



INTRAABDOMINAL PRESSURE AND PREGNANCY

Dr. Bharti Choudhary

MD Anaesthesia, Civil Hospital Nadaun, Hamirpur, Himachal Pradesh.

Dr. Nishchint Sharma*

MD Anaesthesia. Civil Hospital Nadaun, Hamirpur, Himachal Pradesh.
*Corresponding Author

ABSTRACT IAP was defined in 2006 by the World Society of Abdominal Compartment Syndrome consensus definition as the steady state pressure concealed within the abdominal cavity. There are few studies that measures influence, and management of intra-abdominal pressure (IAP) and Intra-abdominal hypertension (IAH) in pregnancy. Preeclampsia, supine hypotension syndrome are common cause of significant morbidity and mortality in both mother and new-born. During critical illness in pregnancy, little importance is given to the possible impact of intra-abdominal pressure (IAP) and intra-abdominal hypertension (IAH) on such conditions.

KEYWORDS : Intraabdominal pressure, Abdominal compartment syndrome, pregnancy, supine hypotension syndrome.

INTRODUCTION:

IAP was defined in 2006 by the World Society of Abdominal Compartment Syndrome consensus definition as the steady state pressure concealed within the abdominal cavity. In general, a normal IAP varies from sub-atmospheric values to 7 mmHg in normal-weight individuals, with higher baseline levels in morbidly obese patients of about 9 to 14 mmHg. Intra-abdominal hypertension (IAH) is defined as a sustained increase in IAP ≥ 12 mmHg and abdominal compartment syndrome (ACS) is defined as IAP >20 mmHg with new onset organ failure. Both IAH and ACS are associated with organ dysfunction, multisystem organ failure, high morbidity and mortality.^{1,2}

Till date, little is known about normal values of IAP during pregnancy, either in healthy or complicated pregnancies. In 1913, Paramore was the first to investigate IAP during pregnancy.³ If IAP is not measured, IAH will often be missed. Further, due to little understanding of IAH in pregnancy risk of delayed diagnosis of ACS resulting in morbidity and mortality is high. Preeclampsia and obstetric hemorrhage are two common cause of significant morbidity and mortality in both mother and newborn^{4,5}. Intensivists are often unfamiliar with maternal-fetal physiology in cases of critical illness.⁴

MATERNAL PHYSIOLOGY AND IAP:

The maternal physiologic changes that occur in pregnancy are multisystemic. On average, the uterus contributes 1 kg to the overall weight gain in pregnancy, while the amniotic fluid, fetus, and placenta comprise approximately 5 kg in additional weight⁶. After 20 weeks of gestation, the uterus size can cause a mechanical aorto-caval obstruction while fully supine and can result in the supine-hypotensive syndrome: significant loss of venous return for which the cardiovascular system cannot compensate.⁶ Only approximately 8% of women at term experience this life-threatening situation, significant compression of the inferior vena cava (IVC) while supine does occur in the majority of women.^{6,7} Whether elevated IAP can exacerbate aorto-caval compression and has a relationship with this syndrome is unknown.

Till date there is very few studies regarding physiologic and pathophysiologic IAP in pregnancy. Chun R et al in 2012 measured the IAP in 20 term parturients under spinal anesthesia. The IAP measurement was significantly higher in the fully supine position (0°) compared to when the operating table was leftward tilted to 10° with the reference point held constant by placing the bladder pressure transducer in a line adjacent to the patient on an intravenous pole. They thus hypothesized that the weight of the gravid uterus might have directly impacted on the bladder, thereby falsely elevating the IAP measurement when fully supine.⁸

Preeclampsia, part of a spectrum of hypertensive disorders of pregnancy, is defined as the development of arterial hypertension and proteinuria after 20 weeks gestation⁹ and is associated with significant maternal morbidity and death.^{5,9} Even in 1900s, investigators had suggested uncompensated elevated IAP as a possible etiologic factor

in the development of preeclampsia.^{3,10} Paramore, also noting this prevalence, hypothesized that nulliparous and muscular women were prone to spastic abdominal wall tone resulting in elevated IAPs, compromising perfusion pressure to the abdominopelvic viscera.^{3,10} It is still unknown, whether preeclamptic patients have IAH and at what intraabdominal pressure this happens. IAH and Preeclampsia relation is still a subject of further study.

CONCLUSION:

Intra-abdominal pressure (IAP) is an important parameter in the surveillance of critical patients, widely used in intensive care units but when it comes to pregnancy little is known. Intraabdominal pressure if increased leads to pathophysiologic changes that results in ACS, which is a life-threatening condition caused by sustained acute elevation of IAP more than 20 mmHg with organ dysfunction, multisystem organ failure, high morbidity and mortality. Further, the lack of knowledge of the pathophysiology of IAH in pregnancy may leads to the missed or delayed diagnosis of ACS resulting in poor outcome. The IAP must be recorded in case of supine hypotension and preeclampsia. Further studies are needed to find out the IAP in different trimesters of pregnancy and its relationship with diseases like preeclampsia and supine hypotension.

REFERENCES

1. Malbrain ML, Cheatham ML, Kirkpatrick A et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. *Intensive Care Med.* 2006;32:1722-32.
2. Cheatham ML, Safcsak K, Sugrue M. Long-term implications of intra-abdominal hypertension and abdominal compartment syndrome: physical, mental, and financial. *Am Surg.* 2011;77:78-82.
3. Paramore RH. The Intra-abdominal Pressure in Pregnancy. *Proc R Soc Med.* 1913;6:291-334.
4. Zeeman GG. Obstetric critical care: a blueprint for improved outcomes. *Crit Care Med.* 2006;34:S208-214.
5. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 100: critical care in pregnancy. *Obstet Gynecol.* 2009;113:443-450.
6. Gaiser R. In: *Chestnut's Obstetric Anesthesia: Principles and Practice*. 4. Chestnut D, Polley L, Tsen L, Wong C, editor. Philadelphia: Mosby Elsevier; 2009. Physiologic changes of pregnancy; pp. 15-26.
7. Scott DB, Kerr MG. Inferior vena caval pressure in late pregnancy. *J Obstet Gynaecol Br Commonw.* 1963;70:1044-1049.
8. Chun R, Baghirzada L, Kirkpatrick A. Measurement of intra-abdominal pressure in term pregnancy: a pilot study. *Int J Obstet Anesth.* 2012;21:135-139.
9. Contreras F, Fouilloux C, Bolivar A, Betancourt MC, Colmenares Y, Rivero M, Israeli ZH, Velasco M. Endothelium and hypertensive disorders in pregnancy. *Am J Ther.* 2003;10:415-422.
10. Paramore RH. Eclampsia and its incidence [abstract] *Proc R Soc Med.* 1922;15:14-16.