e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(11); 1932-1937

Original Research Article

Nubbin Testes A Case Series of 16 Cases-Histological Evaluation of Nubbin Testes with Clinical Correlation

Fakeha Firdous[1], Zu Afshan Sultana[2], Anjani M.[3], Md. Saad Hussain[4], G. J. Vani Padmaja[5]

[1]Associate Professor, Pathology, Government Medical College, Kamareddy
[2]Assistant Professor, Pathology, Niloufer Hospital, Hyderabad
[3]Associate Professor, Pathology, Government Medical College, Jangaon
[4]Medical Intern, Ayan Medical College, Hyderabad
[5]Professor, Pathology, Niloufer Hospital, Hyderabad

Received: 01-09-2025 / Revised: 16-10-2025 / Accepted: 08-11-2025
Corresponding Author: Dr. Fakeha Firdous
Conflict of interest: Nil.

Abstract:

Introduction: "Nubbin" means -a small lump/ residual part/ stunted piece No viable testicular tissue can be grossly identified in a case of impalpable testis. Most important cause of impalpable testis.

Materials and Methods: An observational cross-sectional study was done at a tertiary care hospital in Telangana for biopsies sent as Nubbin testis during the period July 2021 to July, 2023. All biopsies which were sent as Nubbin testis were included in the study.

Results: A total of 16 cases were evaluated in the study period of two years. The age group of these cases ranged from 8 months to 13 years. Histopathological examination of the biopsies revealed fibro collagenous tissue in all the 16 cases, vas deferens in 11 cases, epididymis in 6 cases, hemosiderin laden macrophages in 5 cases and germ cells in one case. Seminiferous tubules remnant, Sertoli cells, calcification, ectopic adrenocortical rests were not identified in any of the cases.

Conclusion: Testicular regression syndrome/vanishing testes is a condition where there is disappearance /atrophy of an initially normal testis. The most characteristic histological features are presence of fibrovascular tissue along with hemosiderin laden macrophages and dystrophic calcification. Presence of viable germ cells and seminiferous tubules is very rare.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

"Nubbin" means -a small lump/ residual part/ stunted piece. No viable testicular tissue can be grossly identified in a case of impalpable testis. Most important cause of impalpable testis

Testicular Nubbin – Criteria

- Impalpable testis during anesthetic inguinal examination
- Blind ending spermatic vessels identified within the retroperitoneum
- Spermatic vessels & vas deferens exiting closed internal inguinal canal

0.8-2% of children at 1 yr age: Persistent undescended testes

Non palpable testis: 20-35%. Causes of non-palpable testis

Non-viable[nubbin]

- Extra abdominal testis
- Intra-abdominal testis

Left sided predominance: 60-72.5%, Compensatory hypertrophy of contralateral testis

Pathophysiology

- Incompletely descended testis - prone to torsion
- Early descent of left testis
- Kinking of left testicular vein
- Disturbances in endothelial development
- Early loss of both testes - genital ambiguity.

Risk of malignancy

- Intra-abdominal testis
- External genital abnormality
- Chromosomal anomaly

Aim: To analyze cases of Nubbin testis based on histopathological features and its correlation with clinical features at a tertiary care centre.

Objectives

- To analyze and describe histopathological features of biopsies sent as Nubbin testis
- To correlate the histopathological features with clinical features: age, site, phenotype, genotype & associated anomalies

Materials and Methods

Observational, cross-sectional study carried out in a Tertiary care hospital, Telangana for a duration of 2 years, July 2021 to July, 2023.

Inclusion Criteria

- Age group 0 days to 16 years.
- Testicular biopsies clinically diagnosed as Nubbin testis.
- Testicular biopsies from undescended testis.

Exclusion Criteria

- Testicular torsion.
- Solid tumours of the testis.

Clinical details like - Age, Site of testis, Laterality (right/left), Opposite testis – Present/ Absent, location, Presence of any associated anomalies, Phenotypical appearance, Sibling history were evaluated.

Intraoperative findings, Gross examination of the biopsies were recorded. Entire tissue was processed and H & E staining in Formalin embedded paraffin sections done.

Histopathological evaluation [to look for]

- Fibrosis.
- Calcification.
- Hemosiderin pigment.
- Semeniferous tubules.
- Paratesticular – Epididymis, Vas deferens.
- Germ cells.
- Ectopic adrenocortical rests.

