Reminder of important clinical lesson

Stroke and myocardial infarction: a terrible association

Marcelo Rodrigues Bacci,¹ Janaina Aparecida Boide Santos²¹Department of General Practice, Faculdade De Medicina Do Abc, Santo Andre, Sao Paulo, Brazil²Department of Cardiology, Faculdade De Medicina Do Abc, Santo Andre, Sao Paulo, Brazil

Correspondence to Professor Marcelo Rodrigues Bacci, mrbacci@yahoo.com

Summary

In the USA coronary heart diseases and cerebral vascular accidents together are the major causes of death regardless of race or sex. More specifically, the acute myocardial infarction and the encephalic vascular accident, as final events of atherosclerosis, correspond to a large part of death cases. Such cases could be potentially prevented with a proper follow-up and the identification of the risk factors. We present a case of a 65-year-old black man, diabetic and hypertensive, with ischaemic stroke diagnosis that progressed to acute ST elevation myocardial infarction. Thrombolysis or platelets antiaggregation were not possible due to the fact that the patient was in the acute phase of his ischaemic stroke. This case shows the association of the two major causes of death in this country, stroke and myocardial infarction, and the challenge for their treatment.

BACKGROUND

Cardiovascular events (coronary heart diseases and cerebral vascular accidents) are the leading causes of death, surpassing trauma and cancer, regardless of race or sex.¹ Besides multiple conditions like diabetes, dyslipidaemia, tobaccoism and systemic hypertension contribute to the onset of atherosclerosis and the consequent ischaemic cerebral and myocardial diseases.

It is estimated that between 6 and 28% of adults have experienced some level of silent ischaemic stroke, a rate that increases proportionally with age.¹

The increase of markers of myocardial necrosis is not frequent in acute encephalic events like ischaemic strokes. However, even if these markers do not indicate a case of acute myocardial ischaemia, a combined evaluation with a resting electrocardiogram (EKG), the serial collection of markers of myocardial necrosis and an echocardiogram should be conducted because of an acute myocardial infarction (MI) risk after the ischaemic insult.

This case clearly exemplifies the possible evolution to complications that follow the ischaemic stroke, like acute MI, pneumonia and sepsis. The proper identification and treatment of patients under these conditions is of great value, despite the limitations imposed by the impossibility of platelets antiaggregation and anticoagulation.

CASE PRESENTATION

A 65-year-old black man, who lived in São Bernardo do Campo, a municipality in São Paulo, Brazil, presented to the emergency department of São Bernardo do Campo Hospital Complex due to a sudden decrease in mental awareness and difficulty to move his left side of the body at home. His medical record showed that he had a hypertension and diabetes history, underwent secondary supra-patellar amputation of the right lower limb and suffered a previous acute arterial occlusion. The patient ignored cases of heart diseases or cerebral vascular accidents in his family history (table 1).

The physical exam revealed complete hemiplegia in the left hemibody associated with a decrease in the level of consciousness, with a score of 12 on the Glasgow Coma Scale, and no sign of meningeal irritation. He also presented conjugate eye deviation to the right, and when admitted to the emergency department, his blood pressure was 240/120 mm Hg with sinus tachycardia of 108 beats/min. No sign of cardiac arrhythmia was detected.

Cranial CT scan showed a widespread ischaemic stroke in the territory of the right-middle cerebral artery. Sodium nitroprusside treatment was started for pressoric control along with the administration of simvastatin and acetyl salicylic acid.

On the second day, there were EKG alterations which were compatible to anterior and lateral walls acute ST elevation myocardial infarction (STEMI) without haemodynamic instability. With the increase of markers of myocardial necrosis, the diagnosis was confirmed. The patient was ranked in Killip class I (figures 1 and 2).

Table 1 Patient's laboratorial data

Variable	Reference range	Admission	Second day
Haemoglobin (g/dl)	13.5–18 (men)	13.5	13.9
Haematocrit (%)	40–54 (men)	42.8	41.8
WBC (mm ³)*	5000–10 000	8400	10 500
Platelets (mm ³)	150 000–450 000	207 000	217 000
INR	0.92–1.09	1.19	1.09
Urea (mg/dl)	17–50	104	124
Creatinin (mg/dl)	0.7–1.3	2.16	3.44
CPK (U/l)	26–189	222	3,391
CK-MB (U/l)	<24	36	75
Troponin T	<0.1	Negative	1.99
Triglycerids (mg/dl)	<150	106	
Cholesterol (mg/dl)	<200	195	
HDL (mg/dl)*	40–60	28	
LDL (mg/dl)*	<100	146	

*WBC, white blood cell count; HDL, high-density lipoprotein; LDL, low-density lipoprotein; INR, international normalized ratio; CPK, creatinophosphokinase.

