Research Article

A study of incidence of osborn waves in patients with subarachnoid hemorrhage

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Abstract

Introduction: The J wave or Osborn wave is the most characteristic E.C.G. manifestation of hypothermia. There have been very few reports of the presence of Osborn waves in patients with SAH. The mechanism of Osborn waves in non-hypothermic conditions like SAH is not yet known. Gussak *et al* have hypothesized that abnormality within the autonomic nervous system probably responsible for these changes. **Aims and objectives**: to study of incidence of Osborn waves in patients with subarachnoid hemorrhage. **Material and Method:** All he patients with cebrovascular accidents were included in the study. The study population consisted of 14 cases of subarachnoid hemorrhage, 22 cases of cerebral infraction and 22 cases of cerebral hemorrhage and. A 12 lead ECG was taken on the day of admission in all patients of subarachnoid hemorrhage and the finding of Osborn waves were noted. **Results:** Total 14 cases of subarachnoid hemorrhage and out of them 93% cases showed abnormal ECG pattern. 50% of patients with subarachnoid hemorrhage showed Osborn waves in the E.C.G. There was only one cases of primary sub arachnoids hemorrhage and it showed Osborn waves in the E.C.G. It was seen that 46% of patients with S.A.H. secondary to intracerebral hemorrhage had Osborn wave in the E.C.G. **Conclusion:** Osborn waves were seen in 50% of patients with subarachnoid hemorrhage. The presence of Osborn waves can be used as a diagnostic pointer to subarachnoid hemorrhage.
Keywords: Osborn wave, subarachnoid hemorrhage.

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INTRODUCTION

The J wave or Osborn wave is the most characteristic E.C.G. manifestation of hypothermia. The Osborn wave is a hump like deflection which occurs at the junction of the distal limb of QRS complex with the ST segment. It is most prominent in leads oriented towards there left ventricle. It may be small or almost imperceptible in the standard and unipolar limb leads where it may manifest as a widening and ledging of the base of the QRS complex thus resembling bundle branch block. There have been very few reports of the presence of Osborn waves in patients with SAH. Osborn or J wave which is the most characteristic E.C.G. sign of hypothermia was first reported by Tomashewski[1] in 1938 in an accidentally frozen man. Osborn in 1953 attributed the J wave to acidosis and called it the current of injury. Abbott *et al*[2] described the non specific camel hump sign in patients with head injury and in SAH. DE Sweit[3] described changes simulating hypothermia in the E.C.G. in patients with SAH. Sridharan *et al*[4] (1983) found the J wave in a few patients with metastatic squamous cell carcinoma with hypercalcaemia. Syed Ameen, Ahmed and K.N. Viswanathan[5] analyzed 120 cases of CVA of whom 51 cases had stroke with SAH (either primary or secondary). Osborn waves in E.C.G. were detected in 71% of patients with SAH. Their study highlighted the importance of the presence of Osborn waves as a diagnostic pointer to SAH. Stroke whether ischemic or hemorrhagic induces cardiac

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damage by non ischemic mechanisms. The evidence derives from autopsy studies and investigation of E.C.G, cardiac enzyme changes and plasma catecholamine changes after stroke. The mechanism of Osborn waves in non-hypothermic conditions like SAH is not yet known. Gussak *et al*[6] have hypothesized that abnormality within the autonomic nervous system probably responsible for these changes.

AIMS AND OBJECTIVES

To study of incidence of Osborn waves in patients with subarachnoid hemorrhage.

MATERIAL AND METHOD

The present cross sectional study was conducted in the Department of General Medicine, Aarupadai Veedu Medical College Hospital, Pondicherry during 2011-2013. All the patients with cerebrovascular accidents were included in the study, except patients with past history of cardiac disease or known hepatic or renal disorder or on drugs like Digitalis and antidepressants. Thus a total 58 cases of cerebrovascular accidents were recorded during the study duration. Out of total 58 cases, 14 cases were of subarachnoid hemorrhage whereas 22 consisted of cerebral infraction and 22 cases of cerebral hemorrhage. Recording of detail history was done and Initial assessment included Serum electrolytes, blood urea and sugar estimation. A CT scan was taken within 48-72 hours after admission in all the patients. The diagnosis of infarction, haemorrhage and subarachnoid haemorrhage was made on the basis of the CT scan. A 12 lead ECG was taken in each individual at a paper speed of 25 mm per second. Adequate precautions were taken to eliminate the external disturbances. From these tracings were obtained the rate, PR interval, QRS interval and QT interval. The Q-Tc interval was calculated according to Bazzett's formula. The P wave amplitude, pathological Q waves, ST segment changes, T wave Changes and U waves were noted from these tracings. In all the patients of subarachnoid hemorrhage the presence of Osborn wave was traced and noted. The Osborn waves were classified according to primary and secondary subarachnoid hemorrhage.

RESULTS

Table 1: various types of cerebrovascular accidents in the study group

Study Group	Total no. of Cases	Abnormal ECG
Cerebral Infarction	22 (100%)	12 (55%)
Cerebral Haemorrhage	22 (100%)	20 (91%)
Subarachnoid Haemorrhage	14 (100%)	13 (93%)
Total	**58 (100%)**	**45 (77.59%)**

In the present study there were total 14 cases of subarachnoid hemorrhage and out of them 93% cases showed abnormal ECG pattern.

