION

Until today, accumulated evidence have indicated that acute cerebrovascular accidents could provoke changes in cardiac electropysiology, function and structure (1,2,3).

Abnormal electrocardiographic changes without detectable heart disease have been known to accompany central nervous system disorders since Byer's original description in 1947 (4).

These electrocardiographic (ECG) changes chiefly and mostly represent repolarization abnormalities and may be very bizarre.

The most frequently mentioned are QT prolongation, T-wave flattening or inversion and ST-segment alternations (5).

Left ventricular dysfunction with cerebrovascular pathology was also observed (6).

There are a few possible explanations for this.

New observations suggest the role of cortical and autonomic nervous system as well as neuro-humoral mechanism in pathogenesis of myocardial damage and electrophysiologic changes (7,8,9,10).

We described the patient, without previous heart disease who developed acute ECG changes in the form of giant T waves and the rhythm of wandering pacemaker coinciding with an acute meningoencephalitis.

This ECG pattern normalized on the second day after hospitalization.

It is well- known that the giant T wave can reflect parasympathetic (Psy) influence on the heart, although there are other explanations of its origin.

Page et all. described the giant T wave in 2% of adult patients with subarachnoidal hemorrhage (11).

According to our opinion, the giant T vawe and left ventricular dysfunction in this case could be the consequence of the strong Psy influence on the heart.

It was shown that strong and simultaneous sympathetic and parasympathetic discharge could lead to exaggerated negative inotropic and chronotropic vagal influence on the heart.

This could be the condition in this case (12,13,14).

Impaired left ventricular function noticed simultaneously with ECG changes may be the second consequence of this.

The importance of adequate interpretation of possible cardiovascular changes coinciding with ongoing brain pathology lies in their potential for making the right diagnosis especially in children.

They cannot always express characteristic clinical signs of CNS derangements and it is important to differentiate, sometimes similar clinical pre-

sentations like intoxication, metabolic or other possible cardiovascular pathologies in contrast to cerebrogenic cardiovascular changes.

According to everything previously mentioned, good orientation in similar situations in clinical practise could help every physician in his everyday work.

REFERENCES

- 1. Oppenheimer SM. Cerebrogenic cardiac arrhythmias; Arch Neurol 1990; 47: 513–520.
- 2. Oppenheimer SM. The anatomy and physiology of cortical mechanisms of cardiac control; Stroke 1993; (suppl) 24;12: 1–3.
- 3. Tobias SL, Bookatz BJ, Diamond TH. Myocardial damage and electrocardiografic changes in acute cerebrovascular hemorrhage; Heart Lung 1987; 16: 521–526.
- 4. Byer E, Ashman R,Toth LA. Electrocardiograms with large, upright T waves and long QT intervals. Am Heart Journal 1947; 33:796–780.
- 5. Rogers MC, Zakha, KG, Nugent SK, Gioia FR, Epple L. Electrocardiographic abnormalities in infants and children with neurologic injury; Crit Care Med 1980; 8: 213–214.
- 6. Pollick C, Cujec B, Parker S, Tator C. Left ventricular wall motion abnormalities in subarachnoid hemorrhage: an echocardiographic study; J Am Coll Cardiol 1988; 12(3): 600–605.
- 7. Gruber A Keneth, Callahan F Michael. ACTH–(4–10) through -MSH evidence for a new class of central autonomic nervous system regulating peptides; Am J Physiol 1989; 257(2): R681–R694.
- 8. Kelly RA, Smith T. Cytokines and cardiac contractile function; Circulation 1997; 95:778–781.

- 9. Mc Intash TK. Neurochemical sequele of traumatic brain injury: therapeutic implications; Cerebrovasc Brain Metab Rev 1994; 6(2): 109–62.
- 10. Tanaka E, Mori H, Chujo M, Yamakawa A, Mohammed MU, Shinozaki Y, Tobita K Sekka T, Ito K, Nakazawa H. Coronary vasoconstrictive effects of neuropeptide Y and their modulation by the ATP-sensitive potassium channel in anesthetized dogs; J Am Coll Cardiol 1997; 29(6) 1380–1389.
- 11. Page A, Boularda G, Guerin J. Electro-cardiographic abnormalities in subarachnoiadal hemorrhage; Arch Mal Coeur Vaiss 1983; 76(9): 1031–1038.
- 12. De Geest H, Levy MN, Zieske H, Lipman RI. Depression of ventricular contractility by stimulation of vagus nerves. Circ Res 1965; 17: 222–235.
- 13. Higgins CB, Vatner SF, Braunwald E. Parasympathetic control of the heart. Pharmacol Rev 1973; 25: 119–155.
- 14. Morady F, Kou HW, Denelson S, Buitler MD, Schmaltz S, Kadish HA, Toivonen KL, Kushner AJ. Accentuated antagonism between beta adrenergic and vagal effect on ventricular refractoriness in humans; Circulation 1988; 77(2): 289–297.

KARDIOVASKULARNE PROMENE U OSAMNAESTOMESEČNOG DEČAKA SA PURULENTNIM MENINGOENCEFALITISOM

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SAŽETAK

Dobro je poznato da akutni cerebrovaskularni poremećaji mogu dovesti do izmena hemodinamike, funkcije i elektrofizioloških dešavanja u srcu.

Ovo je prikaz dečaka sa akutnim purulentnim meningoencefalitisom koji je koincidirao sa znacima disfunkcije leve komore i elektrokardiogramskim izmenama u formi ritma lutajućeg centra vodiča i džinovskog T-talasa.

Ključne reči: meningoencefalitis, EKG, srce