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Case report

Case of ascites in pre-eclampsia

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Introduction

Pre-eclampsia is a life threatening syndromic disorder with variable presentation. The condition is known to increase maternal morbidity and mortality, and also contributes to significant proportion of perinatal mortality.^{1–4} Pre-eclampsia can present to a clinician with a variety of presentation. The patho-physiology of the syndrome is characterized by increased endothelial permeability and microvascular damage.^{2,5,6} But the presence of fluid, in various body cavities is rarely seen in pre-eclampsia^{2,5–9} and the presence of the same is associated with increased morbidity to the mother.^{2,5–9} This case was one in which the patient had fluid collection in multiple body cavities.

Case report

26 years old primigravida came to the labor room at 38 weeks period of gestation (POG) in labor. The history of the patient and the antenatal card showed, she was irregular in her

antenatal check-ups and last visit was 5 weeks back. The records showed her antenatal investigations and the blood pressure recordings were normal till 33 weeks period of gestation. The first trimester and second trimester ultrasound scans were corresponding to period of gestation.

On presentation, she was in active labor with no history suggestive of impending eclampsia. The examination of the patient showed, pulse rate of 90/min, respiratory rate of 14/min and blood pressure recording of 176–188/112–126 mmHg. The abdominal examination showed a term sized uterus with longitudinal lie and cephalic presentation. The per vaginal examination showed soft cervix at mid position, 40% effaced, 2 cm dilated and head at –2/3 station. The urine examination revealed a trace of proteinuria. A complete blood count (Hemoglobin, packed cell volume, platelet count), blood urea, serum creatinine, serum bilirubin, ALT and AST were sent and the reports were in normal limits. Fundoscopy was normal. She was given a single dose of tab nifedipine 10 mg. The patient was put on cardiotocographic recording as per the policy. The cardiotocographic trace showed late decelerations.

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The patient was diagnosed to have fetal distress and was planned for an emergency cesarean section within 45 min of coming to labor room.

She had an uneventful surgery and delivered a live baby. The amniotic fluid, placenta and the cord were stained with fresh meconium. There was no evidence of retro-placental hemorrhage. During the intra-operative period, under general anaesthesia her blood pressure was 156–180/98–120 mmHg and pulse rate of 90–100/min. She was started and prophylactic schedule of injection magnesium sulfate 10 g stat i.m. (5 g in each buttock) then to be repeated every 4 h for 24 h tab nifedipine 10 mg twice a day.

Immediate postoperative period was uneventful. She was started on clear oral fluids 8 h after the surgery, her blood pressure was recorded 142–154/96–100 mmHg and pulse of 98–106/min. She had an episode of hematemesis 16 h after the surgery and she also complained of visual symptoms – blind spots this was associated with distension of abdomen. She was put back on I/V fluids @ 100 ml/h. The blood pressure was 176/120 mmHg and the pulse was 120/min. The funduscopy revealed retinal detachment with hemorrhage. The investigations at this time showed – proteinuria ++, platelet count of 75,000/cubic millimeter, serum bilirubin 1.8 mg%, ALT and AST were normal, LDH was 454 IU/l and there was no evidence of hemolysis in the peripheral smear. She was started on pantaprazole infusion and tab atenolol. She was suspected to be developing HELLP syndrome and was transfused with fresh frozen plasma and single donor plasma platelet concentrate. The ophthalmologist advised optimum control of blood pressure.

On the second postoperative day the patient had no further episode of hematemesis. She continued to have tachycardia (135–154) with hypertension (150–160/100–110 mmHg) and distension of abdomen with breathing discomfort. She was started on injection frusemide to ensure adequate urinary output and oxygen inhalation at 4–5 l/min, the central venous pressure was normal. The ultrasonography showed ascites and bilateral pleural effusion. The echocardiography showed minimal pericardial effusion with normal ejection fraction. On postoperative day 3 and 4, the patient was afebrile, tolerating oral fluids and had adequate urine output. The CVP was normal. She continued to have the similar pulse and BP records. The investigations – complete blood count, BUN, S bilirubin, ALT, AST were normal. The peripheral blood smear was negative for hemolysis and toxic granules.

