

# Crossing Vessels as a Cause of Pelvi-Ureteric Junction (PUJ) Obstruction

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## Abstract

Foetal hydronephrosis is commonly caused by congenital pelviureteric Junction (PUJ) obstruction. It is either due to nerve deficiency or due to intrinsic muscular defect at PUJ or it may be due to lower polar aberrant crossing vessel just compressing the PUJ. The purpose of this study is to see the contribution of crossing vessels in causing the PUJ obstruction. **Aims and objectives:** (1) to find the contribution of crossing vessels in causing PUJ obstruction on naked eye examination peroperatively, (2) To histologically verify the cause of PUJ obstruction in cases of crossing vessels compressing the pelvi-ureteric junction (PUJ). **Patients and methods:** 27 cases of Congenital PUJ obstruction were taken during four year period who

for operation. All underwent Anderson Hynes Pyeloplasty and specimen of PUJ with narrow segment below it was taken and preserved in 10% formalin. The specimen was sent to histopathologist along with other specimens where PUJ obstruction was not caused by crossing vessels for nerve detection by S-100 immunohistochemistry and for histopathological examination. **Results:** Five out of 27 cases of PUJ obstruction were caused by crossing vessels and in these five cases, four (80%) were having normal histology showing that crossing vessels were the cause of PUJ obstruction merely compressing the PUJ mechanically. **Key Words:** PUJ, crossing vessels, Pyeloplasty.

were candidates

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## INTRODUCTION

Pelviureteric junction obstruction is the most common cause of significant dilatation of foetal kidneys.<sup>1</sup> It is now increasingly diagnosed prenatally. Its etiology is unclear.<sup>2</sup> Most common cause is congenital hydronephrosis which is thought to be due to intrinsic defect of muscular development or due to deficient nerves in the obstructed narrow segment. Other causes include obstruction from outside: crossing vessel, tumor compressing PUJ, fibrous cord, enlarged lymph node compressing the PUJ or retroperitoneal fibrosis. Similarly intramural causes include congenital deficiency of nerves or muscles, fibrosis of PUJ due to previous surgery or stones or refluxing ureters, or tumor of the wall of PUJ. Intraluminal causes include stones, polyp, mucosal folds, transitional cell carcinoma of renal pelvis. Congenital Hydronephrosis is associated with crossing vessels in 26% of cases. F.J.B. Sampaio showed 65% relation of the crossing

vessel to the anterior surface of the PUJ and in 45% of these cases the relationship was with the inferior segmental artery.<sup>3</sup> Van Canghai et al obtained digital angiography in patients before endopyelotomy and found an associated vessel in 39% of patients with PUJ obstruction.<sup>4</sup> Crossing vessel is not an aberrant or accessory vessel as it is a misnomer and it is actually a lower polar segmental artery and it would be more appropriate to call it "vascular bar".<sup>5</sup> This segmental artery is end artery. It is mentioned in literature that this crossing vessel is not the primary cause of PUJ obstruction rather PUJ is already obstructed by congenital muscular defect and crossing vessel only causes partial obstruction and redundant pelvis kinks and falls over the vessel increasing hydronephrosis.<sup>6</sup> Others think that this vessel is the mere cause of obstruction and its transposition is all that is required in the management.<sup>7</sup> Recently Stern JM et al showed that obstruction is due to crossing vessel

and not due to intrinsic defect as they per operatively put a 22G cannula in the pelvis and performed the Whitaker test before and after transposing the crossing vessel. In all the patients of crossing vessel the mean renal pelvic pressure significantly declined after transposing the vessel showing that lower polar vessels directly contributed to UPJ obstruction by causing extrinsic compression<sup>8</sup>. If the segment was obstructed on Witaker test after releasing the compression by the vessel then they performed dismembered pyeloplasty and placed the newly formed PUJ anteriorly to the vessel and if it was not obstructed then they just transposed the vessel cephalad and fixed it there with peripelvic fat. Recently this procedure is being done laproscopically<sup>9</sup>. Previously people used to clip this crossing vessel but this is wrong as it causes about 7.4-38 % loss of functional renal parenchymal tissue<sup>3</sup>. Now a days artery is being transposed cephalad and vein may be ligated. Routine transposition of crossing vessel during laparoscopic or robot assisted dismembered pyeloplasty may be unnecessary.<sup>10,11</sup>