Table 2: Incidence of osborn wave in patients with subarachnoid hemorrhage

Study Group	Total No. of Cases	Osborn Wave	Percentage
Primary SAH	1	1	100%
Secondary SAH	13	6	46%
Total	**14**	**7**	**50%**

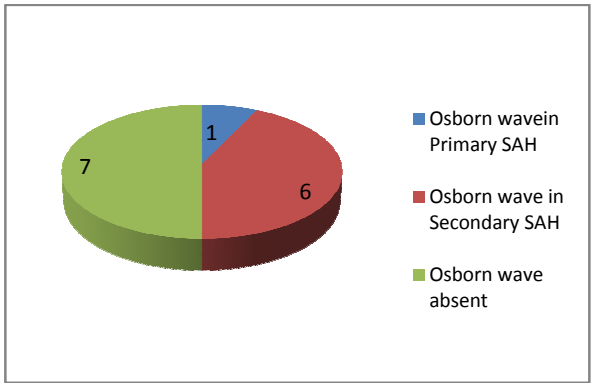

Figure 1: Osborn wave in patients with subarachnoid hemorrhage

It was observed that 50% of patients with subarachnoid hemorrhage showed Osborn waves in the E.C.G. There was only one cases of primary sub arachnoids hemorrhage and it showed Osborn waves in the E.C.G. It was seen that 46% of patients with S.A.H. secondary to intracerebral hemorrhage had Osborn wave in the E.C.G.

Table 3: Distribution of patients according to various ECG changes

ECG change		Subarachnoid Haemorrhage (n=14)
ST segment changes	ST Segment Elevation	1 (7.14%)
	ST Segment Depression	1 (7.14%)
T wave changes	T Wave Inversion	3 (21.43%)
	Tall T wave	2 (14.29%)
Prolonged Q-Tc interval		6 (42.86%)
Pathological Q waves		0 (0.00%)
Abnormal U waves		1 (7.14%)

It was observed that majority of the patients showed T wave changes (35.72%) and Q-Tc changes (42.86%) in the study. It was followed by ST changes.

DISCUSSION

In the present study we studied the incidence of Osborn waves in patients with subarachnoid hemorrhage. It was observed that there were total 14 cases of subarachnoid hemorrhage in the study. Abnormal ECG findings were present in 93% cases of subarachnoid hemorrhage. Similar findings were reported by Arruda and Leacerda[7],

who reported incidence of ECG abnormality in 100% patients with subarachnoid hemorrhage, had an abnormal ECG. In the present study it was observed that Osborn waves were present in 50% of patients with subarachnoid hemorrhage. Viswanathan *et al*[5] demonstrated Osborn waves in 71% of patients with subarachnoid hemorrhage. The variation is probably as a result the larger number of patients in their series. Nevertheless the finding of Osborn waves in our patients with subarachnoid hemorrhage is highly significant as it draws attention to a finding only mentioned in Western literature. Our study group of subarachnoid hemorrhage consisted of one case of Primary S.A.H. and 13 cases of Secondary S.A.H. The Primary S.A.H. was aneurysms of anterior communicating artery. Osborn waves were seen in this patient. Traditionally the Osborn waves of hypothermia are best seen in precordial leads and only rarely they are seen in frontal leads. In our series Osborn waves were best seen in frontal leads especially in lead aVF. Along with Osborn waves other ECG changes were also observed in the study. Prolonged Q-Tc interval (42.86%) was observed most commonly in the patients with subarachnoid hemorrhage. Arruda and Lacerda[7] also observed similar findings in their study. ST segment and T wave changes were also most commonly found in subarachnoid hemorrhage. These findings are consistent with the study by Fentz and Formsen[8] and Kreus *et al*[9]. Several mechanisms for the occurrence of ventricular arrhythmias in association with the Osborn waves have been proposed. The Osborn waves provide an index of the presence of a prominent notch in the ventricular epicardium, with a more negative potential at the end of phase 1 of the action potential. As the termination of phase 1 shifts to negative, the availability of ICa is diminished, and outward currents may overwhelm the active inward currents, resulting in a loss of the action potential dome. Heterogeneous loss of the epicardial action potential dome induces a marked increase in the dispersion of repolarization and phases 2 reentry, which can be responsible for sustained ventricular arrhythmias. Accentuation of the epicardial action potential notch, which can lead to phase 2 reentry, has been demonstrated in canine epicardium exposed to hypothermia[10] increased [Ca2+]o,[11] simulated ischemia,[12] and sodium channel blockers known to augment the J wave in Brugada syndrome.[13,14] Triggered automaticity is the other proposed mechanism for ventricular arrhythmias in patients with Osborn waves. Intracellular Ca2+ overload develops in several conditions which can cause Osborn waves such as hypothermia, myocardial ischemia and hypercalcemia. Early or delayed after depolarizations are likely to occur and form the basis for triggered activity due to the transient inward oscillatory current in Ca2+ overloaded cells.[2,11,13] An autonomic imbalance which could attend myocardial ischemia as well as neurological disorders may be another precipitating factor of ventricular arrhythmias.

CONCLUSION

Osborn waves were seen in 50% of patients with subarachnoid hemorrhage. The presence of Osborn waves can be used as a diagnostic pointer to subarachnoid hemorrhage.

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