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Abnormalities of cardiac conduction in diabetics

The Framingham study showed that death from heart failure in diabetes exceeded that predicted by atherogenic risk factors. Subsequently several workers using echocardiographic or systolic time interval techniques found evidence of subtle defects in myocardial performance in diabetics.¹ Excess fibrosis and capillary microaneurysms have been noted in the human diabetic heart, but no correlation has been found between histopathological findings and specific cardiac abnormalities.² In a study of 100 patients with idiopathic chronic heart block, however, the prevalence of overt diabetes mellitus was 9%, which was well in excess of the prevalence in a group of hospital inpatients of comparable age.³ We attempted to ascertain whether the His-Purkinje system is vulnerable to such a microangiopathic process by comparing the frequency prevalences of conduction disturbances, using standard 12 lead electrocardiography, in diabetics and patients with hypertension.

Patients, methods, and results

We selected consecutively from the diabetic clinic a heterogeneous group of 200 patients with established type I or Type II diabetes but excluded patients with a history of infarction or angina pectoris and those taking drugs known to interfere with cardiac conduction. Standard 12 lead electrocardiography was carried out on patients in the supine position after a rest period of five minutes. Electrocardiography was similarly carried out on 200 patients with hypertension matched for age. All hypertensive patients had severe disease requiring at least triple treatment in the form of a β blocker, vasodilator, and thiazide diuretic. None had clinical diabetes. Electrocardiograms were scrutinised, and the heart rate and prevalences of atrioventricular block and right and left bundle branch block were documented according to the criteria of the Minnesota code (table). Results were analysed with the χ^2 test.

The diabetics had a significantly higher prevalence of both first degree atrioventricular and right bundle branch block compared with the patients

with hypertension. Left bundle branch block was more common in the hypertensive patients, but the difference was not significant. Stratifying the two populations into three groups on the basis of age showed a trend of increasing prevalence of all three conduction disturbances with age. In all three age groups, however, first degree atrioventricular and right bundle branch block were at least twice as common in the diabetics as in the patients with hypertension.

Comment

Although the association between left bundle branch block and hypertension is well recognised and thought to reflect to some degree the severity of left ventricular hypertrophy and arteriosclerotic heart disease, the aetiology of most conduction disturbances remains obscure. Chronic acquired complete heart block is commonly preceded by various conduction abnormalities and is often the end stage of a chronic and progressive disease destroying the conduction system.⁴ Atherosclerosis of the major coronary arteries is not responsible. Hasslacher and Wahl found a prevalence of diabetes mellitus of 41% among 473 patients permanently paced for bradyarrhythmias.⁵

The compact anatomical nature of the right bundle branch and atrioventricular node when compared with the more diffuse left bundle branch may increase their vulnerability to the patchy fibrotic process seen in the diabetic myocardium. We believe that the higher prevalence of both first degree atrioventricular and right bundle branch block in diabetics is related to microangiopathic rather than arteriosclerotic disease. Such findings give further support to the role of diabetes mellitus in the pathogenesis of complete heart block in some cases.

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Characteristics of patients with hypertension and diabetes and prevalence of electrocardiographic conduction abnormalities

	Diabetics	Patients with hypertension	p Value
No of patients	200	200	
Male:female	108:92	104:96	
Mean (range) age (years)	47 (19-65)	48 (19-65)	NS
No in age range:			
19-35	64	67	
36-50	64	65	
51-65	72	68	
Mean (range) heart rate (beats/min)	80.1 (47-112)	83.7 (52-116)	NS
No with first degree atrioventricular block*	14	4	< 0.02 ($\chi^2 = 5.82$)
No in age range:			
19-35	2	1	NS
36-50	5	1	NS
51-65	7	2	NS
No with right bundle branch block†	13	4	< 0.05 ($\chi^2 = 4.98$)
No in age range:			
19-35	4	1	NS
36-50	4	1	NS
51-65	5	2	NS
No with left bundle branch block‡	4	10	NS
No in age range:			
19-35		2	NS
36-50		4	NS
51-65	4	4	NS

*PR interval ≥ 0.22 s.

†QRS ≥ 0.12 s with R prime $> R_1$ in lead VI.

‡QRS ≥ 0.12 s with R peak duration ≥ 0.06 s in any of leads I, II, L, V5, or V6.

Nasal obstruction in healthy neonates

Most newborn infants have to breathe through their noses. Any obstruction in the nose or nasopharynx may cause difficulty in breathing and feeding. We present two cases of nasal obstruction causing respiratory problems in neonates who had normal airways severely congested by nasal mucosa.

Case reports

Case 1—An 18 hour old girl was referred with cyanotic episodes and obstructed nasal breathing. A 6 French gauge catheter was passed with some difficulty into the oropharynx via both nasal cavities. Findings on radiology, microlaryngoscopy, and bronchoscopy were normal. Examination of the nose and postnasal space showed no abnormality apart from severe mucosal congestion, and the nasal airways were easily dilated from 6 to 12 French gauge. Because of persistent difficulty in nasal breathing after this procedure a 3 mm nasopharyngeal airway was inserted. If this was removed obstructed nasal breathing resumed. Nasal stents were therefore inserted under general anaesthesia but were blocked with inspissated nasal secretions within two days despite regular passage of suction catheters and were removed. A 3.5 mm nasopharyngeal airway 7 cm long was placed in the right nostril. The child managed with this and was sent home with the airway in place. The airway was removed six weeks later, by which time she could breathe through her mouth. Three months later she was healthy, breathing easily through nose and mouth, and feeding well.

