ANESTHETIC MANAGEMENT OF PREGNANT PATIENTS WITH VALVULAR HEART DISEASE IN LABOR

Essay

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ACEI	Angiotensin Converting Enzyme Inhibitor
AF	Atrial fibrillation
AHA	American hear association
aPTT	activated partial thrompoplastin time
AR	Aortic regurge
AVD	Aortic valve disease
BPM	Beat per minute.
CO	Cardiac output
CVP	Central venous pressure
CXR	Chest X-ray.
ECG	Electrocardiogram.
HOCM	Hypertrophic obstructive cardiomyopathy
IUGR	Intrauterine growth retardation
LA	Left atrium
LMWH	Low molecular weight heparin.
LV	Left ventricle
MR	Mitral regurge
MS	Mitral stenosis
MVA	Mitral valve area
MVP	Mitral valve prolapse
NYHA	New-York Heart Association
PCWP	Pulmonary capillary wedge pressure
PPCM	Peripartum cardiomyopathy
PS	Pulmonary stenosis
SVR	Systemic Vascular resistance
TIA	Transient ischemic attack
TVD	Tricusped valve disease
UH	Unfractionated heparin
USCS	Upper segment cesarian section
VHD	Valvular heart disease

INTRODUCTION

Valvular heart disease (VHD) is often first recognized during pregnancy, when increased demands on the heart trigger symptoms. They pose additional risk to both mother and fetus. As more women with congenital or acquired cardiac diseases survive into the child bearing age, physicians must be aware of the special difficulties that these women face during pregnancy. Cardiac diseases complicate ١-٤%of pregnancies in women without preexisting cardiac abnormalities; the incidence of the VHD is fairly high (١% to ٢%) among female patients of childbearing age ***(Sui et al., ٢٠٠١).*** Whenever possible, women with preexisting cardiac lesions should be considered in advance about the risk of pregnancy. A working knowledge of the normal physiology of pregnancy is often helpful in the management of patients with heart disease**.** The profound hemodynamic changes associated with pregnancy have marked effects on patients with VHD and require special attention and care**.** Although there is an ever-decreasing prevalence of rheumatic heart diseases in developed nations, it's still represents **٩٠%** of cases in the developing countries and come on the top of the list of acquired heart diseases, followed by ischemic heart diseases and disorders of rhythm ***(Cunningham et al., ٢٠٠١).***

Management of parturient with VHD should be multidisciplinary, obstetric; cardiology and anesthetic opinions should all be sought. Attention should be drawn to accurate diagnosis as to which valves are involved, assessment of the severity of lesion, degree of impairment resulting from the lesion and evaluation of concomitant therapy. It's important that care be carried on into management of pregnant women with preexisting VHD wishing to proceed to term, cardiac status must be optimized preoperatively and planned elective delivery is preferable. Vaginal delivery is preferable, and with careful incremental regional anesthesia is safe in most women with Cardiac disease, in addition, as well as optimizing management during pregnancy and labor, Intensive monitoring should be continued for at least ٧٢ hours after delivery, preferably in a high care intensive care environment, as the majority of reported deaths in cases of VHD in pregnancy occur in the post – partum period ***(Bonow et al., ٢٠٠٤).***

We aim finally of this study to highlight the factors should be recognized by the clinician and address the recent advances in the anesthetic management of pregnant patients with VHD in labor.

PHYSIOLOGICAL CHANGES OF PREGNANCY

Physiological and anatomical alterations develop in many organ systems during the course of pregnancy and delivery. Early changes are due, in part, to the metabolic demands brought on by the fetus, placenta and uterus and, in part, to the increasing levels of pregnancy hormones, particularly those of progesterone and estrogen. Later changes, starting in mid-pregnancy, are anatomical in nature and are caused by mechanical pressure from the expanding uterus. These alterations create unique requirements for the anesthetic management of the pregnant woman. All these changes in addition to V.H.D. impose additional risks added to pregnancy and labor .To understand this properly we will discus each system on brief ***(Elkayam and Bitar, ٢٠٠٥).***

Cardiovascular changes during pregnancy:

