

## Case report

# Cerebral arteriovenous malformation mimicking acute coronary syndrome

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### Abstract

*We report a case of cerebral arteriovenous malformation in a 34 year-old male patient presenting with chest pain. Electrocardiographic findings showed ST elevations in the precordial leads. However, a coronary angiogram showed no coronary lesions. Laboratory tests suggested that troponin and cardiac enzymes were within normal limits. Further investigation led to the diagnosis of a cerebral arterio-venous malformation occupying the left temporal and parietal lobes. The chest pain may be explained by the occurrence of a complex partial seizure.*

**Key words :** Arterio-venous malformation ; chest pain ; epilepsy ; electrocardiographic modifications.

A 34-year-old male presented to the emergency department at 2 o'clock in the morning with dizziness and left thoracic pain. His past medical history was unremarkable and he had no history of ischaemic heart disease in his family. He smoked 20 cigarettes per day but had no other risk factors for vascular disease, although his cholesterol level was unknown. He did not take cocaine, or any derivatives of ergot.

On examination, the patient had a blood pressure of 95/50 mmHg, a pulse of 80/min regular and an axillary temperature of 36.6°C. He was thin and his sclera was anicteric. There were no signs of lymphadenopathy. Heart sounds were normal with no audible murmurs or rubs. Breathing was also normal. The abdomen was soft and non-tender. Neurological examination revealed to be unremarkable.

The Electrocardiogram (ECG) on admission (Fig. 1) showed a sinus rhythm of 89 bpm. The P wave morphology and axis were within normal limits. The P-R interval was 172 msec and QRS duration 90 msec. The QRS axis was also within normal limits. Repolarisation abnormalities were as follows : a rise in J-point of 4 mm with ST elevation in leads V2, V3 and V4 with tall T waves. These ECG changes and chest pain still persisted 30 minutes after administration of sublingual Isosorbide dinitrate.

Laboratory findings revealed that the blood count, liver function tests, cardiac enzymes, urea and electrolytes were all within normal values. Troponin I concentration was 0.2 ng/ml (N < 2.0 ng/ml) and prolactin was 39 ng/ml (N : < 20 ng/ml) upon admission. Cocaine and cannabis derivatives were not detected in the urine sample on admission and 24h thereafter.

The chest X-ray revealed to be normal as was an echocardiogram performed 12 h after admission. An acute coronary syndrome was suspected. The patient being symptomatic, a coronary angiogram was performed. This later turned out to be normal. Symptoms improved and the chest pain regressed spontaneously, but the electrocardiogram remained unchanged. A neurologic examination was performed 24 hours after admission. Further questioning of the patient and his relatives suggested that the patient had experienced a short period of confusion with inappropriate behaviour. For example, the patient was walking automatically towards the kitchen where he stopped for a few minutes and complained of color perception. According to the informant relatives the patient was unable to reply to questions, unaware of his environment and unable to recognise them. During the neurologic examination the patient had complained of a continuous sound in both ears like that of "a flowing river". There were no clinical signs of intracranial hypertension. Physical examination revealed only a harsh bruit that was loudest in the left temporal region.

The patient was then transferred to the neurology department. In view of this clinical setting we suspected a complex partial seizure. An E.E.G. showed only theta and delta waves in the central, left parietal and left temporal region.

A computersied tomography (CT) scan of the head revealed a large arteriovenous malformation occupying the left temporal and left parietal lobe with discrete signs of compression of the homolateral ventricles. Magnetic resonance imaging (MRI) (Fig. 2) confirmed this finding showing no signs of haemorrhage. The MRI also showed a malformation occupying the left temporo-parieto-occipital