Case 2—A day old girl was referred, having developed cyanotic episodes not associated with feeding at 12 hours. Catheters were easily passed into the oropharynx through both nostrils. She was prescribed 0.5% ephedrine nose drops for evident nasal congestion, but her condition deteriorated so the drops were withdrawn. Radiological findings were normal, and examination under anaesthesia showed normal airways except for mucosal congestion in the nose. The nasal passages were dilated up to 14 French gauge. Post-operatively regular nasal suction and decongestants were used. Her condition deteriorated again with 0.5% ephedrine drops, which were again withdrawn. Three days postoperatively she still showed intercostal recession but could breathe through her mouth and was feeding well enough to return to the referring hospital. Three months later she was healthy with no airway or feeding problems.

Comment

Nasal obstruction in infants is usually detected soon after birth either because of obvious respiratory distress or by routine insertion of nasal suction catheters. Septal deformities, choanal stenosis and atresia, and nasopharyngeal masses are recognised causes of obstructed nasal breathing, but we believe that the problem of mucosal congestion alone has not previously been recorded. Once radiological and other findings have excluded any disorder of the upper and lower airways an airway should be provided until the child can breathe orally, which is usually within two to three weeks.¹ In one patient we were able to ease breathing by dilating the nasal airways, although we considered inserting a nasopharyngeal airway to relieve laboured breathing. Interestingly, this child's condition deteriorated when 0.5% ephedrine nose drops were given and improved when they were withdrawn. Despite our considerable experience in telling parents how to manage nasal stents, blockage occurred and the stents had to be removed.

We conclude that nasal congestion causing serious airway obstruction may be overcome by simple nasal dilatation after thorough examination of the airways under anaesthesia. If an airway remains blocked after operation a nasopharyngeal airway can be inserted. This allows more comfortable and "natural" breathing than with an oral airway. It is also preferable to nasal stents because a larger bore tube can be placed unilaterally rather than bilaterally owing to the compliance of the nasal septum. As this airway is likely to be in place for some weeks, however, the parents must be given portable suction apparatus and instructions on how and when to clear the airway. Antibiotic cover is required.

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Serologically proved intrauterine infection with parvovirus

It is well known that systemic viral illness in the mother may have an adverse effect on the outcome of pregnancy. Viruses of the TORCH syndrome (*Toxoplasma*, rubella, cytomegalovirus, herpes virus)¹ have received most attention, the clinical features including intrauterine growth retardation, congenital abnormality, mental handicap, and intrauterine death. Hence viral disease in pregnancy is a major problem.² We describe a case in which a serologically proved parvovirus infection may have contributed to intrauterine death at term in an otherwise uneventful pregnancy.

Case report

A 35 year old para 0+1 booked at 13 weeks' gestation, confirmed by ultrasound. Her husband had ankylosing spondylitis. Antenatal findings were normal, and maternal serum α fetoprotein concentration was 1.1 multiples of the median at 16 weeks. Detailed ultrasound examination showed no congenital anomaly and amniocentesis was performed at 16 weeks with

no complications. Amniotic fluid α fetoprotein and acetylcholinesterase isoenzyme values were normal. Chromosomes showed a normal count with pericentric inversion of one of the number 9 (karyotype 46XX 9qh(inv)), which is not important. Pregnancy proceeded normally to 39 weeks, when the patient reported a flu like illness. This may or may not have been a parvovirus infection, but it did not cause any appreciable maternal problems. Blood pressure remained normal and there was no reduction of fetal activity.

Spontaneous labour began the day after the expected date of delivery. On admission, however, the fetal heart beat was not recordable and real time ultrasound confirmed intrauterine death. Vaginal examination showed the cervix to be dilated 6 cm and amniotomy yielded clear liquor. An epidural was sited for analgesia. Two hours later a stillborn girl was delivered vaginally. The infant was macerated and appeared to have severe ascites but otherwise looked normal. Samples of fetal and maternal blood were taken for viral studies, grouping, and antibody screening.

Necropsy showed a normal stillborn girl weighing 3840 g. Meconium ileus and peritonitis accounted for the ascites, with evidence of obstruction at jejunal level. There was no histological evidence of cystic fibrosis. Subpleural haemorrhages were noted and the cause of death given as intrauterine anoxia.

TORCH screening showed no evidence of recent infection. Nevertheless, both maternal and fetal blood contained parvovirus specific IgM, indicating recent maternal infection and also congenital infection of the fetus in utero. No viral deoxyribonucleic acid was detected in either specimen.

Comment

Parvovirus causes a mild feverish illness with a rash and arthralgia, particularly in adults.³ At the time of this stillbirth the virus was epidemic in south east England. Despite attempts to identify viral DNA in specimens from spontaneous abortions we had been unable to show any evidence of transplacental infection until this case, which was the sixth stillbirth that we had investigated for parvovirus infection. Finding parvovirus specific IgM in fetal blood is definite evidence of in utero infection. Other viral infections transmitted transplacentally may be associated with poor outcome of pregnancy. In utero infection with parvovirus may therefore be expected to be associated with increased fetal morbidity and mortality.

The meconium ileus and peritonitis may not have been related to the infection in this case. There was no evidence of cystic fibrosis, either histologically or in the family history, but this was the most likely cause of the findings.

This is the first recorded example of in utero infection with parvovirus.⁴ There may therefore be a case for testing for this infection in addition to the routine TORCH screen performed on symmetrically growth retarded fetuses and unexplained stillbirths.

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