DOI: 10.7860/JCDR/2015/14966.6614

Case Report

Anatomy Section

Pelvic Floor Support Defect in Apical Anterior Vaginal Prolapse with Cervical Hypertrophy. Review with Case Report in a 20-year-old Cadaver

KALPANA CHHETRI

ABSTRACT

Apical anterior vaginal wall prolapse (AVWP) with central defect is uncommon in young non hysterectomized patients causing considerable mortality after the fourth decade of life. Its high propensity to recurrence poses the greatest challenge to pelvic reconstructive surgeons. Approximately 40% of women with prolapse have hypertrophic cervical elongation and the extent of elongation increases with greater degrees of prolapse. Women with prolapse either have inherent hypertrophic elongation of the cervix which predisposes them to prolapse or the downward traction in prolapse leads to cervical elongation. The Pelvic Organ Prolapse Quantification (POP-Q) examination includes measurement of the location of the posterior fornix (point D) with the assumption that this measurement is associated with cervical elongation. Multifocal site involvement with apical and perineal descent primarily afflicts elderly, postmenopausal women after the fourth decade while cervical hypertrophic elongation with prolapse is observed in younger women less than 40 years of age. A review of the anatomical implication of the association of cervical hypertrophy in prolapse is carried out in this article.

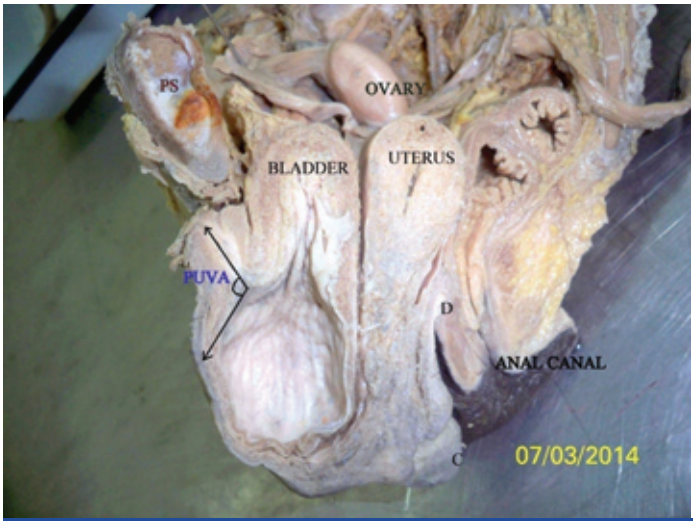
We observed a combination of distension type anterior vaginal prolapse with apical descent and cervical hypertrophy in a 20-year-old cadaver during routine dissection for undergraduate medical students at Sikkim Manipal Institute of Medical Sciences in 2013.

Distension type anterior vaginal prolapse with central defect is rarer as most reported cases are of the displacement type, paravaginal defect. Hypertrophic cervical elongation is either the cause or consequence of prolapse and its identification before reconstructive surgery is paramount as uterine suspension in the face of cervical elongation is contraindicated. Inappropriate identification of all support defects and breaking of tissues is the primary cause of failure of laparoscopic pelvic reconstructive surgery.

Keywords: Anterior vaginal wall prolapse (AVWP), Arcus tendineus fasciae pelvis (ATFP), Cervical hypertrophy, Utero-vaginal, Vault

CASE REPORT

During routine dissection for undergraduate students we observed distension type AVWP of fourth degree with hypertrophic cervical elongation in a 20-year-old female cadaver. The bladder has herniated into the anterior vaginal fornix lying 4.5cm below the pubic arch. Shortening of urethra (2cm) with loss of urethro-vesical crease and increased Posterior Urethro-Vesical Angle (PUVA) 110° is observed [Table/Fig-1]. The descent of both POP-Q point C (leading edge of cervix) and D (posterior fornix) implicates loss of apical support and transverse defect of the pubocervical fascia from the pericervical ring into which the cardinal and uterosacral ligaments insert. The increased distance (65mm) between point C and D implicates cervical hypertrophy [Table/Fig-2]. Central defect with loss of level 1 support of uterosacral-cardinal ligament complex from the pericervical ring is observed. Shortening of urethra demonstrates its avulsion from the urogenital diaphragm, level III support anteriorly. The prolapse was graded according to the Pelvic Organ Prolapse Quantification (POPQ) system. The findings were photographed and labeled [Table/Fig-3].