Crossing vessel can be diagnosed by different ways preoperatively. One way is renal angiography but this is a difficult way and not cost effective. Second way is to do CT IVU but its availability is also a problem.<sup>12</sup>. Thirdly MRI with contrast can tell about the crossing vessel. A paper has shown that it can be diagnosed by ante grade pyelography which shows a typical finding of acute posterior angulation of ureter just below the PUJ<sup>13</sup>. There are different ways of treatment of congenital PUJ obstruction ranging from minimal invasive procedures like endopyelotomy with cold knife, electrocautery or laser and balloon dilatation to other different surgical procedures namely Foley VY Pyeloplasty, Scardino and Prince flap technique, Culp Devede Pyeloplasty and the classical dismembered Anderson Hynes Pyeloplasty. Anderson Hynes Pyeloplasty is still the gold standard as it has 95% success rate<sup>14</sup>. Recently with improvement of surgical techniques these procedures are being done laproscopically. Robotics have also tried this procedure with excellent success rate.<sup>15,16</sup>

The purpose of this article is to evaluate that what is the incidence of the crossing vessel in our cases and whether this is the sole cause of obstruction or PUJ is congenitally obstructed by intrinsic muscular defect or nerve deficient.

**Figure-1**  
**X-ray showing the right PUJ obstruction**



#### **AIMS AND OBJECTIVES**

- To see the contribution of crossing vessels in causing the PUJ obstruction
- To confirm that mechanical compression of crossing vessel is the cause of PUJ obstruction in crossing vessel cases or there is some intrinsic defect of morphology as well.

#### **MATERIALS AND METHODS**

**Study Design:** Cross Sectional Study

**Setting:** Departments of Urology Allied Hospital Faisalabad and Mayo Hospital Lahore.

**Duration of Study:** 4 years and 3 months from January 2006 to September 2010.

#### **Main Outcome Measures**

1. Per operative finding of crossing vessels
2. Presence / obscene of nerves at PUJ
3. Normal / abnormal histology at PUJ level

**Subjects:** 27 cases of PUJ obstruction fulfilling the inclusion criteria were included in the study. Patients were taken from Urology OPDs of Allied Hospital Faisalabad & Mayo Hospital Lahore.

**Sample selection:** The patients having the following characteristic were included in the study.

1. Patient having PUJ obstruction diagnosed on the basis of ultrasound, IVP and / or DTPA renal scan.
2. Patients having indication for surgery like symptomatic patient, B/L PUJ Obstruction, Unilateral PUJ obstruction with decreased renal function of 40% or less as compared to normal contra lateral kidney function on DTPA renal scan, deteriorating renal function of > 10% on serial DTPA renal scan and grade III or IV hydronephrosis.

3. American Association of Anesthesiologist (ASA) score I or II

**Exclusion criteria**

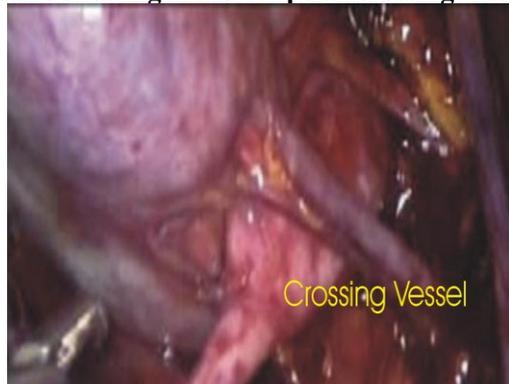
1. Patient having secondary PUJ obstruction
2. Patients having stone with PUJ obstruction as inflammation from stone can hamper histological outcome.

**Data Collection Technique:** After approval from Hospital Ethical Committee and taking informed consent patients fulfilling the inclusion criteria were included.

1. All patients underwent Anderson hynes pyeloplasty
2. Per operatively finding of crossing vessel was noted.
3. Crossing vessel was transposed posteriorly in case of anterior crossing vessel and newly formed PUJ was constructed in front of the crossing vessel.
4. Specimen of redundant pelvis, PUJ and narrow segment of ureter below PUJ was taken and preserved in 10% formalin solution and sent to the histopathologist for histopathological examination and for nerve detection by immunohistochemical stain S-100.

All the data like patient age, sex, side, number of cases of crossing vessels and their histopathological findings and presence or absence of nerves was recorded on a specially designed proforma.

