HYPERTENSIVE CRISIS IN POSTOPERATIVE CASE OF WILMS TUMOUR-AN UNUSUAL CASE

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Abstract

Wilms Tumour also known as nephroblastoma,a highly malignant embryonal neoplasm,involving one or both kidney. It affects children between 3 to 5 years of age and unilateral but 5% cases bilateral. Disease occur in about 1 out of 2-2.5 lakh children. Hypertension is noted in children with wilms tumour either at

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Abstract :

Wilms Tumour also known as nephroblastoma,is a highly malignant embryonal neoplasm,involving one or both kidney. It affects children between 3 to 5 years of age and unilateral but 5% cases bilateral. Disease occur in about 1 out of 2-2.5 lakh children. Hypertension is noted in children with wilms tumour either at the time of initial presentation or in the post operative period due to increase renin secretion. We report a case of postoperative hypertension in a case of wilms tumour. The complexity of anaesthesia was determined by the size of the tumour ,increased intra abdominal pressure & hypertension.

Key words:Wilms Tumour,Hypertension.

Introduction :

Wilms Tumour also known as nephroblastoma, is a highly malignant embryonal neoplasm,involving one or both kidney. It affects children between 3 to 5 years of age and tumour is unilateral,but in 5% cases bilateral. Disease occur in about 1 out of 2-2.5 lakh children. Due to increase renin secretion hypertension can be detected in children with wilms tumour either at the time of initial presentation or in the post operative period and symptoms may include abnormally large abdomen, fever, nausea, vomiting, hypertension, tachycardia, and tachypnea. Diagnostic evaluation includes ultrasound, computed tomography (CT) scans, complete blood count (CBC),urinalysis, and coagulation tests. Treatment consists of multimodal treatment with subsequent surgery[1-2].

Anaesthesia management is challenging as large size of wilms tumour (12.9*10*11),hypertension,blood loss and anemia. The challenging part of our care was postoperative hypertensive crisis and it was managed successfully.

Case Report:

1 year 1 month old male baby,weighing 9.8kg presented with complaints of increased size of the abdomen on left side since 1 year. Preoperative evaluation was done which revealed increased size of the abdomen. Baby

was average built and playful,heart rate 100-106/minute, preoperative blood pressure 90-100/60-70 mmHg, SpO2 100%.

Abdomen examination revealed swelling extending from the left side of abdomen crossing midline with firm consistency.Lab results revealed Hemoglobin-9gm% and rest all investigations are normal. Computed tomography showed a solid-cystic, enhancing mass lesion seen arising from the lower polar and inter polar region of the left kidney with the size of 12.9*10*11 with foci of peripheral calcification suggestive of neoplastic etiology wilms tumour.

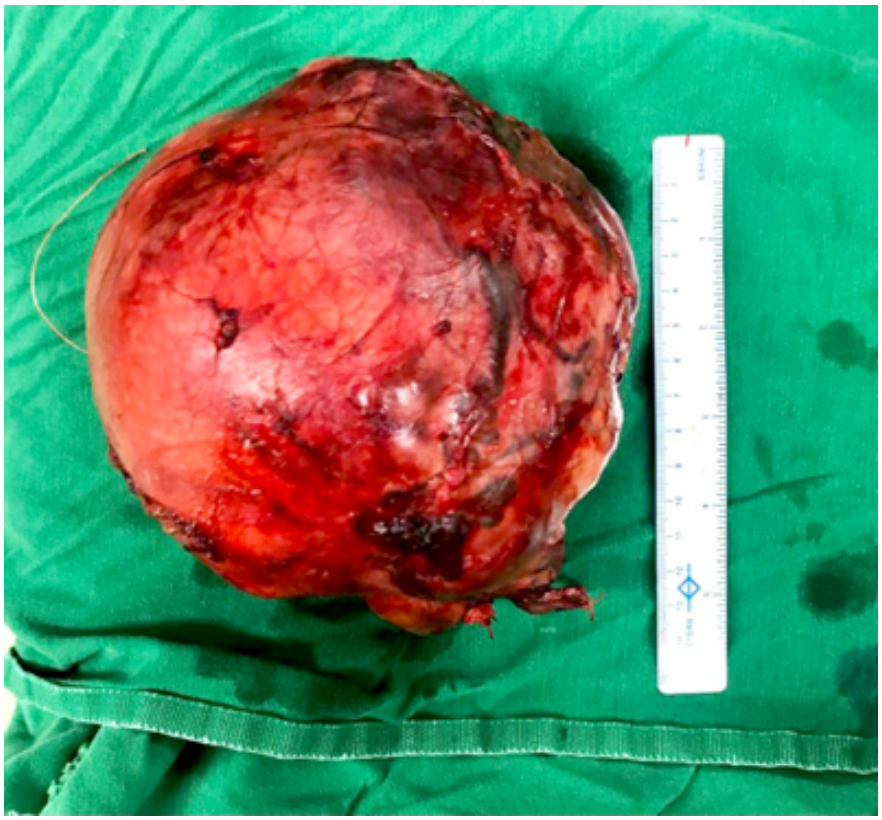

Figure-1 Macroscopic Appearance

Gross Examination of left kidney:Solid cystic mass which was occupied the lower and inter polar region.