[Table/Fig-1]: Shows grade three anterior vaginal wall prolapse with herniation and exteriorization of base of bladder into the anterior fornix. Rectocoele with grade two uterine prolapse is also seen. Shortening of urethra (2cm) with cervical hypertrophic elongation (distance between POP-Q point D (posterior fornix), C-leading edge of cervix). Vault prolapse with descent of point D and increased posterior urethro-vesical angle (PUVA) 110°

[Table/Fig-2]: Finger in lateral vaginal sulcus which is effaced and lies below pubic arch. Loss of rugae of anterior vaginal wall observed in distension type prolapse is portrayed. Hypertrophied lips of the cervix and elongation is evident

	Berger		Present study
	Berger NORMAL	PROLAPSE	PRESENT CASE
Ba (cm)	–1.8 ± 0.8	2.8 ± 1.8	7
C (cm)	–6.6 ± 1.5	–1.0 ± 4.2	9
D (cm)	–8.7 ± 1.6	–5.6 ± 2.7	-2
Distance between Points C and D (cm)	2.0 ± 1.1	4.6 ± 2.9	6.5
Bp (cm)	–1.8 ± 0.6	–1.1 ± 1.5	-6
Most Dependent Point(cm)	–1.5 ± 0.7	3.0 ± 2.0	9
Genital hiatus	3.2 ± 1.0	5.8 ± 1.5	6
Perineal Body	3.6 ± 1.2	2.9 ± 0.9	3
Total vaginal length	10.4 ± 1.2	9.9 ± 1.1	7

[Table/Fig-3]: Comparison of points of measurements of POPQ System between study by Berger et al., and present study

DISCUSSION

Utero-vaginal prolapse primarily occurs in paruos, elderly peri and postmenopausal women causing considerable mortality after the fourth decade of life [1,2]. Multifocal site involvement with vault prolapse is observed in women above 40 years while lower grades of prolapse with cervical hypertrophy without apical descent is documented in younger women [3]. Onowhakpor et al., in their study demonstrated higher incidence of utero-vaginal DH3 prolapse among patients involved in physically exerting occupations the incidence of which decreased as the exposure to physical stress decreased. They observed that 1.6% of major gynecological surgeries and 11.3% of total hysterectomies were for utero-vaginal prolapse in postmenopausal women. They documented 61.9% of patients had prolonged labour of >12 hours with 42.9% of the cases having history of prolonged labour along with difficult vaginal delivery and 19.0% cases of forceps delivery [1]. Okonkwo and colleagues in a study of women subjected to pelvic reconstructive surgery observed lower grades of prolapse with cervical elongation in younger women of ≤ 40 years with a mean age of 32.8 years while multifocal site involvement with apical and perineal descent was non-existent in the younger non hysterectomized women. Hypertrophic cervical elongation was demonstrated in 6.3% of the younger group with an incidence of 1.58% per year, cystocele in 8.9%, rectocoele in 6.3% and vaginal cuff prolapse (apical descent) in 8.9% with no perineal descent with 27.8% having grade one prolapse, 50% grade two and 22.2% grade three prolapse while in the older group it was observed to be 8.6%, 31.4% and 60% respectively. They argued that hypertrophic cervical elongation existed without any other anatomical defect in younger women and is more common in the child bearing age where prolonged labour and cervical dystocia are the aetiological factors while vaginal vault prolapse, multifocal site prolapse and higher grades were observed in patients of ≥ 40 years [3]. They also reported that 69.6% of the cases of prolapse occurred in older women with a mean age of 56.543 years, but without cervical hypertrophy and history of chronic medical conditions.

