How often can an embolic stroke be diagnosed clinically? a clinicopathological correlation

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Summary: A retrospective case note survey of 103 autopsy proven cases of cerebral infarction was carried out to assess how often a cardiac source for embolism had been correctly suspected on clinical grounds. Only 61% of 46 patients with cardiac embolic sources were so identified. Cases of rheumatic heart disease and bacterial endocarditis were more frequently identified than cases of mural thromboembolism from ischaemic heart disease. This relative failure of unaided clinical diagnostic criteria probably accounts for the discrepancy between the autopsy evidence of cardiac embolism as a cause of stroke (about 40%), and clinical studies (about 20%).

Introduction

Embolism from the heart is a common cause of cerebral infarction. The true incidence is difficult to define. There is conflict between clinical studies (Carter, 1957; Groch *et al.*, 1961; Kurtzke, 1976), most of which suggest that only 5–20% of strokes arise from cardiac emboli, and post-mortem data which show that as many as 50% of fatal strokes are so caused (Blackwood *et al.*, 1969; Torvik & Jorgensen, 1969). It is possible that cardiac embolic strokes are more frequently lethal so biasing the results of autopsy studies but it also seems likely that clinical detection is faulty. Only Gautier & Morelot (1975) have found clinical evidence to suggest a comparable high percentage of emboli from the heart.

In the clinical studies, the frequency with which a diagnosis of cardiac embolism is made depends both on the population studied and on the difficulties in detecting potential sources of emboli using only clinical or non-invasive investigations. Furthermore, the detection of a cardiac lesion as a potential source of embolism does not necessarily mean that embolism has occurred. This is a particular problem in the elderly where cardiac and cerebrovascular disease commonly coexist.

The present study was designed to analyse the clinical details of patients whose post-mortem findings have already been published (Blackwood *et al.*, 1969), in order to see how disparate were the clinical and

post-mortem signs of cardiac disease. Of the 105 patients in that study, 48 were thought to have had cardiac emboli, and 57 cerebral infarcts unrelated to cardiac disease. Cerebral infarction was considered to be due to embolism from the heart only when a clear source for embolism was found. Thirty of the 48 cases also had evidence of emboli in the spleen and kidney. Fifty seven cases in which there was no evidence of cardiac emboli were called 'thrombotic', though some probably had artery to artery embolism from demonstrable atheroma in major neck vessels.

Methods

The clinical details of the 105 patients reported by Blackwood *et al.* (1969) who had been seen at The National Hospital, Queen Square, over the period 1909–1964 were analysed. Unfortunately due to inadequate data two sets of notes had to be discarded. There remained 46 cases due to cardiac emboli, 7 of whom had the post-mortem findings of bacterial endocarditis, 11 mural thrombus over a myocardial infarct, 3 isolated atrial fibrillation with emboli, and 25 valvular heart disease, which comprised rheumatic, non-metastatic and other non-bacterial thrombotic endocarditis.

The clinical data collected were used to answer the following questions: (1) How commonly and in which groups were the presence of emboli suspected on clinical grounds? (2) How useful was the medical history and the clinical examination of the cardiovascular system in detection of embolic sources?

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Table I Clinical suspicion of embolism and post-mortem confirmation according to cardiac pathology in cases of cerebral embolism

	<i>Post-mortem diagnosis</i>				<i>Total</i>
	<i>Bacterial endocarditis</i>	<i>Valvular heart disease</i>	<i>Mural thrombus</i>	<i>Lone atrial fibrillation with clot</i>	
Emboli suspected clinically	3	18	4	3	28
Emboli not suspected clinically	4	7	7	0	18
Total	7	25	11	3	46

Relevant events in the past comprised a history of rheumatic or valvular heart disease, ischaemic heart disease or cardiac failure. Auscultation was considered to be abnormal if there was either a gallop rhythm or the signs of classical mitral or aortic valvular dysfunction. The following were not considered abnormal: (a) soft systolic murmur at the base of the heart; (b) apical systolic murmur; (c) tic tac heart sounds; (d) isolated accentuated or softened heart sounds. Insufficient electrocardiograph (ECG) records were available for review to make their assessment worthwhile.

