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ACUTE HYPERTENSIVE CARDIAC FAILURE IN PREGNANCY

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Acute hypertensive cardiac failure in pregnancy is a serious complication usually associated with severe pre-eclamptic toxaemia. It is difficult to manage and is associated with an appreciable mortality.[1] Fortunately it is not common, though I feel its relative importance will increase in future as the mortality and morbidity of valvular heart disease in pregnancy become controlled. Moreover, with the widespread recommendation and use of modern contraceptive measures, family spacing can be more successfully carried out. It is thus conceivable that more women will be pregnant by design rather than by accident at a later age than is the case today, with a consequently greater likelihood of cardiovascular and other degenerative disorders being associated with their pregnancies.

INCIDENCE

This condition is apparently not encountered in British obstetrics today. Thus Gilchrist[2,3] has never encountered it in Scotland and does not mention it. It is mentioned only in passing in some modern English obstetrical textbooks, being usually described under morbid anatomical changes of the heart occurring in fatal cases of eclampsia.[4-9]

In some textbooks no reference at all is made to it.[10,11] One could possibly ascribe this fortunate lack of occurrence to excellent preventive antenatal services in a welfare state, but then even older British textbooks give the same scant attention to this dangerous complication.[12,13]

Is it possible that this condition had been overlooked in the past? My own opinion is that this could quite easily happen. However, it is probably more reasonable to suggest that these cases represent a type of clinico-pathological entity not seen in Britain because of local variants in this disease. Similar differences have been noted in other disease processes in South Africa, e.g. in pelvic infection,[14] in tuberculosis, etc. These implications will be discussed later.

In the USA, in contrast to England, this is a well-known and feared complication, being associated with an appreciable mortality. Dieckmann[1] diagnosed this syndrome in 19 cases, 2 patients dying. He also mentions 15 cases of Teel, Reid and Hertig.

This syndrome is discussed in some detail by Eastman and Hellman[15] who also mention the great respect Stroganoff had for its occurrence. McCartney[16] mentions the treatment of this condition in his discussion of the complications of eclampsia.

In our own maternity units there were 429 cardiac cases during the 5-year period 1958 - 1962; of these, 16 were classified as suffering from hypertensive cardiac disease. This diagnosis connotes cardiac failure associated with gross hypertension.

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More important and impressive than the mere incidence of this syndrome, and illustrating its seriousness, is the fact that in 5 out of 33 maternal deaths over the same 5-year period, the clinical cause of death was ascribed to this type of cardiac disease. In addition it is thought that in 7 deaths associated with eclampsia and hypertensive accidental haemorrhage this syndrome played a contributory role.

In many cases of seriously ill obstetric patients with marked hypertension, critical analysis shows that cardiac failure is clinically missed in its early stages as the accoucheur is likely to concentrate chiefly on the 'obstetric' aspects. Consequently the incidence of this condition must be even higher than indicated by the figures quoted.

The following case report clearly illustrates the complex nature of the syndrome and the difficulties in management.

CASE REPORT

J.F., aged 32 years. Gravida 6, para 2, with 3 abortions. Last menstrual period in September; expected date of confinement in June. She was thus 34 weeks pregnant when admitted. There were no special features in her past history.

With the present pregnancy she was first examined when 22 weeks pregnant. She was noted to be obese (weight 160 lb.): her urine was clear; there was no evidence of peripheral oedema and her blood pressure was 120/70 mm.Hg. She remained well and attended the antenatal clinic regularly except for the last 2 weeks before admission.

When she was next seen at the clinic she was 34 weeks pregnant with a blood pressure of 210/150 mm.Hg; her urine was solid with albumin; there was gross oedema of the feet, legs, hands, face and abdominal wall; and there was an increase of 30 lb. in her body-weight. The pulse rate was 108 beats/min. and regular in rhythm. Clinically there was no cardiomegaly and the lung fields were normal.

Course and Management

She was immediately admitted and given 7 ml. of Avertin dissolved in water. After the sedative effect had taken place a vaginal examination with low amniotomy was performed.