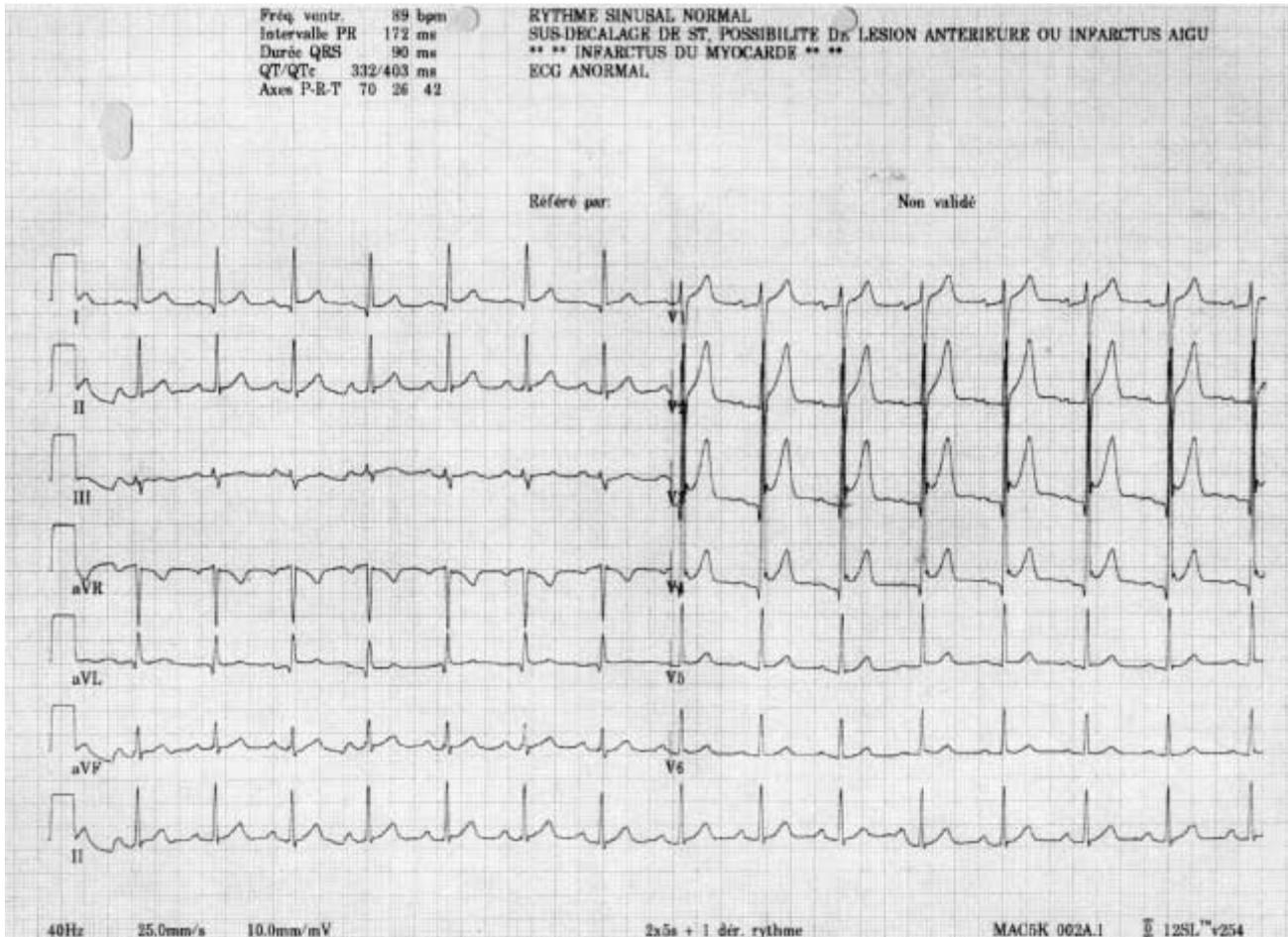


FIG. 1. — ECG repolarisation abnormalities on admission : rise in J-point of 4 mm with ST elevation in leads V2, V3, V4 with tall T waves.

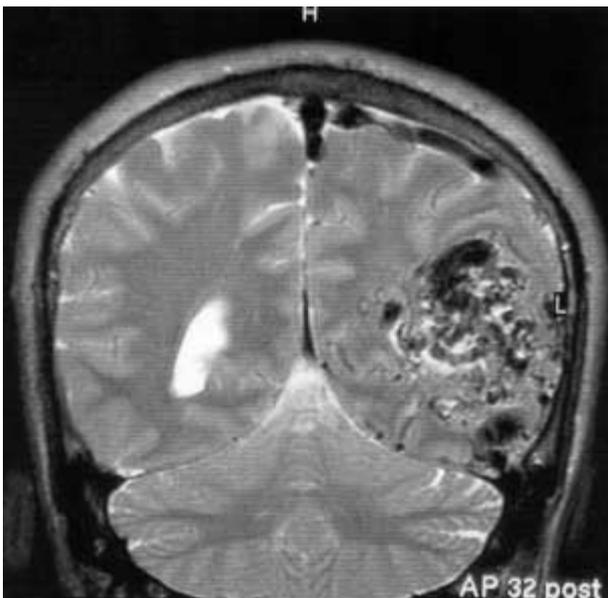


FIG. 2. — T2 weighted MRI showing a large left temporo-parieto-occipital arterio-venous malformation. The nidus of this malformation is localised in the parietal lobe. The malformation is drained by the longitudinal and transverse sinus. A cerebral angiogram also confirmed the presence of this arterio-venous malformation.

lobes with the centre of the nidus localised in the parietal lobe.

The patient was treated with valproic acid and referred to the neuroradiological department. Neurosurgical procedures were not undertaken in view of the size of the malformation. The patient is being followed by one of us in the outpatient clinic. At 2 months the patient was readmitted with generalized tonic-clonic seizure.

