Delirium due to contrast toxicity after coronary angioplasty

Koroner anjiyoplasti sonrası kontrast toksisitesine bağlı deliriyum

Neurological complications appear to be very rare after coronary angiography, occurring at a rate of about 0.6% (1, 2). Delirium or acute confusion state is a transient global disorder of cognition.

A 64-year-old man was referred to our clinic for coronary angiography after a non-ST elevation myocardial infarction. Upon presentation, the patient was conscious, oriented and cooperative, without any chest pain. All vital signs were stable before the coronary procedure. The coronary angiography was performed and showed 99% stenosis of the major obtuse marginal branch (OM) of the circumflex (CX). In the same session, a 3.018 mm bare-metal stent was successfully implanted in the major OM subtotal lesion without peri-procedural complications. Iopromide 120 cc (Ultravist® 370, Schering AG) was used throughout the the procedure. After the procedure, his vital signs and neurological status remained stable. Forty-five minutes after the procedure, the patient became agitated and started to sing nonsense words. He experienced disorientation to time and place, could not recognize relatives, and repeatedly requested to get out of bed. During the night, 12 hours after the first symptoms occurred, the patient experienced visual hallucinations. There were no focal neurological deficits. Laboratory values, blood gas analysis, and vital signs were checked again to determine the etiology of the patient's delirium. However, no abnormal findings were observed. Haloperidol (2.5 mg) was administered intravenously as initial treatment, and continued orally thereafter. Improvement was observed in the patient's state of delirium 24 hours after the onset of symptoms. A diffusion magnetic resonance imaging, performed after the patient was stable, did not show any abnormalities. Two days after the onset of agitation, the patient's mental status was returned to normal, although he had no recollection of his previous state of delirium.

Neurological complications of coronary angiographic procedures are uncommon, varied, and include central nervous toxicity caused by contrast agents, as well as, ischemic and hemorrhagic stroke (1-4). Contrast medium neurotoxicity is thought to be caused by the osmolality, lipid solubility, viscosity, and ionic properties of the contrast agent. The contrast medium opens tight capillary connections and passes the blood-brain barrier by increasing endothelial pinocytosis. Then reaches the cerebral cortex and affects the neuronal membrane (5). Delirium is a mental disorder of acute onset and fluctuating course which is characterized by disturbances in consciousness, orientation, memory, perception, and behavior. Delirium occurs often those who previously experienced dementia and appears to be independently associated with significant increases in functional disability, length of hospital stay, rates of admission to long-term care institutions, mortality rates, and healthcare costs. Moll et al. (5) described two patients who experienced severe agitation and hyperventilation after coronary angiography. The first and second cases occurred 20 minutes and 2 hours after coronary angiography, respectively. Both patients completely recovered after 12-24 hours. Our patient's symptoms began 45 minutes after the procedure. After 24 hours the symptoms of delirium were reduced, and after 48 hours, complete recovery was observed. Our patient received Iopromide, while the two cases reported by Moll et al. (5) received Ioversol. Both are types of nonionic contrast media with similar osmolality and similar iodine content. Benzodiazepines and haloperidol were used in the previous cases, while only haloperidol was administered to our patient. A small number of cases in the literature report patients developing contrast neurotoxicity and there is not enough information on patient management.

After coronary angiography, neurological symptoms and delirium may occur even without a cerebrovascular event. Temporary delirium due to contrast toxicity should be kept in mind by clinicians.

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Clinical analysis of neonates with congenital heart disease in the neonatal intensive care unit: a 5-year experience

Yenidoğan yoğun bakım ünitesinde doğumsal kalp hastalıklı yenidoğanların klinik analizi: 5 yıllık deneyim

Congenital heart diseases (CHD) are the most common form of major birth defects (1). The occurrence rate of CHD is approximately 4.5% of neonates admitted to the neonatal intensive care unit (NICU) (2). Data of 3334 neonates were scanned and 394 of them who had a complete two-dimensional and pulsed Doppler echocardiographic examination by a single pediatric cardiologist were included in this study. The neonates presenting with central cyanosis (<85% SpO_2 with pulse oximetry), heart murmur, feeding or breathing difficulties first underwent a neonatologist examination, then after a pediatric cardiologist examination with the clinical suspicion of CHD. Clinical characteristics of the neonates who had normal echocardiographic findings (named as group 1) were compared with the neonates with CHD (named as group 2). Congenital heart disease frequency was found to be 4% (136/3334) in our NICU population. The relative percentage of acyanotic (n=136) and cyanotic cardiac disorders (n=30) was 81.9% vs 18.1% respectively. There was no significant difference between group 1 and 2 in terms of gestational age, birth weight, birth length, head circumference, APGAR scores and