Results

Table 1:

(16 cases)	
Age	8 months to 13 years.
Laterality	Left(14) >> Right (2)
Site	Undescended - 13 Descended - 3
Phenotype	Male – 16
Contralateral testis	Hypertrophy - 8
Associated anomalies	Micropenis - 1
Sibling history	Insignificant
Karyotyping	46xy[all cases]
Gross features	Membranous bits 2 cm – 8cm

Table 2:

Histopathologic findings	**No. of cases (16)**	**Percentage (%)**
Fibrosis	16	100
Calcification	2	12.5
Hemosiderin pigment	4	25
Semeniferous tubules	O	0
Paratesticular tissue: Epididymis	8	50
Vas deferens	11	68.75
Germ cells (IHC –Oct3/4)	? 1	?6.25
Ectopic adrenocortical rests	?1	?6.25

Table 3:

Serial no.	**Age**	**Laterality**	**location**	**Others**
1	8 years	Left	Undescended	
2	10 years	Right	Undescended	
3	3 years	Left	Scrotal	
4	9 months	Left	Scrotal	
5	10 months	Left	Undescended	
6	8 years	Left	Undescended	
7	1 year 3 months	Left	Undescended	
8	13 years	Right	Undescended	
9	10 years	Left	Undescended	

10	4 years	Left	Undescended	
11	10 years	Left	Undescended	Micropenis
12	1year 6 months	Left	Undescended	
13	8 years	Left	Scrotal	
14	1year 6 months	Left	Undescended	
15	3 years	Left	Undescended	?germ cells
16	1 year	Left	Undescended	

