Journal of Clinical and Basic Cardiology

An Independent International Scientific Journal

Journal of Clinical and Basic Cardiology 2000; 3 (1), 67-68

Fulminant course of hypertension caused by phaeochromocytoma

Metzler B, Bilgeri R, Pachinger O

Indexed in
Chemical Abstracts
EMBASE/Excerpta Medica

Krause & Pachernegg GmbH · VERLAG für MEDIZIN und WIRTSCHAFT · A-3003 Gablitz/Austria

Fulminant Course of Hypertension caused by Phaeochromocytoma

B. Metzler, R. Bilgeri, O. Pachinger

A 58 year old man, with a well known history of coronary heart disease and arterial hypertension, was admitted to hospital complaining of severe central chest pain. At time of admission BP was significantly raised and the patient presented with clinical shock-symptoms. ECG and laboratory controls showed no signs of acute myocardial ischaemia. Despite intravenous treatment with nitroglycerine, urapidil and beta-blockers blood pressure remained persistently high. Deterioration of the patients general condition eventually required intubation and controlled ventilation. Electromechanical dissociation appeared shortly thereafter and could not be overcome by prolonged resuscitation. *Post mortem* examination revealed a large phaeocromocytoma and myocardial infarction of the posterior wall as the immediate cause of death.

It is of interest that in this case the primary onset of a formerly undiagnosed phaeocromocytoma lead to prolonged hypertension almost insusceptible to standard treatment and eventually to fatal myocardial infarction within hours of onset of symptoms. *J Clin Basic Cardiol 2000: 3: 67–8.*

Key words: phaeochromocytoma, hypertension, myocardial infarction

A 58 year old man was admitted to hospital complaining of severe central chest pain without radiation. He had a well known history of coronary heart disease (CHD) with two myocardial infarctions in 1989 and 1993. Since the last myocardial infarction there was no history of angina pectoris. Onset of chest pain occurred about four hours before admission with an immediate beginning, accompanied by dizziness, nausea, vomiting and dyspnoea.

On examination in hospital he was overweight, the pulse was regular with 62/min, and blood pressure (BP) was raised on both sides to 205/130 mmHg. There was no pathological heart murmur and there were no clinical signs of congestive heart disease (no pulmonary congestion no peripheral oedema).

The laboratory parameters were as follows: Leucocyte counts were elevated with 17.4 G/l, as well as blood-glucose with 210 mg/dl, GOT 26, GPT 30, gamma-GT 53, LDH 268 U/l, K^+ 5.6 mmol/l. Within the normal range were creatine kinase activity (64 U/l), the isoenzyme CKMB (8 U/l), troponin T, alkaline phosphatase, CRP, fibrinogen.

Consecutive controls of these parameters revealed no significant changes.

In the ECG a normofrequent sinus rhythm, with a QRS axis of 73° and regular ST-segments in all leads was shown. The only abnormality was a q-wave in leads II, III and aVF reflecting an old inferior myocardial infarction.

The chest X-ray revealed a biventricular enlargement of the heart but no signs of pneumonia, pulmonary congestion or pleural effusion.

To exclude a possible dissecting aneurysm of the aorta a computer tomography (CT) of the thorax was performed, showing multiple small peripheral pulmonary embolisms but no sign of aortic dissection.

Treatment and course

Given the known history of CHD with two preceding myocardial infarctions, the patient was treated as a hypertensive crisis with unstable angina pectoris. To lower the extensively raised BP of 250/150 mmHg at arrival the first-aid doctor administered 0.4 mg of nitroglycerine *s.l.,* followed by 25 mg urapidil i.v. and 500 mg acetylsalicylic acid i.v. On admission to hospital BP was 205/130 mmHg, therefore i.v. administration of nitroglycerine and urapidil was continued as permanent infusions as well as i.v. administration of unfractionated heparin (1000 IE/h) and the patient was transferred to the coronary care unit (CCU).

Despite lowering the BP to values of 160–180 mmHg systolic, severe chest pain persisted and was just partly reduced by administration of pethidine.

On several occasions during the hours following admission to the CCU and continous monitoring of the patient, BP fell spontaneously to hypotensive levels of 50–60 mmHg systolic within seconds (Figure 1), accompanied by cyanosis, hypoxia and signs of a general state of shock. By immediately stopping of antihypertensive therapy and infusion of plasma expander BP could be raised instantly and again reached hypertensive values within minutes after dropping, renewing the necessity of antihypertensive treatment. Several ECG- as well as laboratory controls done during that time showed no changes compared to those performed on admission. Further deterioration of the general condition of the patient with increasing dyspnoea and tachypnoea, falling oxygen saturation despite oxygen administration via a face mask and clinical shock symptoms such as pale skin and cold sweat required intubation and controlled ventilation (BIPAP-mode). Only minutes after intubation, electromechanical dissociation

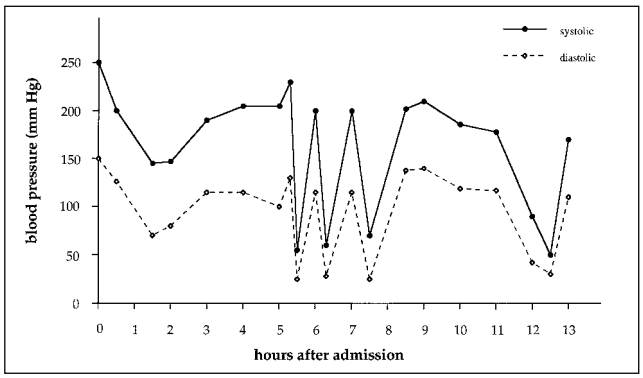

Figure 1. Thirteen-hour systolic and diastolic blood pressure monitoring graph of an antihypertensive treated patient with an unknown Phaeochromocytoma. The x axis indicates the time after admission to hospital (h); the y-axis the blood pressure in mmHg.

