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Review

Electrocardiographic abnormalities and cardiac arrhythmias in structural brain lesions

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ABSTRACT

Cardiac arrhythmias and electrocardiographic abnormalities are frequently observed after acute cerebrovascular events. The precise mechanism that leads to the development of these arrhythmias is still uncertain, though increasing evidence suggests that it is mainly due to autonomic nervous system dysregulation.
In massive brain lesions sympathetic predominance and parasympathetic withdrawal during the first 72 h are associated with the occurrence of severe secondary complications in the first week. Right insular cortex lesions are also related with sympathetic overactivation and with a higher incidence of electrocardiographic abnormalities, mostly QT prolongation, in patients with ischemic stroke. Additionally, female sex and hypokalemia are independent risk factors for severe prolongation of the QT interval which subsequently results in malignant arrhythmias and poor outcome. The prognostic value of repolarization changes commonly seen after aneurysmal subarachnoid hemorrhage, such as ST segment, T wave, and U wave abnormalities, still remains controversial. In patients with traumatic brain injury both intracranial hypertension and cerebral hypoperfusion correlate with low heart rate variability and increased mortality.
Given that there are no firm guidelines for the prevention or treatment of the arrhythmias that appear after cerebral incidents this review aims to highlight important issues on this topic. Selected patients with the aforementioned risk factors could benefit from electrocardiographic monitoring, reassessment of the medications that prolong QTc interval, and administration of antiadrenergic agents. Further research is required in order to validate these assumptions and to establish specific therapeutic strategies.

1. Introduction

Electrocardiographic (ECG) abnormalities and arrhythmias are very common after acute cerebrovascular events, even in the absence of structural heart disease. It has been demonstrated that ECG abnormalities are evident in a significant proportion of patients (>90%) with either ischemic stroke, intracerebral hemorrhage, subarachnoid hemorrhage, or stroke of indeterminate etiology. Some of these ECG findings seem to be associated with the specific type of stroke. Interestingly new arrhythmias can be found in up to 25% of patients with no previous evidence of abnormal ECGs [1].

It has been shown that the incidence of cardiac arrhythmias after acute stroke may reach the level of 28%, with the highest rate noticed in patients with subarachnoid hemorrhage (37.5%) [2]. Of note, the risk for the development of arrhythmias in ischemic stroke was significantly higher in patients with right sided lesions compared to those with left sided lesions [2].

The aim of this comprehensive review is to provide a concise overview of available data regarding epidemiology and pathophysiology of ECG abnormalities and arrhythmias after particular cerebrovascular events and brain trauma, as well as to elucidate their relation to long-term outcome. Sources included MEDLINE and EMBASE (last search update performed on 30 December 2011). The search strategy was based on the combination of the following terms: stroke; intracranial hemorrhage; subarachnoid hemorrhage; brain trauma; ECG abnormalities; cardiac arrhythmias; autonomic nervous system; sympathetic response; and outcome. References of retrieved articles were also screened. All the available data are summarized in Table 1.

2. Pathogenesis of cerebrogenic cardiac injury

Already by the late 19th century increased intracranial pressure had been related with the reduction of heart rate and hypertension, a response later known as the “Cushing's reflex” [3]. Bradycardia in

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Table 1
Summary of the literature data.

Author (year)	Brain lesion (No. of patients)	ECG abnormality	Arrhythmia	Time frame	ANS modification	Prognosis
Sander et al. (2001)	Insular lesions in acute ischemic stroke	QTc prolongation	–	24-h ECG	Related with increased norepinephrine levels	Impaired 1-year outcome (mRS and Barthel index)
Afsar et al. (2003)	Acute ischemic (30) and hemorrhagic (6) stroke	QTd, QTcd	–	24-h ECG 72-h ECG	–	Increased QTd related with large infarcts on 24-h ECG and right lesions on 72-h ECG
Christensen et al. (2005)	Acute ischemic stroke (692)	A-V block ST depression ST elevation Inverted T-wave	AF	Admission ECG 12–24 h telemetry	–	Impaired 3-month outcome (mRS)
Christensen et al. (2005)	Insular lesions in acute stroke (43)	A-V block Inverted T-wave	AF Ectopic beats	12–24 h ECG	–	Impaired 3-month outcome (mRS)
Stead et al. (2009)	Acute ischemic stroke (345)	QTc prolongation	–	Admission ECG	–	Impaired 3-month outcome (mRS)
Mulcahy et al. (2010)	Acute ischemic stroke (45)	QTd	–	Pre-stroke ECG, post-stroke ECG	–	No significant QTd prolongation
Chao (1995)	Brainstem hemorrhage (1)	QT prolongation ST-T changes T-wave alternans	TdP (several episodes)	24-h ECG 96-h ECG	–	–
Maramattom (2006)	Supraprentorial cerebral hemorrhage (110)	QTc prolongation ST-T changes 1st degree heart block	Sinus bradycardia AF, Af VPC	Continuous ECG monitoring	–	60 day mortality not related to ECG changes
Van Bree (2010)	Non traumatic, intraparenchymal ICH (31)	QTc prolongation ST-T changes Inverted T-wave	Sinus bradycardia	48-h ECG	–	QTc prolongation related with insular involvement, intraventricular blood and hydrocephalus
Di Pasquale et al. (1987)	SAH (120)	ST changes T-wave abnormalities Prominent U-waves QT prolongation	Benign and malignant (TdP, Vf, VF) arrhythmias	24 h Holter monitoring	–	QTc prolongation and hypokalemia are associated with serious ventricular arrhythmias
Manninen et al. (1995)	SAH (70)	T-wave abnormalities ST changes Q waves	Sinus bradycardia Sinus tachycardia VPC	–	–	Related with increased intracranial blood or intracerebral clots, not with outcome
Zaroff et al. (1999)	SAH (58)	T-wave inversion ST elevation ST depression Q waves	–	–	–	Related with more impaired neurological status, not with mortality
Hirashima et al. (2001)	aSAH (118)	T-wave inversion QT prolongation ST elevation ST depression	–	Admission ECG	–	Related with the amount of blood on quadrigeminal cistern and right sylvian fissure
Kawahara et al. (2003)	aSAH (42)	QT prolongation U waves present ST depression AV-block	Sinus tachycardia Sinus bradycardia SVPC VPC	Admission ECG 48-h ECG Holter monitoring on admission	Elevated plasma catecholamine, higher HF component and lower LF/HF ratio in the acute rather than in the chronic phase	In the acute phase of SAH both sympathetic and vagal activity are enhanced
Fukui et al. (2003)	aSAH (100)	QTc prolongation	–	Admission ECG	Related with higher serum Glc, adrenaline, noradrenaline, dopamine, ADH and hypokalemia	–
Collier et al. (2004)	tSAH (104)	QTc prolongation	–	24-h ECG	–	Relates to the severity of the tSAH
Sakr et al. (2004)	aSAH (159)	T-wave alterations QTc	Sinus bradycardia Sinus tachycardia	24-h ECG	–	Only ST depression associated with impaired outcome (GOS)

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