However Strobhen et al., report a higher incidence of chronic medical conditions in the younger group in their study 1997. They observed a mean age of 30.3(± 3.4) in younger patients, and 60.6 (± 11.9) in older women and concluded that congenital anomalies, neurologic or connective tissue disorders predisposed younger women to prolapse. They identified higher prevalence of underlying medical disorders (22.2%) in younger women than in (6.7%) older women (p <0.05) and lower mean parity (2.8 versus 3.4 p <.05), single site prolapse (56% versus 23 %) and lower grades of prolapse (grade three and above 33% versus 87%) in younger patients [4]. Fourth degree prolapse with multifocal site involvement and perineal descent in a 20-year-old is by far the youngest reported case of utero-vaginal prolapse. It could be attributed to some congenital predisposing factors of connective tissue disorders like Marfan syndrome and Ehlers Danlos syndrome as reported by Jab et al., & Carley et al., [5,6]. Chaffarino et al., also documented a familial history in urogenital prolapsed [7]. Dietz in his study of bladder neck descent in nulligravid twins and siblings observed a genetic predisposition to bladder neck mobility and prolapse. The monozygotic twins had a greater correlation for bladder neck descent than dizygotic twins and non twin sisters. His dizygotic/sister correlation was 0.42, for bladder neck descent and 0.18 for oblique bladder neck descent and 0.07 for urethral mobility. Low dizygotic correlation suggests the influence of dominant genes. His study of nulligravid women ruled out child birth as a major environmental risk factor for the development of prolapse. He observed that additive genes accounted for 59% of cases of bladder-neck mobility and descent [8]. Doung in his study observed that though the prevalence of prolapse is higher in white women who had higher grades of prolapse occurring after the fourth decade of life more severe degree of prolapse occurred in relatively younger women in Asian and African sub continents [9]. Distension type AVWP results from overstretching of the vaginal canal during delivery or with atrophy of ageing of tissues with loss of vaginal rugae. The displacement type or paravaginal prolapse which is more prevalent is attributed to pathological detachment of the anterolateral support from the arcus tendineus fasciae pelvis causing anterior segment descent with intact rugae [10-12]. The detachment of the pubocervical fascia from the white line leads to loss of the anterolateral fornices while the vaginal rugae remain undamaged [13]. The presence of combined features of both distension and displacement type of prolapse with central defect is infrequent and can be seen in the present case. Detachment of the pubocervical fascia and uterosacral-cardinal ligament complex from its insertion in the pericervical ring results in transverse defect leading to greater descent of the bladder base into anterior vaginal fornix. Midline and distal defect have resulted in avulsion and shortening of the urethra [14,15]. Anterior vaginal wall prolapse (AVWP) poses the greatest challenge to pelvic reconstructive surgeons on account of its high propensity to recurrence. Inappropriate identification of all support defects and inadequate suspension of the anterior wall apex is the major cause of failure of long term success of anterior wall reconstructive surgeries [16]. De Lancy et al., describes a three level support of the pelvic viscera, the level I support to the upper vagina and cervix by the uterosacral-cardinal ligament complex. Level II support is by the attachment of ATFP anteriorly to pubic bone and posteriorly by levator ani to the lateral pelvic wall. The level III support is by the perineal membrane and deep perineal muscles anteriorly and perineal body posteriorly. Vault prolapse depicts the disruption of (level I support) uterosacral-cardinal ligament complex to vaginal apex and upper cervix. The detachment of ATFP (level II) from the pubis as described has lead to greater descent of the anterior vagina and bladder than uterus in the present case. Uterosacral -cardinal ligament complex is disrupted and the pubococcygeus muscle shows localized atrophy however the lateral attachment of levator ani and ATFP appears intact posteriorly [17]. The effacement of the lateral vaginal sulci is evident with the anterior and posterior fornices having descended outside the introitus which suggests a combination of paravaginal and central defect. Decreased length (2cm) of the urethra suggests a distal avulsion of the urethra from the urogenital diaphragm due to disruption of deep perineal muscles (level III) [14,16,17]. In another study of women with AVWP in 2002 De Lancy observed that most cases had either right (89%) or left (87%) paravaginal defect and reported that the anterior attachment of ATFP to the pubic bone was intact while its posterior part was detached from the ischial spine for a variable distance with atrophy of the pubococcygeus muscle in more than half of the patients [18]. A study in Tamil Nadu, India in 1997 revealed that 40% of the women suffered from uterine prolapse after the very first delivery and 31% from second delivery onwards. It was observed that 18 of the 32