Table 1. Physical signs and diagnosis of the neonates at admission to the NICU

Variables	Group 1 (n=222) n (%)	Group 2 (n=166) n (%)	Total (n=388) n (%)	*p
Physical signs				
Breathing difficulties	126 (56.8)	68 (40.9)	194 (50.0)	0.02
Feeding difficulties	32 (14.4)	16 (9.6)	48 (12.3)	0.42
Jaundice	34 (15.3)	27 (16.2)	61 (15.7)	0.83
Cardiac arrhythmia	8 (3.6)	2 (1.2)	10 (2.5)	0.28
Central cyanosis	44 (19.8)	55 (33.1)	99 (25.5)	0.04
Heart murmur	72 (32.4)	83 (50.0)	155 (39.9)	0.02
Neurological symptoms	18 (8.1)	11 (6.6)	29 (7.4)	0.72
Major congenital abnormalities	18 (8.1)	30 (18.0)	48 (12.3)	0.02
Diagnosis				
Pulmonary pathologies (RDS∞, TTNμ, MASα)	28 (12.6)	24 (14.4)	52 (13.4)	0.58
Sepsis (early and late onset)	68 (30.6)	38 (22.8)	106 (27.3)	0.24
Indirect hyperbilirubinemia	38 (17.1)	22 (13.2)	60 (15.4)	0.48
Lower tract infection	70 (31.5)	13 (7.0)	83 (21.3)	0.001
Macrosomia or infant of diabetic mother	14 (6.3)	5 (3.0)	19 (4.8)	0.35

αMAS-meconium aspiration syndrome, ∞RDS-respiratory distress syndrome, μTTN-transient tachypnea of the newborn

Table 2. Echocardiographic diagnosis in neonates at the neonatal intensive care unit

Cyanotic congenital heart diseases	n (%)
D-Transposition of great arteries	6 (20)
Single ventricle-single atrium	4 (13.3)
Tetralogy of Fallot	12 (40.0)
Double outlet right ventricle	2 (6.7)
Tricuspid atresia	2 (6.7)
Hypoplastic left heart syndrome	2 (6.7)
Total anomalous pulmonary venous drainage + tetralogy of Fallot	2 (6.6)
Acyanotic congenital heart diseases*	
Isolated Ventricular septal defect	25 (15.0)
VSD + ASD	12 (7.2)
VSD + PDA	8 (4.8)
Patent ductus arteriosus (PDA)	30 (18.0)
Isolated ASD (ASD)	15 (9.0)
Atrial septal defect+PDA	4(2.4)
Coarctation of the aorta	14 (8.4)
Coarctation of the aorta +ASD	6(3.6)
Hypertrophic cardiomyopathy	3 (1.8)
Aortic stenosis	3 (1.8)
Bicuspid aortic valve	3 (1.8)
Pulmonary stenosis (mild and moderate)	13 (7.8)

*Acyanotic/cyanotic CHD cases contains ≥1 cardiac disorders
∞Patent ductus arteriosus ≥37 weeks of gestation

maternal and paternal ages. NICU stay was statistically lower in neonates with CHD. First-degree parental consanguinity was statistically higher in neonates with CHD. The neonates with severe CHD can be presented with central cyanosis, heart murmur, respiratory distress, shock or collapse in the early postnatal life (3). Heart murmur, central cyanosis and major congenital abnormalities were found statistically higher in the neonates with cardiac disorder. The frequency of breathing difficulties as physical finding and lower respiratory tract infections were statistically higher in neonates with normal echocardiographic finding. Sepsis and indirect hyperbilirubinemia were the most common diagnosis in the neonates with CHD, but they were not statistically significant (Table 1). Archer et al. (4) reported that CHD is probably more frequent in VLBW infants treated in NICUs than in the general live-born population. With this study we observed that birth weight and gestational age are not statistical associations for the CHD. According to Aydoğdu et al. (5) and Shima et al. (2) the most common cyanotic congenital heart disease was tetralogy of Fallot and the most common acyanotic heart disease was VSD in the NICU. In our study results were same with those published reports (Table 2). The mortality rate was 19.1% (26/136) in the neonates with CHD. Mortality rate was significantly lower in the neonates with cardiac disorder at 38-42 weeks of gestation and >4000 g birth weight. In the non-survivor neonates with CHD; gestational age, birth weight, birth length, head circumference, APGAR score min 1 and 5 were statistically lower than survivors. Archer et al. (4) also reported that VLBW infants with serious congenital heart disease have a higher mortality rate. In our study, the non-survived neonates with CHD had lower birth weight, were more premature and were associated with lower APGAR scores than survivors with CHD. Finally, congenital heart defect was 8 times more frequent in neonates in the NICU than in live born neonates in population.

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