### Discussion

Arteriovenous malformations of the brain manifest themselves either by haemorrhage or epileptic seizures (Davidson *et al.*, 1973). Previous studies (Marriott, 1960 ; Byer *et al.*, 1947 ; Eisalio *et al.*, 1972) have indicated that intracranial processes are known to cause electrocardiographic changes. These changes may resemble abnormalities typical of myocardial ischaemia or myocardial infarction (Nakamura *et al.*, 1989). Misinterpretation of ECG can lead to erroneous diagnosis and to inappropriate treatment such as thrombolysis. Therapeutic intervention can greatly differ depending on



FIG. 3. — Cerebral angiogram showing a large temporo-parieto-occipital arteriovenous malformation with a nidus located at the parietal level.

whether ST segment elevation reflects organic obstruction of the coronary artery or vasospasm.

If consciousness is disturbed, patients with such a disorder may not necessarily complain of headache or chest pain. Before the era of CT subarachnoid haemorrhage was sometimes misdiagnosed as myocardial infarction (Levin, 1953). In our patient the ST changes evoked primarily the diagnosis of acute coronary syndrome. The CT and MRI scans showed slight compression of the ipsilateral ventricles but no radiological signs of intracranial hypertension. The mechanism of repolarisation abnormalities is unclear but autonomic disturbances in the insular cortex could be a possible mechanism (Oppenheimer *et al.*, 1992). Thus we think that the ECG modification (ST elevation) may be due to stimulation of the insular cortex. On the other hand, the behaviour and the abnormalities in color perception are compatible with a paroxysmal brain activity arising from the temporal lobe and spreading to the occipital lobe.

Electrolyte disturbances may have a role, but these values were normal in our patient. Others have reported (Conner 1970, and Shanlin *et al.*, 1988) that myocardial damage may be due to high levels of epinephrine, or follow from vasospasm of myocardial vessels (Shanlin *et al.*, 1988). We hypothesize that the diagnosis of a complex partial seizure is likely due to stimulation by the left temporal arteriovenous malformation. The ECG on admission was interpreted as an onset of an acute ischaemic event, as witnessed by tall T waves in this symptomatic young patient.

## REFERENCES

- MARRIOTT H. J. L. Coronary mimicry : normal variants, and physiologic, pharmacologic and pathologic influences that stimulate coronary patterns in the electrocardiogram. *Ann. Int. Med.*, 1960, **52** : 411-427.
- BYER E., ASHMAN R., TOTH L. A. Electrocardiograms with large, upright T waves and long Q-T interval. *Am. Heart J.*, 1947, **33** : 796-806.
- CONNER R. C. Fuscinophilic degeneration of myocardium in patients with intracranial lesions. *Br. Heart J.*, 1970, **32** : 81-84.
- DAVIDSON S., MURRAY A. FALCONER. Cerebral arteriovenous malformation causing cardiac enlargement and epilepsy : Correction after operation. *BMJ*, 1973, **2** : 754-755.
- EISALIO A., PARASALO J., HALONEN P. I. Electrocardiographic abnormalities and some laboratory findings in patients with subarachnoid haemorrhage. *Br. Heart J.*, 1972, **34** : 217-226.
- ESTANOL B. V., BAUDUI E. D., CESARMAN E., MARIN O. S. M., LOYO M., VARGAS B. L., PEREZ P. O. Cardiac arrhythmias associated with subarachnoid haemorrhage ; prospective study. *Neurosurgery*, 1979, **5** : 675-680.
- ESTANOL B. V., LOYO M. V., MATEOS J. H., FOYO E., CORNEJO A., GUEVARRA. Cardiac arrhythmias in experimental subarachnoid haemorrhage. *Stroke*, 1977, **8** : 440-447.
- JACHUCK S. J., RAMANI P. S., CLARK F., KALBAG R. M. Electrocardiographic abnormalities associated with raised intracranial pressure. *Br. Med. J.*, 1975, **1** : 242-244.
- LEVIN H. D. Non specificity of the electrocardiogram associated with coronary heart disease. *Am. J. Med.*, 1953, **15** : 480-484.
- NAKAMURA Y., KASENO K., KUBO T. Transient ST-segment elevation in subarachnoid haemorrhage. *J. Electrocardiol.*, 1989, **22** : 133-13.
- OPPENHEIMER S. M., CECETTO D. F. Cardiac organization of the rat insular cortex. *Brain Research*, 1990, **12** : 66-72.
- OPPENHEIMER S. M., GELB A., GIRVIN J. P., HACHINSKI V. C. Cardiovascular effects of human insular cortex stimulation. *Neurology*, 1992, **42** : 1727-1732.
- ZAMRINI E. Y., MEADOR K. J., IORING D. W. Unilateral cerebral inactivation produces differential left/right heart rate responses. *Neurology*, 1990, **40** : 1408-1411.
- SHANLIN R. J., SOLE M. J., RAHIMIFAR M., TATOR and FACTOR S. M. Increased intracranial pressure elicits hypertension, increased sympathetic activity, electrocardiographic abnormalities and myocardial damage in rats. *J. Am. Coll. Cardiol.*, 1988, **12** : 727-736
- WOLFHART B. A simple model for demonstration of STT-changes in ECG. *Eur. Heart*, 1993, **J. 8** : 409-416.

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