Figure 1: Gross of nubbin testis

Figure 2: Gross of Nubbin testis

Figure 3: Gross of Nubbin testis

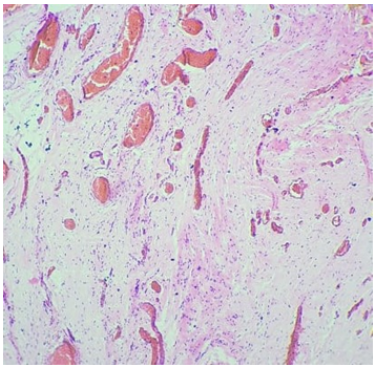

Figure 4: HPE-Fibrocollagenous tissue

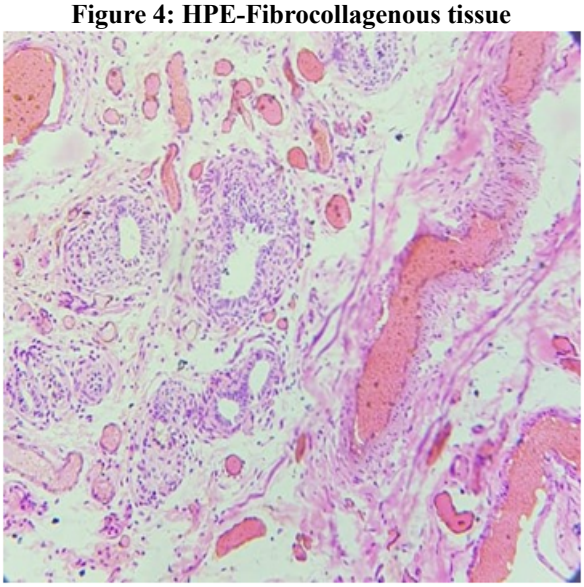

Figure 5: HPE Epididymis

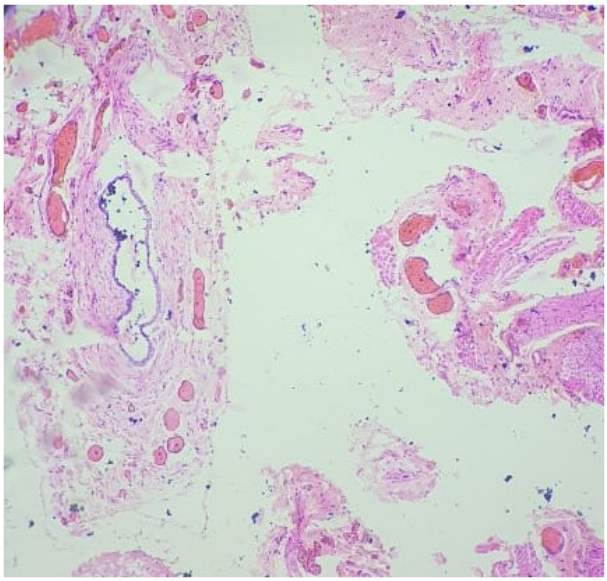

Figure 6: HPE Vas Deferens

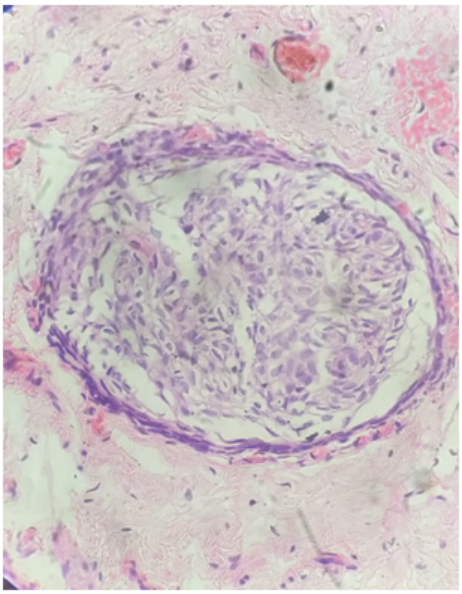

Figure 7: HPE-?Adrenocortical rests in Nubbin Testis

Discussion

The term Vanished testes coined by Abeyarafne et al. Testicular regression syndrome/vanishing testes Disappearance /atrophy of an initially normal testes Torsion or infarction in intrauterine life/perinatal life - Absence of testes in a genotypically normal male Prevalence <5% of cryptorchid cases

Cryptorchidism: 3% of full-term neonates - incompletely descended testes at birth.[5]Testes not palpable: 10-20% of cryptorchid cases TRS accounts for 35-60%. [6-10]

Causes of Non palpable testes -presence of testes

- intra-abdominal site
- inguinal
- intra-abdominal vanishing testes.[11,12]

Vanishing testes is more common than testicular agenesis in patients with non-palpable testes.'[13]

Presence of cord structures-evidence of presence of testes (intrauterine life) No endocrinopathy.

Lou and coworkers - 64% had cryptorchidism [16], In our study 81.25% cases have cryptorchidism.

Testicular Regression Syndrome is associated with:Genetic abnormality: microdeletion of Y chromosome Boys: persistent mullerian duct structures.[23] Sporadic - In majority of cases. No family history.

Possible genetic basis [25,26] may be considered:

- Chromosomal abnormalities in siblings
- Mental retardation
- Occurrence in several members of same family
- Male phenotype >>> Female phenotype
- Normal male with unilateral non palpable testes
- Phenotypic male with micropenis
- Extent & timing of intrauterine accident in relation to sexual development. [3,28,29]
- Characteristic histological features

1. Fibrovascular tissue
2. Hemosiderin laden macrophages
3. Dystrophic calcification
4. Residual testicular tubules [seminiferous tubules] Rare (< 10%) of the cases.
5. Viable germ cells (0-16%)) & Ectopic adrenocortical rests - risk of malignant transformation.

- Risk of malignancy in undescended testis is 8-10 %
- In nubbin-0-1.1%
- Risk of malignancy: surgical excision

Table 4:

Smith et al	Cendron et al	Present study
• Dense fibrovascular tissue • Absence of testicular elements. [20]	• Histological evaluation of 25 vanishing testes specimens • No identifiable testicular elements [10]	• Vas deferens in 8 cases • Epidydimis in 6 cases • Semeniferous tubules – nil

Conclusion

Nubbin testes - ischemic and atrophic due to Vascular insult. More common in undescended testes.

Development of external genitalia depends on chronology of gonadal injury. Presence of remnant cord structures. Identifying germ cells & Ectopic adrenal cortical rests. Confirmation using IHC [Prognostic value, Risk of malignancy].

Take home message: Create awareness - entity of Nubbin testis. Significance of Histopathological examination and looking for Germ cells & adrenocortical rests.

Role of HPE, IHC, karyo typing. Plan surgical intervention & follow up.

The age of diagnosis is variable,so early physical examination of testis, will be helpful for the patient to detect nubbin testis and plan the treatment accordingly.