Over the following 12 hours:

1. Her blood pressure increased rapidly and steadily once the effects of the drugs had worn off. As a result Avertin had to be repeated 3 times and intravenous Nepresol was also administered.
2. She became very restless at times and difficult to control. Sedatives such as morphine, $\frac{1}{4}$ gr., and 10 ml. of paraldehyde had to be given in addition to the Avertin.
3. Her pharynx had to be kept clear of copious secretions by suction. The problem was made more acute by restlessness in spite of heavy sedation and the difficulty of maintaining an adequate airway. The latter was aggravated by a short, thick neck—a physical attribute of which anaesthetists are particularly wary.
4. The respirations increased in rate up to 38/min. and the pharyngeal secretions became frothy and blood-stained. At this stage diffuse crepitations were heard on auscultation of the lungs.
5. The pulse rose to 136 - 158 beats/min. but remained regular.

It was difficult to decide in the early stages whether the tachycardia was due to the disease process, which had become accelerated with resultant cardiac decompensation, or to the

drugs used; for it is known that Avertin can cause a rise in pulse rate of 20 beats/min.[17]

Electrocardiographs showed a sinus tachycardia with mild left ventricular hypertrophy. There was no evidence of ischaemia.

All these changes occurred insidiously, and it only ultimately became apparent that the patient had passed into hypertensive cardiac failure with pulmonary oedema.

At this stage she presented a grotesque appearance and a formidable clinical problem. Her eyelids were practically closed by oedematous puffiness, she was restless with copious pulmonary secretions, the breathing was stertorous and obstructed by a flabby tongue, a short thick neck made the retention of an airway difficult, the pulse was racing and the blood pressure elevated and, to complete the picture, her uterine contractions were irregular, short and weak.

Conventional medical treatment resulted in great improvement in her cardiac condition. A caesarean section was contemplated at this stage, but an oxytocic intravenous infusion containing 10 units of Syntocinon, which had been started 12 hours after admission, produced active labour. A speedy, normal vaginal delivery of a live 5 lb. 5 oz. baby took place 24 hours after admission. Her subsequent course was uncomplicated. She was discharged with her baby on the 12th postpartum day with a blood pressure of 120/80 mm.Hg, albumin-free urine, and no evidence of oedema.

CLINICAL FEATURES OF THE SYNDROME

The following features were noted in this series:

Age

It is usually the 30 - 40 year age-group which is affected. In our cases the ages varied between 31 - 41 years. It is understandable that in the older age-groups the incidence of essential hypertension would be higher, and this is an important factor in the pathogenesis of this syndrome. Kaltreider[18] found that in the 'elderly multip', i.e. over 35 years of age, hypertensive heart disease, often leading to failure, is almost twice as common as in younger multigravidae.

Parity

A high parity is ultimately associated with the older age-groups. In our cases the parity varied from 6 to 14.

Obesity

Recently attention has been focused on obesity in relation to cardiac disease. It is stated that fat people have a poorer life expectancy than thin ones, though the reason for this is not fully known. Obesity causes a higher incidence of coronary artery disease by increasing the incidence of diabetes and hypertension, both of which themselves damage blood-vessels.[19] Alexander *et al.*[20] analysed the cardio-respiratory effects of obesity. They found that hypertension, cardiac enlargement, congestive cardiac failure, oedema and dyspnoea can all be associated with obesity *per se*. Kerr[21] confirmed the clinical impression that obesity is a function of parity and found that both hypertension and pre-eclampsia are directly related to obesity. Fishberg considers that obesity displaces the heart to a more transverse position, which may result in it functioning at a mechanical disadvantage.

One of the characteristics of our patients was obesity, usually obvious in spite of the gross oedematous state. One must therefore agree with Mendelson[22] that overweight is a contributory cause of the severe heart failure in these cases.

In addition, it must be mentioned that dietary intake of protein and related vitamins is deficient in most of our patients, but the exact part this plays in the syndrome is difficult to assess.

Onset

The manifestations of this syndrome can occur insidiously and be missed by the accoucheur in the early phases until the full-blown picture has developed. The onset can also be acute, the signs and symptoms becoming manifest within a short period of time. This type is usually recognized soon after its occurrence. But, however it presents, this syndrome can be as frightening and formidable as the acute pulmonary oedema associated with rheumatic valvular disease which occurs so dramatically after the third stage of labour.