From the Division of Cardiology, Department of Internal Medicine, University Hospital of Innsbruck, Austria.
Correspondence to: Otmar Pachinger, M.D., Division of Cardiolgy, Department of Internal Medicine, University Hospital of Innsbruck, Anichstraße 35, A-6020 Innsbruck, Austria

(EMD) occurred, necessitating external cardiac pressure resuscitation. EMD was followed by asystolia, which could not be overcome by prolonged CPR including intravenous and intratracheal administration of high doses of catecholamines.

Autopsy findings

The autopsy showed a tumor of the right suprarenal gland with a diameter of about eighty millimetres. The histological examination showed a chromogranine and neuron-specific enolase positive phaeocromocytoma.

As cause of death the autopsy result stated an acute myocardial infarction of the posterior wall. In contrast to the CT-finding no sign of pulmonary embolism could be detected.

Discussion

Phaeochromocytomas are rarely seen tumors with an incidence of about 2 per 1 million people, they are responsible for about 0.1 % to 0.2 % of causes of high blood pressure. The diagnosis of phaeocromocytoma is typically made in young or middle aged adults. Typically, symptoms are caused of episodic catecholamine release, including paroxysmal hypertension, diaphoresis, headache, sweating and tachycardia [1, 2].

In our case, the phaeochromocytoma was undiagnosed during life, corresponding with the fact that in patients over the age of 60 years phaeochromocytomas are especially likely to produce minor or no symptoms [3].

Because the arterial hypertension was known and treated for many years, no sign was typical for a phaeochromocytoma until the day when the patient was admitted to hospital due to a hypertensive crisis. Since no indication of an acute myocardial infarction or dissecting aneurysm of the aorta was given, the patient was treated as a hypertensive crisis with unstable angina pectoris. The hypertension which was relatively refractory to medical management in connection with the extreme lability of the blood pressure, together with the pale skin and cold sweat, and also the high serum glucose level (210 mg/dl) and the elevated blood leucocyte count (17.4 G/l) retrospectively could provide some hints to the diagnosis of phaeochromocytoma.

Because hypertension is so commonly encountered in clinical practice, and phaeochromocytoma is so distinctly unusual, other criteria such as the urinary catecholamine level and their metabolites (24-h samples) should be investigated in suspected patients [4]. In our case it was not possible to do that, because of first, the short time between admission to hospital and death of the patient and second, the determination of these parameters is mostly not possible to perform routinly in CCU.

Since in our patient the computer tomography to exclude a dissecting aneurysm of the aorta was just performed for the thorax, the abdominal mass was not detected with this examination.

Another point of interest is that the autopsy showed signs of acute myocardial necrosis, which were aged between 8–10 hours. Interestingly, there was no intracoronary thrombus formation found. In contrast to that finding, consecutive controls of the ECG and CK-activity, the last done about one hour before death (about twelve hours after admission to hospital), showed no signs of acute coronary ischaemia.

Herewith we describe the first time a case, where a patient with an unknown phaeochromocytoma was admitted to hospital due to a hypertensive crisis, almost insusceptible to standard treatment, and subsequently developed a fatal myocardial infarction within hours, which also could be diagnosed just *post mortem*.

References

1. Northfield TL. Cardiac complications of phaeochromocytoma. Br Heart J 1967; 29: 588–93.
2. Lamberts R, Kreuzer H. Phaeochromocytoma-induced multiorgan failure. An internal medicine and surgical emergency. Dtsch Med Wochenschr 1996; 121:479–84.
3. O'Connor DT. Adrenal medulla. In: Bennett ID, Plum F (eds). Cecil Textbook of Medicine. 20th edition. WB Saunders, Philadelphia, 1996; 1253–7.
4. Bravo EL, Gifford RW Jr. Pheochromocytoma: diagnosis, localization and management. N Engl J Med 1984; 311: 1298–303.

Mitteilungen aus der Redaktion

Besuchen Sie unsere

zeitschriftenübergreifende Datenbank

☑ **Bilddatenbank** ☑ **Artikeldatenbank** ☑ **Fallberichte**

e-Journal-Abo

Beziehen Sie die elektronischen Ausgaben dieser Zeitschrift hier.

Die Lieferung umfasst 4–5 Ausgaben pro Jahr zzgl. allfälliger Sonderhefte.

Unsere e-Journale stehen als PDF-Datei zur Verfügung und sind auf den meisten der marktüblichen e-Book-Readern, Tablets sowie auf iPad funktionsfähig.

☑ **Bestellung e-Journal-Abo**