women with prolapse were exposed to heavy manual labour within a fortnight following delivery. They observed 50% of the women had been living with the condition for more than ten years. The mean age at which women developed symptoms was 26.2 years. They observed 40% of the women had all the deliveries at home, while 20% had hospital deliveries [19]. Similar conclusions were made by Radl and colleague in a study in Nepal. They reported that sociocultural risk factors like early marriage, multiple deliveries, and desire for male child could not be reformed. The present case supports the notion that higher grades of prolapse afflict younger women in developing countries where women are subjected to strenuous physical labour, early marriage, inaccessibility to health care and delivery by untrained persons [20]. Point D the presumed insertion site for the uterosacral ligament is included in the POP-Q classification of prolapse. We observed descent of point D (0.5cm) way below the pubic symphysis. When using the POP-Q prolapse quantification system a patient with cervical hypertrophy without vault prolapse has a positive point C and well supported point D [21]. Pelvic organ prolapse is commonly believed to be associated with cervical elongation [22]. The cervix being inherently elongated in women with prolapse or the downward traction of prolapse causing hypertrophic cervical elongation indicating that cervical elongation is a familial trait that predisposes women to prolapse. Berger et al., in their study revealed that cervical elongation is invariably associated with prolapse with one third (40%) of women with prolapse having hypertrophic cervical elongation that afflicted younger women and extent of elongation was directly proportional to the degree of prolapse. They defined cervical elongation as an absolute cervical length greater than 33.8mm and documented that prolapse was associated with cervical elongation and uterine corpus elongation but the cervical elongation was more. They documented that cervix was 36.4% (8.6mm)longer in 40% women with prolapse and the corpus length (internal os to fundus) was 4.9mm (11.3%), (p=0.012) and the total uterine length was 20.1% (13.5mm) longer in women with prolapse ($p < 0.001$). They observed that cervix to corpus length ratio was 21.8% greater in women with prolapse than in those with normal support with no difference in the location of point D in women with prolapse with or without cervical elongation. Their patients with cervical elongation had lower parity and were younger than the women with prolapse with normal cervical length [23]. Uterine preservative surgery is contraindicated in prolapse associated with cervical elongation. Uterine suspension or McCall culdoplasty alone is insufficient in such cases where persistent cervical prolapse necessitates partial cervical amputation or Manchester procedure [24,25]. Cervical elongation in AVWP also influences the surgeon's decision of peritoneal entry site and the position of initial circumferential incision and appropriate extraperitoneal dissection during vaginal hysterectomy [26]. Ibeanu et al., describe cervical elongation as a distance of ≥ 8 cm from point C to point D and observed that cervical elongation increased with parity while the opposite was observed by Berger et al. We observed a length of 6.5cm between point C and D which was more than that of Berger but less than that of Ibeanu and a corpus length of 3.5cm which is less than the normal upper limit described by Berger. Both the above studies observed that the average location of POPQ point D remains well suspended while we observed descent of both point C and D which were outside the introitus [23,26,27]. Significant cervical hypertrophy (65mm) with vaginal vault prolapse with decreased uterine corpus length (45mm) is evident in the present case. Cervical hypertrophy is frequently associated with anterior vaginal prolapse and a thorough evaluation of the anatomical support defect and the differential descent of the POPQ point C and D can be used as targets for presurgical evaluations for the right surgical approach. Hypertrophy of cervix and elongation is suggestive of cervical tear during delivery and on account of prolapse. Perineal descent with a wide genital hiatus indicates perineal tear during labour. The uterus is retroverted with loss of angle of ante-flexion. Women with retroverted uterus are more prone to prolapse and accounts for 69% cases of grade 2-4 prolapse and present with urogynaecological symptoms at earlier age [28]. Acquired retroversion is frequently caused by endometriosis, pelvic inflammatory disease and pelvic tumours as well as prolapse [29-31]. The retroversion of the uterus observed here is either on account of prolapse or pelvic inflammatory disease. Absence of the vaginal rugae is suggestive distension type prolapse on account of detachment of the vaginal epithelium from the endopelvic fascia [10,32].