Previous Hypertension

It is impossible to determine this in most of our patients as so many attend for their first examination when their pregnancy is well advanced or are admitted as emergencies. Needless to say, their pre-pregnancy status is never known. However, in at least 4 of the maternal deaths in this series, previous hypertension with superimposed pre-eclamptic toxaemia was diagnosed. It is obvious that the addition of pre-eclamptic toxaemia—especially if acute in onset and severe in nature—to a heart already labouring under chronic hypertension, might easily precipitate acute decompensation in that organ.[23,24]

The clinical signs of the heart failure are the same as for cardiac failure in general. Initially, there is left ventricular failure with dyspnoea, orthopnoea, tachycardia, basal crepitations and other features of pulmonary oedema. Later the right ventricle also fails, so that increased systemic venous pressure becomes manifest.[25]

Mechanism of the Cardiac Decompensation

The exact mechanism is not clear. Several factors are involved.

1. *'Myocarditis'*. Abnormal electrocardiographic patterns in toxaemia and eclampsia have been described by many authors. Kellar[24] has found that ECG changes indicate that myocardial damage is frequently associated with heart failure in eclampsia.

Eastman and Hellman[15] quote P. D. White, who states in his well-known book on heart disease that 'associated with acute hypertension in the toxaemia of pregnancy, there may be serious myocardial dilatation with acute heart failure and pulmonary oedema'. They also quote the work of Wallace *et al.*, who examined ECGs of 12 toxaemic patients and found significant changes in 6, with actual left ventricular failure in 2. These workers felt that the alterations simulated those occasionally seen in acute nephritis and attributed them to focal myocardial necrosis, infiltration or oedema, rather than to myocardial infarction secondary to occlusion of the coronary arteries. Szekely and Snaith also found ECG changes in toxaemic patients.

Hamilton, on the other hand, is still seeking a patient with myocarditis. He states that 'our present opinion is that uncomplicated toxaemia causes neither heart failure nor significant acute myocarditis'. However, Eastman and Hellman,[15] who quote Hamilton, remain impressed with the abundant evidence documenting the contrary viewpoint.

Mendelson[26] postulates various causes. One is that in chronic hypertensive vascular disease, cardiac hypertrophy results in a disparity between heart muscle mass and blood supply and creates a state of relative myocardial hypoxia.

2. *Superelevation of blood pressure.* There is no close correlation either between the height of the blood pressure or an acute rise in blood pressure and the liability to cardiac failure, though the former has often been invoked in cases of cardiac failure in acute nephritis.[27]

3. *Coronary atherosclerosis.* Fishberg[27] notes that in this condition there may be neither clinical nor ECG changes. Mendelson[22] mentions this lesion as an added factor in the production of myocardial hypoxia in cases of chronic hypertensive vascular disease. In our type of cases coronary insufficiency must always be excluded.

4. *Left ventricular failure and hypervolaemia.* The latter is the most important factor in the pathogenesis of this syndrome. Mendelson[22] states that in severe toxaemia the decrease in renal blood flow can lead to impaired sodium excretion, water retention and hypervolaemic pulmonary oedema. A situation akin to this is found in acute nephritis with hypervolaemic cardiac failure.[27]

McCartney[16] ascribes the pulmonary oedema to left ventricular failure, to cor pulmonale resulting from widespread constriction of the pulmonary arterioles, to an increased permeability of the alveolar epithelium, or to a combination of these factors.

Llewellyn-Jones[25] states that the onset of pulmonary oedema may be due to a failing heart or to increased transudation of fluid into the interstitial spaces of the lung and into the alveoli, consequent upon pulmonary hypertension. Kellar[24] indicates that the pulmonary oedema may also be due to a bronchiolitis.

In our patients, in addition to the above factors, we strongly suspect that underlying nutritional myocardial damage must have played some part in the pathogenesis of this syndrome, though this has not been proved.