On postoperative day 5, the patient was started on injection dexamethasone 24 mg/day in three divided doses for the benefit in cases of HELLP syndrome and complicated pre-eclampsia. The rest of the treatment with tab frusemide, atenolol and nifedipine and oxygen was continued. The pulse rate after 3 doses of inj dexamethasone was 90–100/min with no requirement for oxygen inhalation. After 72 h the ascites, pleural effusion and the pericardial effusion had cleared up and the pulse settled to 60–70/min with BP of 140/90 mmHg. On the postoperative day 8, she was put on tapering doses of oral steroids (tab prednisone) by the ophthalmologist for a period of 6 weeks. She was discharged from the hospital on tab atenolol, nifedipine and prednisone with the instructions for weekly follow up at 2 weekly follow up at ophthalmology and gynecology OPD respectively. She became normotensive after 10

weeks and the eye symptoms regressed after a period of 3 months.

Discussion

Pre-eclampsia is a condition involving all the organ systems. The basic patho-physiology involves the release of vasoconstrictive agents, endothelial damage, hyperpermeability of the capillaries and microangiopathic hemolysis. This results in the presentation of the condition in various forms – hypertension, HELLP syndrome, visual symptoms, seizures, pulmonary edema, proteinuria etc.^{1,2,5,6} Ascites has been reported at cesarean section in severe pre-eclampsia cases. The analysis of this fluid has showed it to be transudate.^{2,6,10} The exact etiology of the same is not known, but intrahepatic portal hypertension is one possible explanation.^{2,10} The case presented above had a varied presentation with the collection of the fluid in the major body cavities and HELLP syndrome. She also continued to manifest with tachycardia of unknown cause – likely the part of generalized leaking of capillaries and microangiopathy, which settled with the use of steroids.

Woods et al² in a retrospective study found, the incidence of large-volume ascites in patients with HELLP syndrome who had caesarean was about 10%. They compared HELLP syndrome patients without ascites, to those with HELLP-associated ascites and found six-fold increase in the incidence of congestive heart failure and a nine-fold increase in the incidence of adult respiratory distress syndrome.

Woo et al⁶ in a series of cases reported the requirement of early termination of pregnancy in cases of women with ascites in pre-eclampsia. They observed a rapid deterioration in maternal condition with rise in blood pressure and increasing proteinuria.

Conclusion

Women with pre-eclampsia in the presence of ascites require intensive monitoring to avoid complications during the intra, and postpartum period. The management of this case with use of high dose steroids which were started for the ascites and later continued in the oral form for the ocular condition indicates the response and their use in complications of pre-eclampsia.

Conflicts of interest

None identified.

REFERENCES

1. Vajjyanath AM, Nayar B, Malhotra N, et al. Massive ascites in severe pre-eclampsia: a rare complication. *J Obstet Gynaecol Res.* 2002;28:199–202.
2. Woods JB, Blake PG, Perry Jr KG, et al. Ascites: a portent of cardiopulmonary complications in the preeclamptic patient with the syndrome of hemolysis, elevated liver