CONCLUSION

Inherent cervical hypertrophic elongation is either the cause or consequence of pelvic organ prolapse as it is usually associated with prolapse. Advanced stage of prolapse in a 20-year-old cadaver divulges the sad socio cultural practices and dismal state of health care facilities in developing nations where women die suffering. Prolapse with apical descent and multifocal site prolapse is primarily a disease of peri or postmenopausal women. However younger patient population occurs in developing nations where the sociocultural risk factors such as early marriage, multiple births at short intervals and heavy physical labour in the perinatal periods can never be overcome. This case substantiates these observations of aforementioned studies where cervical and perineal damage during vaginal delivery as the major risk factor for prolapse. Retroversion of the uterus is yet another risk factor for prolapse. Cervical elongation is either the cause or effect of prolapse and its identification before reconstructive surgery is paramount as uterine suspension in the face of cervical elongation is contraindicated. Identification of specific support defect of the posterior fornix and uterine cervix is essential for developing innovative techniques and refining the established pelvic reconstructive surgery. Extensive public health education on proper antenatal care, supervised hospital deliveries and other preventive and corrective measures is the need of the hour. The various features reviewed in this article would provide a better understanding of the anatomical imperfections for the advancement of suitable procedures for pelvic reconstructive surgeons and urogynaecologists.

REFERENCES

[1] Onowhakpor EA, Omo-Aghoja LO, Akani CI, Feyi-Waboso P. Prevalence and determinants of utero-vaginal prolapse in southern Nigeria. *Niger Med J*. 2009;50:29-32.

[2] Wilson D, Herbison P. Conservative management of incontinence. *Curr Opin Obstet Gynecol*. 1995;7:386– 92.

[3] Okonkwo JEN, Obiechina NJA, Obionu N. Incidence of pelvic organ prolapse in nigerian women. *J of National Medical association*. 2003;95(2):132-36.

[4] Strohbehn K, Jakary JA, Delancy JOL. Pelvic organ prolapse in young women. *Obstet Gynecol*. 1997;90:3-6

[5] Jabs CFI, Monga A, Stanton SL, Child AH. Stress incontinence and pelvic organ prolapse in women with Marfan syndrome. *Int Urogynecol J*. 1999;10:S1 –6.

[6] Carley ME, Schaffer J. Urinary incontinence and pelvic organ prolapse in women with Marfan and Ehlers Danlos syndrome. *Int Urogynecol J*. 1999;10:65.

[7] Chiaffarino F, Chatenoud L, Dindelli M, et al. Reproductive factors, family history, occupation and risk of urogenital prolapse. *Eur J Obstet Gynecol Reprod Biol*. 1999;82:63–67.

[8] Dietz HP, Hansell NK, Grace ME, Eldridge AM, Clarke B, Martin NG. Bladder neck mobility is a heritable trait. BJOG: *An International Journal of Obstetrics & Gynaecology*. 2005;112(3):334-39.

[9] Duong TH, Korn AP. A comparison of urinary incontinence among African American, Asian, Hispanic women. *Am J of Obstet Gynecol*. 2001;184:1083-86.

[10] Nichols DH, Randall CL. Vaginal Surgery. 4th ed. Baltimore: Williams & Wilkins; 1996.

[11] White GR. A radical cure by suturing lateral sulci of vagina to white line of pelvic fascia. *JAMA*. 1909;21:1707–10.

[12] Richardson AC, Lyon JB, Williams NL. A new look at pelvic relaxation. *Am J Obstet Gynecol*. 1976;126:568–73.

[13] Miklos JR, Moore RD, Kohli N. Laparoscopic surgery for pelvic support defects. *Curr Opin Obstet Gynecol*. 2002;14(4):387-95.

[14] Berker B, Sahmohamedy B, Saberi N, Nezat C. "SLS Prevention and Management of Laproendoscopic Surgical Complication": Laparoscopic Pelvic Reconstructive surgery. 3rd Edition. Society of Laproendoscopic Surgeons. Miami, Florida-35143-4825. USA.2010.