On the day of surgery consent taken ,blood & blood products booked, ASA standard monitors attached. Premedicated with Inj Glycopyrrolate (0.004mg/kg),Inj Midazolam (0.02mg/kg),preoxygenated with 100% O2,Inj.Fentanyl 20mcg was given and induced with Inj Propofol (2mg/kg),Inj.Vecuronium 1 mg given and after three minute intubation was done orally with size 4.5 uncuffed endotracheal tube. After checking air entry,tube was fixed and inj.Vecuronium 0.5mg for maintenance was given.Ryles tube 10french & right internal jugular vein 5french 8 cm central line was secured.

Patient was maintained on O2:AIR(50:50) , isoflurane at 0.8. In left lateral position epidural catheter was secured at L1-L2 level and Inj.Ropivacaine 0.2% infusion started to maintain blood pressure and intra operative analgesia. Intraoperative blood loss due to lot of adhesions and pressure symptoms causing fluctuations of BP and CVP and was managed with PRBC 8-10ml/kg and fluids.Vitals BP was maintanined between 90-100/60-70mmhg intraoperatively. After visualizing the mass,nephrectomy was performed.Blood loss 200 ml,urine output-40ml,total IV fluids-410ml,inj.Dexa1mg &inj.PCM 15mg/kg was given.

After surgery baby started taking spontaneous attempts and BP started rising and initially treated with Inj.Xylocard 1mg/kg & Inj.Labetalol 0.2mg/kg. Due to persistent high BP inj.Labetalol 0.5mg/kg/hr started, shifted to PICU, mechanically ventilated for better control of BP and extubated on post op day1.Intraoperatively hypertension could not be appretiated due to epidural infusion and blood loss.

Postoperatively inj.Labetalol continued for 3 days and discharged after 10 days of hospitalization with T.Amlodipine 2.5 mg OD, T.Minipress 2.5mg BD and T.Labetalol (1-3mg/kg)BD

Discussion :

Wilms Tumour is a highly malignant embryonal neoplasm. It may involve one or both kidneys. Usually the tumor is unilateral but in 5% cases it may be bilateral. Disease occur in about 1 out of 2-2.5 lakh children[2].

HT is known to be associated with several childhood cancers at diagnosis,such as WT, neuroblastoma, brain tumors, and pheochromocytoma. It could be due to renin secretion by the tumor or secondary to mechanical mass effect causing renal vascular compression or thrombosis. Hormonal secretion of glucocorticoids or catecholamines, treatment with steroid chemotherapy, and increased intracranial pressure could be other causes of HT[3].

In children with WT, HT results from increased renin production secondary to intra-renal vascular compression. Alternatively, renin may also be produced by tumor cells. Increased plasma renin concentration has been reported in approximately 80% of hypertensive WT children at diagnosis, and relapse was observed in 3 out of 4 patients with increased plasma prorenin/renin concentrations. Renin production is controlled by the renin–angiotensin system, which plays a decisive role in maintaining BP homeostasis.

It could be due to increased renin secretion in response to renal ischemia produced by the pressure of the tumor on the hilar or intrarenal vessel[4]. Moreover, the tumor itself could directly be responsible for HT by producing renin itself. In addition, as the Brenner–Barker hypothesis noted, there is a significant reduction of nephrons with the development of renal HT and progressive renal failure. Approximately 20–55% of children with WT reportedly present with HT at diagnosis

Anesthetic management can be challenging in children with HT, diagnosed in the pre-operative period. HT could be severe enough to cause encephalopathy and cardiovascular compromise. Secondary hyperaldosteronism and hypokalemia could manifest itself as chronic HT significant intraoperative bleeding could result from improperly controlled HT. Pre-operative recognition of HT and appropriate pre- and perioperative treatment is mandatory for safe surgical treatment. Angiotensin-converting enzyme inhibitors are the drugs of choice[5]. One could add a short-acting beta-antagonist to control HT in severe cases. Babies should be evaluated preoperatively and prophylactic treatment of borderline BP in WT can be considered to prevent post op complications.

Conclusion:

Preoperative Hypertension may not be diagnosed in WT. Intraop and post operative HT may usually present after surgery due to renal ischemia. Multimodal Antihypertensive treatment and close monitoring lead to a successful outcome in our case.

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