**Figure- 2**  
Picture showing the lower polar crossing vessel

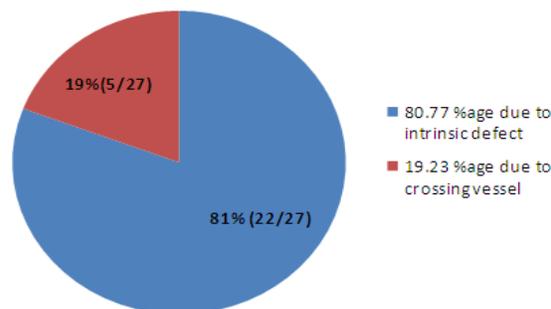


**RESULTS**

27 patients were taken for this study from Urology Department Allied Hospital Faisalabad and Mayo Hospital Lahore who underwent Anderson Hynes

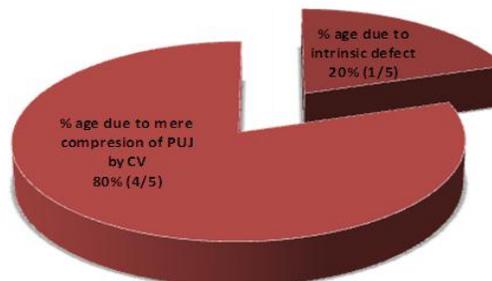
pyeloplasty during the four years period. Eighteen (66.7%) patients were male and 9 (33.33%) were female showing a male to female ratio of 2:1. Sixteen (59.29%) patients were having left sided PUJ obstruction and 9 (33.33%) patients were having right sided PUJ obstruction, while 2 (7.4%) patients were having bilateral PUJ obstruction. Out of 27 patients who were operated in Urology Department for PUJ obstruction, 5 (18.52%) were diagnosed per operatively to be due to lower polar crossing vessel.

**Figure-3**  
Showing the percentage of PUJ obstruction due to crossing vessel and due to intrinsic defect



In all of these, Anderson Hyens pyeloplasty with transposition of vessel posteriorly was done and specimen taken for histopathology. Interestingly four out of five PUJs showed the presence of nerves in these specimens showing that this crossing vessel is the mere cause of compression of PUJ leading to obstruction of PUJ and these PUJs were having normal histology as opposite to previous belief that these PUJs are congenitally obstructed with deficient nerves and crossing vessel only increases the angulation of redundant pelvis augmenting the defect.

**Figure-4**  
Graph showing the distribution of normal histology among the 5 Crossing Vessels (CV) cases

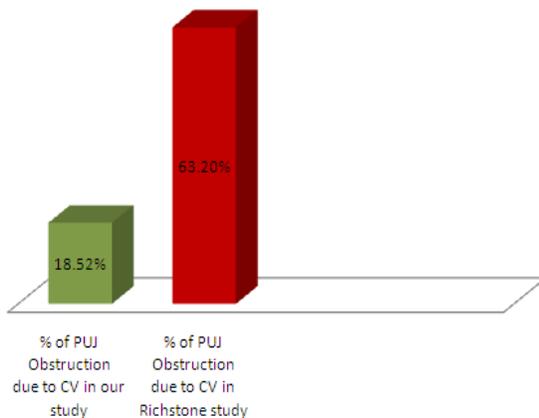


## DISCUSSION

The PUJ obstruction is the most common cause of hydronephrosis in infants. The male to female ratio of 2:1 in our study is about the same as that mentioned in literature<sup>17</sup>. Side wise distribution in our study is that left side is involved in 59.29% patients of PUJ obstruction and it is consistent with literature review<sup>18,19</sup>.

Crossing vessels were found in 18.52% of cases in our study which is in contrast with the results published in literature where Richstone L and colleagues found crossing vessels in 63.2 % of cases of UPJ obstruction<sup>20</sup>.

**Figure-5**  
**Graph showing the comparison of our study with Richstone study**



In our study 80% of cases were having normal histology and normal nerve distribution meaning that compression by crossing vessel was the only cause of PUJ obstruction whereas 20% had intrinsic defect and it is in concordance with the results showed by Jenny H and colleagues where pathologist was able to identify the cause of obstruction in 71% of cases as there was no intrinsic defect and only muscle thickness was more in crossing vessels cases.<sup>21</sup> This finding is in contrast with the findings of Stern JM and colleagues who found that intrinsic defect was found in 57% cases of crossing vessels<sup>8</sup>.

Our finding of normal histopathology is also consistent with the finding of Kajbatzadeh AM and colleagues<sup>21</sup>.

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