Case Report

Protracted neurocardiogenic reaction

Jiří Hlas*, Miloš Holub, Petr Šimek, Zdeněk Klimsa

Kardiovaskulární centrum pro dospělé Nemocnice Jihlava, Czech Republic

1. Case description

A 36-year-old male, obese hypertensive, smoker, with a positive family history of CAD was referred to our cardiology department for urgent coronarography from the regional hospital with a history of 2 h lasting chest pain and a suspicion of STEMI of the inferior wall.

On arrival at our coronary unit his chest pain persisted and the ECG showed only non-specific changes in the area of the inferior wall (negative T waves) without any abnormalities in the initial laboratory examination, including cardiac enzymes, ions, renal and liver functions. The physical examination was without any remarkable findings and the patient was without any signs of heart failure, without any neurological deficit.

Due to persistent chest pain in a risk patient we decided to perform left cardiac catheterization—with a physiological finding in the coronary arteries and ventriculogram. Repeated cardiac markers were negative, myocardial infarction excluded.

The postprocedural course was initially without any complications, the patient had no subjective complaints, he was in stable haemodynamics status, the intervened right groin was fine, without hematoma or resistance, ECG monitoring with sinus rhythm of appropriate rate and O₂ saturation was normal.

According to our standard procedure we removed a 5F sheath from the right groin after 3 h and we applied specific compressive device called “femostop” after 5 min lasting manual compression.

Seventeen minutes after removal of the sheath (e.g. with applied femostop) a vagal reaction occurred—a sudden weakness, sweating, nausea, fast shift to bradycardia followed by asystolia (see Fig. 1).

Immediately we started indirect cardiac massage, ventilation with Ambu-Vac and administered i.v. atropine (total dose 2 mg), i.v. adrenaline (1 mg), i.v. colloids (250 ml of 6% hydroxyethyl starches). After 3 min a recovery of the vital functions occurred, including sinus rhythm restoration and increased blood pressure to 124/74. However, impairment of consciousness persists—immediately after restoring breathing and circulation an ament condition with aggressiveness
(restlessness, screaming, jeopardizing the staff) occurred which required pharmacological sedation (i.v. diazepam 10 mg), followed by sopor and coma vigile (literally from the decursus "eyes open, do not fix, without photoreaction, without reaction to algesic stimuli").

The neurologist concluded a suspicion of embolism into the vertebrobasilar system with hypoxic–ischemic encephalopathy. The bedside echocardiography, brain CT and ultrasound of the carotid arteries were negative; the laboratory results were without any pathological finding. Crystalloids, nootropics, prophylactic LMWH were administered.

A slight improvement of the overall condition occurred 15 min after the occurrence of the vagal reaction—the patient began responding when addressed, he could move his limbs if requested, but he was not still capable of verbal contact. Due to persistent qualitative consciousness impairment a transfer to the neurological intensive care unit was performed. EEG and repeated contrast brain CT were negative. The neurological condition was restoring very slowly and a complete normalization of the neurological functions occurred 36 h(!) after the vagal reaction.

After carotid massage and electrophysiological examination (performed at a superior department) we close the condition as atypical impairment of consciousness during severe vagal reaction. The patient is without any problems after discharge and during the subsequent follow-up till now.

2. Discussion

Vasovagal reactions belong to relative common complications related to cardiac catheterization. These complications are seen in 1.9–3% of patients undergoing catheterization procedures. They are mostly in a relationship with puncture of femoral/radial artery (80%) or with sheath removal (16%) and can be triggered by pain, anxiety, hypovolemia and patients' low knowledge about the nature of the examination [1]. Treatment usually consists of removal of the painful stimulus, volume administration, atropine (0.5–1 mg intravenously), and if necessary—adrenaline.

We can meet mild (nausea, paleness) and moderate (bradycardia or/and hypotension) forms in any department of cardiology quite often, but severe vasovagal complications are very rare. These conditions evolve into acute pulmonary edema, myocardial infarction or asystolia [2]. Especially patients with critical coronary or valvular disease are at high risk—even irreversible decompensation from vagally-mediated hypotension may occur. Rapid reversal of hypotension is crucial in this setting.

There has been published a lot of references in the world regarding periprocedural vasovagal complications, including cardiac catheterization too, but only few case reports with severe vasovagal complication requiring cardiopulmonary resuscitation. Some periprocedural vasovagal complications have been mentioned in case reports related to spinal analgesia [3,4], arthroscopy [5], but there was no reference related to cardiac catheterization found. Therefore we present this case report which is interesting especially because of prolonged changes in neurological status of relatively healthy man with no history of syncpe. We wanted to point out that not every vasovagal complication following catheterization procedures has a benign characteristic.

REFERENCES