Figure 1 ST elevation in V4–V5–V6 derivations.

Figure 2 Arterial territory affected marked with red arrows.

OUTCOME AND FOLLOW-UP

On the third day, the patient was febrile and remained drowsy. With the worsening of the lung radiographic image, orotracheal intubation was made necessary along with antibiotic therapy (ceftriaxone and clindamycin). It progressed to a mixed septic and cardiogenic shock case.

CT image of the encephalic vascular accident remained unchanged, and it delimited all the territory of the right-middle cerebral artery showing no sign of midline shift or intracranial hypertension. An echocardiogram was performed and it showed left ventricular segmental cardiomyopathy associated with diastolic dysfunction. Ejection fraction was 31%. Carotid and vertebral Doppler ultrasonography could not be performed to investigate the emboligenic focus owing to the limitations on hospital services.

The patient continued with haemodynamic instability and received volemic expansion with crystalloid solutions and vasopressor support. There was simultaneous deterioration of renal function, secondary to the acute MI, with no need of renal replacement therapy. He died 7 days after his admission to the hospital emergency department.

DISCUSSION

Recent statistics show that in the USA cardiovascular disease is accounted for 32.8% of all deaths, corresponding to one out of three deaths. It is estimated that there are around 795 000 cerebral vascular accident cases every year and 87% of these cases are of ischaemic origin.¹

Diabetes, hypertension, dyslipidaemia, arrhythmia, tobaccoism, sedentarism and chronic renal disease are the isolated risk factors for the development of stroke.¹

Wilson *et al*² reached the conclusion in a risk model for the development of coronary heart disease that hypertension leads to a 1.84-fold increase in chance of developing it.

Stroke and MI both bear risk factors in common, like hypertension, tobaccoism, diabetes, dyslipidaemia, obesity, sedentarism and atherosclerosis. REACH study showed that 40% of 19 000 patients with stroke presented atherosclerotic disease in one or more territories like the coronaries or the peripheral circulation.³

Touzé *et al*⁴ showed that patients with stroke have a higher death risk of MI (around 2.2% a year) when compared with the general population.

EKG alterations are common in patients with stroke. Goldstein⁵ found 97% of some kind of alteration in 150 patients who were admitted with stroke. However, ST elevation was detected in only 6% of the evaluated cases.

According to the RANTTAS study, angina, MI and cardiac ischaemia occur in 6% of stroke cases, and within this percentage 1% corresponds to more severe evolutions.⁶

In the first 3 months after the stroke, 2–6% of the patients die of cardiac causes, and severe cardiac events occur in 19% of the cases. This may be the reflection of pre-existing cardiac disease or a cardiac dysfunction caused by acute neurohumoral change due to autonomic dysfunction resulted from stroke.⁷

Cerebral events that involve the insular cortex, irrigated by the middle cerebral artery, are associated with the highest rate of cardiac complications like arrhythmia and ischaemia. The insular is crucial in the regulation of the autonomic nervous system; therefore, it leads to the increase of circulating catecholamines that act in cardiac muscle.⁸

The diagnostic investigation through markers of myocardial necrosis, like troponin T and CK-MB, must be part of the first approach. However, CK-MB is less specific to determine the acute coronary syndromes when compared to troponin T in patients with stroke.^{9 10}

The present treatment for acute STEMI includes double platelets antiaggregation with acetyl salicylic acid and thienopyridines (clopidogrel, prasugrel, ticagrelor), anticoagulation with heparin (unfractionated or of low molecular weight), primary angioplasty or fibrinolysis as a resource whenever the proven morbidity and mortality improvement, immediate and in the long run, is not available.¹⁰

In the event of a stroke in less than 3 months, fibrinolytics are strictly contraindicated, and anticoagulation and platelets antiaggregation must be used cautiously due to the risk of haemorrhagic transformation.¹⁰ This is a condition that limits the treatment of acute MI and thus worsens the patient's prognostic.

Learning points

- ▶ There is a great correlation between acute myocardial ischaemic events and stroke.
- ▶ A sequential evaluation with EKG and cardiac enzymes dosages should be performed systematically in all patients with stroke during their hospital stay.
- ▶ The treatment of choice for acute STEMI is impaired by the restriction on the use of anticoagulation and fibrinolysis in the early stages of ischaemic stroke.
- ▶ There is a great chance for the onset of aspiration pneumonia in patients with the lowest levels of consciousness and subsequent evolution to sepsis. It is important to reinforce measures that prevent bronchoaspiration and to intensify respiratory physiotherapy.

Competing interests None.

Patient consent Obtained.

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