[15] Fenner DE, Hsu Y, Morgan DM. Anterior vaginal wall prolapse: The challenge of cystocele repair. *OBG Management*. 2004;16(5):16-32.
[16] Biller DH, Davila GW. Vaginal vault prolapse: Identification and surgical options. *Cleve Clin JM*. 2005;72(Suppl 4):S20-27.
[17] DeLancey JOL. Anatomic aspects of vaginal eversion after hysterectomy. *Am J Obstet Gynecol.* 1992;166:1717–28.
[18] DeLancey JOL. Fascial and muscular abnormalities in women with urethral hypermobility and anterior vaginal wall prolapse. *Am J Obstet Gynecol*. 2002;187:93–98.
[19] Ravindran TKS, Savitri R, Bhavani A. Women's experience of utero-vaginal prolapse; A qualitative study from Tamil Nadu. Reproductive health matters safe motherhood initiative. *Critical issue*. 1999; 166-17.
[20] Radl C, Rajwar R, Arja RA. Uterine prolapse prevention in Eastern Nepal: the perspectives of women and health care professionals. *International Journal of Women's Health*. 2012;4:373-82.
[21] Bump RC, Mattiasson A, Bo K, Brubaker LP, DeLancey JO, Klarskov P, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. *Am J Obstet Gynecol.* 1996;175:10–17. [Pub Med: 8694033]
[22] Rogers, RM.; Richardson, AC. Clinical evaluation of pelvic support defects with anatomic correlations. In: Bent AE, Ostergard DR, Cundiff GW, Swift SE, editors. Ostergard's Urogynecology and Pelvic Floor Dysfunction. 2003.
[23] Berger MB, Ramanath R, Guire KE, De Lancey JOL. Is cervical elongation associated with pelvic organ prolapse? *International Urogynecol J.* 2012;23(8):1095–103.
[24] Walters MD. Uterovaginal prolapse in a woman desiring uterine preservation. *Int Urogynecol J Pelvic Floor Dysfunct*. 2008;19:1465-70. [Pub Med: 18688561]
[25] Zucchi A, Lazzeri M, Porena M, Mearini L, Costantini E. Uterus preservation in pelvic organ prolapse surgery. *Nat Rev Urol*. 2010;7:626–33. [Pub Med: 21068763]
[26] Karram, MM. Vaginal hysterectomy. In: Baggish, MS.; Karram, MM. editors. Atlas of pelvic anatomy and gynecologic surgery. St. Louis, MO: Elsevier Saunders; 2011.
[27] Ibeanu OA, Chesson RR, Sandquist D, Perez J, Santiago K, Nolan TE. Hypertrophic cervical elongation: clinical and histological correlations. *Int Urogynecol J Pelvic of Floor Dysfunct*. 2010;21:995–1000.
[28] Egbase PE, Al-Sharhan M, Grudzinskas JG. Influence of position and length of uterus on implantation and clinical pregnancy rates in IVF and embryo transfer treatment cycles. *Human Reproduction*. 2000;15(9):1943-46.
[29] Shaw RW, Soutter WP, Stanton SL Eds (2003) Gynaecology. Third Edition Churchill Livingstone, Philadelphia.
[30] Fielding WL, Lee SY, Friedman EA. Continued pregnancy after failed first trimester abortion. *Obstet Gynecol*. 1978;52(1):56-58.
[31] Donald I. Abortion and Intrauterine death. Practical obstetric problems. Chapter-2, 5th edition: pp 29-66.BI Publications Pvt-Ltd. New Delhi.1998.
[32] Zimmerman CW. Pelvic organ prolapse. Basic principles. In Rock JA, Jones III HW. Eds.10th Edition .Lippincott Williams & Wilkins. 2009.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Anatomy, Sikkim Manipal Institute of Medical Sciences, Gangtok East Sikkim, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Kalpana Chhetri,
Assistant Professor, Department of Anatomy, SMIMS, 5th Mile, Tadong, East Sikkim-737102, India.
E-mail: drkalp7@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **May 22, 2015**
Date of Peer Review: **Jun 16, 2015**
Date of Acceptance: **Aug 07, 2015**
Date of Publishing: **Oct 01, 2015**