- enzymes, and low platelets. *Obstet Gynecol.* 1992;80:87–91.
3. Ko Ma-Lee Ko, Huang Lee-Wen Huang, Chang Jier-Zen Chang. Massive ascites complicating pre-eclampsia. *Tai J Obstet Gynecol.* 2009;44:267–269.
 4. Ashmore J Ashmore, Attapattu JA Attapattu. Massive ascites and bilateral hydrothorax complicating severe pre-eclampsia. *Ceylon Med J.* 1998;43:235–237.
 5. Foreman Jr CS Foreman. Massive ascites as a complication of severe pre-eclampsia. A case report. *J Reprod Med.* 1989;34:307–310.
 6. Woo JS Woo, Liang ST Liang, Wong VT Wong. Ultrasonic detection of maternal ascites in patients with severe pre-eclampsia. *Aust N Z J Obstet Gynaecol.* 1982;22:237–239.
 7. Lilford RJ Lilford, Lubbe WF Lubbe. Multiple serous effusions complicating pre-eclampsia. A case report. *S Afr Med J.* 1978;54:619–621.
 8. Suonio S Suonio, Saaranen M Saaranen, Saarikoski S Saarikoski. Left-sided hydrothorax in connection with severe pre-eclampsia. Case reports. *Int J Gynaecol Obstet.* 1984;22:357–361.
 9. Daponte A Daponte, Skentou H Skentou, Dimopoulos KD Dimopoulos. Massive vulvar edema in a woman with preeclampsia: a case report. *J Reprod Med.* 2007;52:1067–1069.
 10. Calvin S Calvin, Silva M Silva, Weinstein L Weinstein, et al. Characterization of ascites present at cesarean section. *Am J Perinatol.* 1991;8:99–102.

FURTHER READING

- Tukur J Tukur. The use of magnesium sulphate for the treatment of severe pre-eclampsia and eclampsia. *Ann Afr Med.* 2009;8:76–80.
- Woudstra DM Woudstra, Chandra S Chandra, Hofmeyr GJ Hofmeyr. Corticosteroids for HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome in pregnancy. *Cochrane Database Syst Rev.* 2010:CD008148.

Book review

Mastering Orthopaedic Techniques-Total Hip Arthroplasty, in: Rajesh Malhotra (Ed.), 1st ed. Jaypee Brothers Medical Publishers (P) Ltd., Hardbound, 2012, p. 642, ISBN: 978-81-8448-898-2.

Total hip arthroplasty has gradually evolved over the last century from an exotic unusual surgery in the early twentieth century to be hailed as the “Operation of the Century” now, becoming a standard of care surgery for advanced hip arthritis. The editor has been successful in providing a diversity of opinion about this surgery with a galaxy of eminent authors from all over the globe, quite a large number of them enjoying international reputation. At the same time, there are fairly large number of authors from India who maintain the emphasis on clinical problems quite unique to India.

The book successfully provides the readers the complete range of perspective from the full range of surgical approaches to the hip to the wide variety of implants in current usage, including latest in the implant market as well as some devices which still remain under development. The early chapters explain the principles, design rationale, tribology, pre-operative planning, soft tissue balancing and almost all surgical approaches used in hip arthroplasty. At this stage, one does feel that probably a chapter on hip biomechanics and revision of some unique features of hip anatomy would have provided a strong basis on issues being covered in these early chapters. About one fourth of the book (Ten chapters) has been dedicated to explain the surgical techniques of various manufacturer specific implant systems. The unique problems of difficult primary hip arthroplasty in hip dysplasia, protrusion, inflammatory arthritis, acetabular fractures and bony ankylosis have been described in detail. Emerging role of robotics and navigation in total hip arthroplasty has also been adequately covered.

The book remains a good guide for orthopaedic surgeons doing primary hip arthroplasty only. This book can only be an

adjunct to revision hip arthroplasty surgeons with its sections on periprosthetic fractures and infections. But the book is targeted at orthopaedic surgeons at the beginning of their hip arthroplasty practice and those who are targeting only primary hip arthroplasty. This targeted reader group will definitely find excellent information to provide a solution to the challenging problems they face in their practice.

The final two chapters are written by international authors primarily based on the practice conditions and environment in their respective countries of origin (United Kingdom & USA). It would be quite difficult to implement any of these ideas in India with absence of any joint registry or any long term data in most of the institutions. Quality and safety can be improved only if one can analyze own results with long term follow up data. It definitely brings to attention the burning requirement of a joint registry in India, starting at an institutional level and going to national level. Perhaps, armed forces can once again take a lead in this field by setting up a joint registry.

To conclude, the editorial team of this book needs to be commended for their special efforts to bring out a comprehensive textbook on total hip arthroplasty with a team of authors who are internationally recognized authorities in the field. This book will surely adorn the shelves of the library of all institutions where total hip arthroplasty is being practiced.

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