The cardiovascular system (CVS) undergoes remarkable changes in pregnancy, these changes enhance the blood supply to the uterus, maintain fetal growth and support the changes in the breast in preparation for lactation, and most of the CVS changes are well underway by the ١st ٨ weeks of pregnancy ***(Capless and Clapp, ١٩٨٩).***

The stimulus behind CVS adaptation is probably endocrinal, there is a marked decrease in systemic vascular

tone perhaps induced by prostaglandins, this triggers a compensatory increase in heart rate, stroke volume and activation of volume restoring mechanisms, estrogen infusion are shown to mimic these changes ***(Duvekott et al., ١٩٩٣).***

Blood Volume: Increases progressively from ٦-٨ weeks of gestation) and reaches a maximum at approximately ٣٢-٣٤ weeks with little change thereafter. The increase in plasma volume (٤٠-٥٠%) is relatively greater than that of red cell mass (٢٠-٣٠%) resulting in hemodilution and a decrease in haemoglobin concentration. Intake of supplemental iron and folic acid is necessary to restore hemoglobin levels to normal (١٢ g/dl). The increased blood volume serves two purposes, first, it facilitates maternal and fetal exchanges of respiratory gases, nutrients and metabolites, second, it reduces the impact of maternal blood loss at delivery. Typical losses of ٣٠٠-٥٠٠ ml for vaginal births and ٧٥٠-١٠٠٠ ml for Caesarean sections are thus compensated with the so-called "autotransfusion" of blood from the contracting uterus ***(Mable et al., ١٩٩٤).***

Cardiac Output (CO): Increases to a similar degree as the blood volume. During the first trimester cardiac output is ٣٠-٤٠% higher than in the non-pregnant state. Steady rises are shown on Doppler echocardiography, from an average of ٦. ٧ liters/minute at ٨-١١ weeks to about ٨.

٧ liters/minute flow at ٣٦-٣٩ weeks; they are due primarily to an increase in stroke volume (٣٥%) and, to a lesser extent, to a more rapid heart rate (١٥%). There is a steady reduction in systemic vascular resistance (SVR), which contributes towards the hyperdynamic circulation observed in pregnancy ***(Oppen et al., ١٩٩٦).***

During labor, further increases are seen with pain in response to increased catecholamine secretion; this increase can be blunted with the institution of labour analgesia. Also during labour, there is an increase in intravascular volume by ٣٠٠-٥٠٠ ml of blood from the contracting uterus to the venous system., following delivery this autotransfusion compensates for the blood losses and tends to further increase cardiac output by ٥٠% of pre-delivery values. At this point, stroke volume is increased while heart rate is slowed ***(Maroo and Raymond, ٢٠٠٤).***

Cardiac Size/Position/ECG: There are both size and position changes which can lead to changes in ECG appearance. The heart is enlarged by both chamber dilation and hypertrophy. Dilation across the tricuspid valve can initiate mild regurgitant flow causing a normal grade I or II systolic murmur. Upward displacement of the diaphragm by the enlarging uterus causes the heart to shift to the left and anteriorly, so that the apex beat is moved outward and upward. These changes lead to common ECG findings of left axis deviation, sagging ST segments and frequently

inversion or flattening of the T-wave in lead III ***(Clark, ١٩٩١).***

Blood Pressure: Systemic arterial pressure is never increased during normal gestation. In fact, by midpregnancy, a slight decrease in diastolic pressure can be recognized. Pulmonary arterial pressure also maintains a constant level, however, vascular tone is more dependent upon sympathetic control than in the non pregnant state, so that hypotension develops more readily and more markedly consequent to sympathetic blockade following spinal or extradural anaesthesia. Central venous and brachial venous pressures remain unchanged during pregnancy, but femoral venous pressure is progressively increased due to mechanical factors ***(Cunningham et al., ٢٠٠١).***

Aortocaval Compression: From mid-pregnancy, the enlarged uterus compresses both the inferior vena cava and the lower aorta when the patient lies supine., obstruction of the inferior vena cava reduces venous return to the heart leading to a fall in cardiac output by as much as ٢٤% towards term. In the unanaesthetised state, most women are capable of compensating for the resultant decrease in stroke volume by increasing systemic vascular resistance and heart rate. There are also alternative venous pathways, the Para vertebral and azygos systems. During anesthesia, however, these compensatory mechanisms are reduced or abolished so that significant hypotension may rapidly

develop. Obstruction of the lower aorta and its branches causes diminished blood flow to kidneys, uteroplacental unit and lower extremities. During the last trimester, maternal kidney function is markedly lower in the supine than in the lateral position furthermore, the fetus is compromised by insufficient transplacental gas exchange ***(Okaly, ١٩٩٠).***