Results

Table I shows that emboli were suspected in 28 (61%) of 46 cases. This occurred far more commonly in those with subsequently proven valvular heart disease, where 18 out of 25 cases were thought to have had embolic strokes, than in those with bacterial endocarditis (3 out of 7).

In the embolic group, 20 (43%) had definite auscultatory abnormalities. In the thrombotic group there were 2 patients (4%) with abnormal auscultatory findings, one with a mid diastolic murmur and presystolic murmur and the other with a soft diastolic

murmur over the aortic area; both had only left ventricular dilatation at post-mortem with no signs of embolism.

The presence of auscultatory abnormalities was again most useful in those with valvular heart disease (Table II), although even in 9 of these 25 cases no abnormality was elicited on examination. Of these 9, 2 had a past history of rheumatic fever, 2 were in rapid atrial fibrillation but the other 5 had no clinical evidence of abnormality. Four out of 7 cases with bacterial endocarditis and none of those with left ventricular mural thrombi had auscultatory abnormalities. Using the definitions described in the methods, 4 patients in the embolic and 18 in the thrombotic group were considered to have non-significant auscultatory abnormalities.

Atrial fibrillation was present in 11 (14%) of the embolic group compared with 1 patient (2%) in the thrombotic group. As expected atrial fibrillation was most common in those with valvular heart disease, although even in this group only 20% were in atrial fibrillation (Table III). Three patients were in lone atrial fibrillation in the embolic group compared with 1 in the thrombotic group.

A history of heart disease was obtained in 21 (46%) of the embolic group, the majority in those with valvular heart disease and bacterial endocarditis. It

Table II Prevalence of abnormal auscultatory findings according to type of cardiac disease encountered at post-mortem, in cases of cerebral embolism

	<i>Post-mortem diagnosis</i>				<i>Total</i>
	<i>Bacterial endocarditis</i>	<i>Valvular heart disease</i>	<i>Mural thrombus</i>	<i>Lone atrial fibrillation with clot</i>	
Abnormal auscultation	4	16	0	0	20
Normal auscultation	3	9	11	3	26
Total	7	25	11	3	46

Table III Prevalence of atrial fibrillation according to type of cardiac disease detected at post-mortem, in cases of cardiac embolism

	<i>Post-mortem diagnosis</i>				<i>Total</i>
	<i>Bacterial endocarditis</i>	<i>Valvular heart disease</i>	<i>Mural thrombus</i>	<i>Lone atrial fibrillation with clot</i>	
Atrial fibrillation	1	5	2	3	11
Sinus rhythm	6	20	9	0	35
Total	7	25	11	3	46

was rare in those with left ventricular mural thrombus (Table IV). In the thrombotic group, 5 patients (9%) gave a history of ischaemic heart disease and 2 (3%) of rheumatic fever.

If all three clinical parameters, namely auscultatory abnormalities, atrial fibrillation and past medical history were considered, then at least one abnormality was present in 35 patients (76%) in the embolic group compared with 9 patients (16%) in the thrombotic group ($\chi^2 = 37$, $P < 0.001$) (Table V).

Even here, however, 7 of the 11 patients with left ventricular mural thrombus had an entirely negative

past medical history and cardiovascular examination.

If a history of cardiac disease, or the finding of atrial fibrillation or auscultatory abnormalities were considered indicative of embolism, a false positive rate of 20.5% and a false negative rate of 18.6% would have been found.