MANAGEMENT

A. Prophylactic Therapy

It can be confidently stated that adequate antenatal care would virtually eliminate this syndrome. In fact, the low incidence of this syndrome in England can probably be ascribed to this very assertion. A perfunctory perusal of our annual reports will clearly indicate just how far we are from achieving this essential state of affairs. The antenatal doctrine is being slowly spread among the underprivileged sections of our community.

1. Early, adequate control of essential hypertension is obviously helpful.
2. The treatment of obesity must be energetically approached.
3. The epic work of Hamlin, Hughes and Dawson has demonstrated that toxaemia of pregnancy can be reduced to a minimum by early, adequate treatment.

B. Medical Treatment

This follows the conventional method of treating pulmonary oedema and heart failure.

1. *Digitalis.* As a result of our harrowing experiences with the early cases in this series, we are now in complete agreement with the American authors who advise prophylactic digitalization of all severe pre-eclamptic cases as well as of eclamptics.[15,16,29] If this has been omitted and pulmonary oedema ensues, rapid digitalization by the intravenous route is advised initially, e.g. 0·5 - 1·0 mg. of Digoxin intravenously, *stat,* followed 4 - 6 hours later by 0·5 mg. intramuscularly.

2. *Aminophyllin.* This is given intravenously in doses of 20 ml. and may be repeated 4 - 6-hourly.

3. *Mersalyl.* Though the use of this heavy-metal diuretic in the presence of renal damage and scanty output is not generally recommended, mersalyl has been of immeasurable value in at least 1 patient. It is such a potent diuretic that we have used it as a routine in these waterlogged patients.

4. *Oxygen.* This is usually administered to patients, especially if they have had convulsions.

5. *Venesection.* This can be life-saving. Both phlebotomy and 'medical' venesection are recommended.

6. *General care.* In our units Avertin *per rectum* is used. Once patients have been rendered unconscious by the Avertin, they represent a very difficult nursing problem. They have to be nursed flat, being turned from side to side periodically. In spite of this, infective chest conditions are common. Fortunately these respond rapidly to antibiotics. Penicillin and streptomycin intramuscularly are used as a routine medication, taking full cognizance of the urinary output. Chest physiotherapy is also instituted.

Maintenance of a clear airway is made difficult by the build of the patient with a short, thick neck and by copious secretions which have to be cleared by suction. Atropine is often given to these patients, but this renders the secretions more viscid and thus more difficult to aspirate. It also produces a tachycardia which adds further to the increased heart rate caused by the Avertin.[17] Thus it is easy to understand how the drug-induced tachycardia and copious secretions can mask the initial stages of heart failure. Menon[30] and Jones[28] (1961) have largely overcome these problems by using 'The lytic cocktail' of phenothiazine derivatives.

Though we have not used antihypertensive agents, this syndrome usually occurs as a fulminating toxaemic process and these drugs have not been uniformly successful in pre-eclampsia and eclampsia.

C. Obstetrical Management

The pregnancy must be terminated as soon as possible, but only after the cardiac failure has responded to treatment. This policy is in complete contrast to the conservative approach adopted in valvular heart disease.

The termination should be by full surgical induction with the use of a pitocin drip if necessary, for if good labour does not ensue within a reasonable period of time (about 12 hours) a caesarean section should be performed, as only by emptying the uterus will the toxaemic process abate. A caesarean section should also be done if the cervix is found to be 'unripe'.

SUMMARY

1. Attention is focused on acute hypertensive cardiac decompensation in pregnancy.

2. A high local incidence is compared with a virtual non-existence in the United Kingdom and a waning incidence in the USA. There is also a high associated mortality.

3. A typical case is detailed, illustrating the nursing difficulties and the problem in diagnosing and treating the condition.

4. The association with the older age-groups, high parity and obesity are noted.

5. The various factors in the pathogenesis of this syndrome are fully discussed.

6. The careful assessment of the use of various drugs is outlined. It is strongly recommended that prophylactic digitalization of severe pre-eclamptic as well as eclamptic patients be carried out as a routine procedure.

7. In contrast to cardiac failure associated with valvular heart disease, early rapid termination of the pregnancy is advocated in these patients.

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