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 diabetes. Excess fibrils and capillary microaneurysms have been noted in the human diabetic heart, but no correlation has been found between histopathological findings and specific cardiac abnormalities. In a study of 100 patients with idio- pathic 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. We attempted to ascertain whether the His-Purkinje system is vulnerable to such a microangiopathie 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 heterogenous 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 χ² 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. 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.

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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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) 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. We describe a case of 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 ultrasonography. Her blood picture was unremarkable. 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 acetylcysteinesterase isoenzyme values were normal. Chromosomes showed a normal count with pericentric inversion of one of the number 9 (karyotype 46XX q9q18), 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. 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 anaesthesia. Two hours later a stillborn girl was delivered vaginally. The infant was macerated and appeared to have severe asities 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 asities, with evidence of obstruction at jejunoileal 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. 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 first published for this virus 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 the first 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. There may therefore be a case for testing for this infection in addition to the routine TORCH screen performed on symptomatically growth retarded fetuses and unexplained stillbirths.