Venous distension: Increases approximately to ١٥٠% during the course of gestation and the venous ends of capillaries become dilated, causing reduced blood flow. These vascular changes contribute to delayed absorption of subcutaneously or intramuscularly injected substances. Distension of the extradural veins heightens the risk of vascular damage during institution of a regional block. The increased venous volume within the rigid spinal canal reduces the volume or capacity of the extradural and intrathecal spaces for local anaesthetic solutions. This will therefore increase the spread of injected drugs ***(Jonathan et al., ٢٠٠٢).***

Heart rate: On physical examination of the normal parturient there's a slightly fast resting heart rate, about ١٠-١٥ beat per minute, the first heart sound may be louder than normal, with prominent splitting due to mitral valve closing earlier than tricuspid ***(Baughman, ٢٠٠٣).*** The second heart sound is usually physiologically split but may also widen and appear fixed during the later stages of pregnancy and lastly the third heart sound can be recorded in **٨٤%** of pregnant women ***(Hunter and Robson, ١٩٩٢).***

Murmurs: A large proportion of pregnant patients have peripheral oedema together with distension of the central veins and many have audible flow murmurs and a third heart sound indicative of volume overload .Two innocent continuous murmurs that can be heard frequently during gestation are the cervical venous hum and the mammary soufflé. The venous hum is usually heard maximally over the right supraclavicular fossa but can radiate to the contra lateral area and sometimes to the area below the clavicle. The mammary soufflé which is heard mostly over the breast late in gestation or in the lactating women postpartum is caused by increased flow in the mammary vessels and can be either systolic or continuous. The murmur decreased or vanished when pressure is applied on to the stethoscope, or when the patient moves into the upright position ***(Braunwald,*** ١٩٩٢***).*** An innocent grade I to II systolic ejection murmur develop in most of the women usually in early systole and are the result of hyperkinetic circulation of pregnancy, they are best heard along the left sternal edge and over the pulmonic area radiating to the suprasternal notch and some times to the right side of the neck ***(Hunter and Robson,*** ١٩٩٢***).*** The systolic murmur lasts from before mid-pregnancy until the first week postpartum. Doppler echo studies suggest that a proportion of these systolic murmurs may be due to functional tricuspid regurgitation due to dilatation of the valve annulus in pregnancy. This is not associated with any other clinical or pathological features ***(de Swiet,*** ١٩٩٥***).***

Large minorities of women will demonstrate Doppler evidence of physiological M. R in the absence of structural

valve disease. Appreciation of these echocogdiographic finding in normal individuals is an important foundation for the non-invasive evaluation of subjects with suspected valvular disease**.** A medium to high pitched diastolic murmur is heard occasionally in a number of normal pregnant women, it is best heard along the left sternal border and is considered to be the tricuspid flow murmur of no significance ***(Bonow et al.,*** ١٩٩٨***).*** The increased blood volume and enhanced cardiacoutput associated with normal pregnancy can accentuate the murmurs associated with stenotic heart valve lesions (e. g. Ms, As) on the other hand, murmurs of AR or MR may actually attenuate in the face of lowered systemic vascular resistance **.**Many normal women manifest subtle signs of cardiac failure during uncomplicated pregnancy and delivery. Dyspnoea and fatigue are common, together with a reduction in exercise capacity ***(Bonow et al .,*** ١٩٩٨***).***

These normal findings must be differentiated from these indicating heart disease which include:

a) A systolic murmur greater than grade III,

b) Any diastolic murmur,

c) Severe arrhythmias

d) Unequivocal cardiac enlargement on x.- ray.

Where underlying VHD present, it is hardly surprising that symptoms and signs of cardiac failure may occur during pregnancy or at the onset of labor ***(Prasad and Ventura*** ٢٠٠١***).***