Discussion

The purpose of this study was to ascertain the value of the clinical history and examination in the detection of a cardiac source of emboli. Of the group proven at

Table IV History of heart disease in different types of cardiac disease detected at post-mortem in cases of cerebral embolism

	<i>Post-mortem diagnosis</i>				<i>Total</i>
	<i>Bacterial endocarditis</i>	<i>Valvular heart disease</i>	<i>Mural thrombus</i>	<i>Lone atrial fibrillation with clot</i>	
History of heart disease	4	13	2	2	21
No history of heart disease	3	12	9	1	25
Total	7	25	11	3	46

Table V Combined clinical criteria of possible cardiac source of embolism compared with post-mortem findings in cases of cerebral embolism

	<i>Post-mortem diagnosis</i>				<i>Total</i>
	<i>Bacterial endocarditis</i>	<i>Valvular heart disease</i>	<i>Mural thrombus</i>	<i>Lone atrial fibrillation with clot</i>	
Atrial fibrillation or past medical history or auscultation abnormal	6	22	4	3	35
All above negative	1	3	7	0	11
Total	7	25	11	3	46

post-mortem to have cardiac emboli by Blackwood *et al.* (1969), only 61% were thought clinically to have had an embolic stroke prior to death. Thus, though autopsy showed 45% of fatal strokes to be due to cardiac emboli, clinical detection was only achieved in 24%, a figure in keeping with clinical surveys from a similar era. The discrepancy between clinical and autopsy data is thus seen to be largely explicable on the basis of poor clinical diagnosis.

As would be expected, abnormal auscultatory findings and atrial fibrillation were particularly useful in the detection of possible cardiac emboli. A medical history of cardiac disease was of some, albeit rather limited, value. Using all these criteria, however, there was a false negative rate of 18.6% and a false positive rate of 20.5%. These parameters were useful in the detection of rheumatic heart disease and bacterial endocarditis. With the decrease in incidence of valvular heart disease since the period covered by the cases reported by Blackwood *et al.* (1969), it is likely that a greater percentage of emboli will be due to lone atrial fibrillation (Gautier & Morelot, 1975) and mural thrombi following myocardial infarction. The study emphasizes the difficulty in detecting clinically the small percentage of mural thrombi which produce symptomatic embolic events. Thus, bedside clinical criteria alone failed to pick up 7 out of 11 cases (64%). Unfortunately, routine electrocardiograms were not available on the majority of these patients as Blackwood's study covered the period 1909–1964; furthermore, 3 of the 11 patients were drowsy on admission and not able to give a full history. Of importance is the time of occurrence of cerebral emboli after an acute myocardial infarct. While 84% of emboli occur in the

first month, 14% occur in the second and third months when little or no residual clinical evidence of their myocardial infarction may be present (Bean, 1938).

If routine electrocardiograms suggest a prior myocardial infarction, can a mural thrombus be detected non-invasively? Donaldson *et al.* (1981) have shown that M-mode echocardiography is useful in detecting any valvular abnormality or atrial tumour but is of limited use in the detection of thrombi in contact with the atrial or ventricular wall. Two dimensional echocardiography is superior but even here thrombi usually need to be greater than 2 cm in size to be detected (Ports *et al.*, 1978), and can be missed due to acoustical similarity between small thrombi and surrounding blood (Asinger *et al.*, 1981).

Radionuclide scanning may be able to identify akinetic segments or aneurysms which increase the likelihood of the presence of intraventricular thrombus. Labelled platelet scanning, CT scanning and digital subtraction angiography all promise to increase the detection rate.

Knopman *et al.* (1982) and Burnett *et al.* (1984) have emphasized that the information from two dimensional echocardiography can substantially alter the management of patients with cerebral infarction even though the yield in cases without clinical signs of cardiac disease is low. It is clear therefore that the detection of mural thrombus by clinical criteria remains a major problem especially since anticoagulants are probably indicated for cardiac embolic stroke. It is evident from our study that clinical assessment misses many cardiac sources of emboli, and that an improved detection rate depends on current and future developments in investigative technology.

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