References

1. Natural history of testicular regression syndrome and consequences for clinical management. Hegarty PK, Mushtaq I, Sebire NJ. J Pediatr Urol. 2007; 3:206–208.
2. Diamond DA, Caldamone AA. The value of laparoscopy for a 106 impalpable testis relative to clinical presentation. J Urol. 1992; 148:632–634.
3. Spires SE, Woolums CS, Pulito AR, Spires SM. Testicular regression syndrome: a clinical and pathologic study of 11 cases. Arch Pathol Lab Med. 2000; 124:694–698.
4. Mouriquand PD. Undescended testes in children: the paediatric urologist's point of view. Eur J Endocrinol. 2008; 159:83–86.
5. Rozanski TA, Wojno KJ, Bloom DA. The remnant orchiectomy. J Urol. 1996; 155:712–714.
6. Storm D, Redden T, Aguiar M, Wilkerson M, Jordan G, Sumfest J. Histologic evaluation of the testicular remnant associated with the vanishing testes syndrome is surgical management necessary. Urology. 2007; 70: 1204–1206.
7. Koyama T, Nonomura K, Ameda K, Kakizaki H, Matsugase Y, Shinno Y, Kanno T, Yamashita T, Murakumo M, Koyanagi T. Laparoscopic evaluation and the management of the nonpalpable testis. Diagn Ther Endosc. 1997; 4:69–74.
8. Elder JS. Laparoscopy for impalpable testes: significance of the patent processus vaginalis. J Urol. 1994; 152:776–778.
9. Cendron M, Schned AR, Ellsworth PI. Histological evaluation of the testicular nubbin in the vanishing testis syndrome. J Urol. 1998; 160:1161–1162.
10. Redman F. Impalpable testis: observations based on 208 consecutive operations for undescended testis. J Urol. 1980; 124:379–381.
11. Smolko MJ, Kaplan GW, Brock WA. Location and fate of the nonpalpable testis in children. J Urol. 1983; 129:1204–1206.
12. Merry C, Sweeney B, Puri P. The vanishing testis: anatomical and histological findings. Eur Urol. 1997; 31:65–67.
13. Lou CC, Lin JN, Tung TC, Wang KL. Anatomical findings of the vanishing testis. Changgeng YT Xue ZA Zhi. 1994; 17:121–124.
14. Bar-Maor JA, Groisman G, Lam M. Antenatal torsion of the testes, a cause of vanishing testis syndrome. Pediatr Surg Int. 1993; 8:236–238.
15. Wright JE. The atrophic testicular remnant. Pediatr Surg Int. 1986; 1:229–231.
16. Papparella A, Zamparelli M, Cobellis G, Amici G, Saggiomo G, Parmeggiani P, Fioretti GP. Laparoscopy for nonpalpable testis: is inguinal exploration always necessary when the cord structures exit the inguinal ring. Pediatr Endosu Innov Tech. 1999; 3:29–33.
17. Smith NM, Byard RW, Bourne AJ. Testicular regression syndrome-a pathological study of 77 cases. Histopathology. 1991; 19:269–272.
18. Calogero AE, Garofalo MR, Barone N, De Palma A, Vicari E, Romeo R, Tumino S, D'Agata R. Spontaneous regression over time of the germinal epithelium in a Y chromosome-microdeleted patient: case report. Hum Reprod. 2001; 16:1845–1848.
19. Rattanachaiyanont M, Phophong P, Techatraisak K, Charoenpanich P, Jitpraphai P. Embryonic testicular regression syndrome: a case report. Med Assoc Thai. 1999; 82:506–510.
20. Grouchy J, de, Gompel A, Salomon-Bernard Y, Kuttenn F, Yaneva H, Paniel JB, Le Merrer M, Roubin M, Turleau C. Embryonic testicular regression syndrome and severe mental retardation in sibs. Ann Genet. 1985; 28:154–160.
21. Marcantonio SM, Fechner PY, Migeon CJ, Perlman EJ, Berkovitz GD. Embryonic testicular regression sequence: a part of the clinical spectrum of 46, XY gonadal dysgenesis. Am J Med Genet. 1994; 49:1–5.
22. Josso N, Briard ML. Embryonic testicular regression syndrome: variable phenotypic expression in siblings. J Pediatr. 1980; 97:200–204.
23. Coulam CB. Testicular regression syndrome. Obstet Gynecol. 1979; 53:44–49.
24. Naffah J. Familial testicular regression syndrome. Bull Acad Natl Med. 1989; 173:709–714.
25.