

## Histopathological study of 40 Orchidectomy specimens in a tertiary care hospital in North Telangana

S. Srikanth

Associate Professor, Dept. of Pathology, Prathima Institute of Medical Sciences, Karimnagar, Telangana

Email: drshastrysri@yahoo.com

### Abstract

**Introduction:** Testicular tumours are the cause of about 1% of all cancer deaths. They are more frequent in white male population but are less common in Africans and Asians. They have trimodal age distribution – a peak during infancy, another during late adolescence and early adulthood and a third peak after 60 years of age.

**Materials and Method:** The present study is a retrospective and prospective study. A total of 40 orchidectomy specimens were received during a period of three years. Clinical and radiological history was available for all cases and histopathological diagnosis was given after routine processing and staining.

**Results:** Out of 40 cases, seminoma was the commonest neoplastic lesion, followed by maturation arrest with atrophic changes and torsion of testis. Left testis was involved more compared to right.

**Conclusion:** Histopathological diagnosis is very essential and important although new techniques in imaging and tumour marker assay the diagnosis of testicular lesions are available.

**Keywords:** Testis, Seminoma, Torsion, Orchidectomy

### Introduction

Testis is a male gonad, it is homologous with the ovary of the female genital system and it is a unique and important organ of male reproductive system.<sup>(1)</sup> Testicles are a very delicate part of male body. Testis is a paired oval organ that lies within scrotum suspended by spermatic cord.<sup>(2)</sup> There are various testicular lesions, ranging from paediatric to adult age groups. They usually present with scrotal swelling, pain in scrotum and mass per abdomen. Testicular neoplasms span an amazing gamut of anatomic types. They are divided into two major categories; germ cell tumours and non germinal tumours derived from stroma or sex cord. Approximately, 95% arise from germ cells. Most of these germinal tumours are highly aggressive cancers that are capable of rapid, wide dissemination, although with current therapy, most can be cured. Non-germinal tumours in contrast, are generally benign, but some elaborate steroids, leading to interesting endocrinologic syndromes.

Testicular carcinoma follows a reverse pattern to most cancers with decreasing incidence rate with increasing age. Cryptorchidism, Klinefelter syndrome and strong family are the predisposing risk factors in development of testicular germ cell tumors.

Significant advances in the understanding of diseases, various investigative modalities per say, Routine tests, X-ray, Ultrasound, CT scan, Intravenous urography, tumour marker assay and finally histopathological examination is of useful guide. Despite new techniques in imaging and tumor marker assay the diagnosis of testicular lesions is primarily dependent upon histopathological examination.

Clinical data, operative findings and gross features of lesions may provide important and at times decisive

diagnostic clues. The present study is undertaken to study the diverse histopathological patterns of testicular lesions and thus offering a specific diagnosis which is of paramount clinical significance.

### Materials and Method

The present study is a retrospective and prospective study done on 40 orchidectomy specimens which are received in Department of Pathology. Clinical details and investigations were recorded. Each specimen was subjected to gross examination and histopathological features were noted on H & E stained slides of all specimens. Gross examination was done and findings like right or left side, condition of the scrotal skin and tunica albuginea, consistency, size of the tumour, cut surface appearance, any areas of necrosis or hemorrhage, colour, adjacent testicular tissue, epididymis and spermatic cord were observed carefully and also examined for any lymph nodes.

### Results

Out of the total 40 orchidectomy specimens, 24 cases involved left testis while 16 involved right side. Seminoma was the most common neoplastic lesion in our study constituting 10 cases, followed by embryonal cell carcinoma, mixed germ cell tumour and sertoli cell only syndrome. Among the non-neoplastic lesions maturation arrest with atrophic changes constituted highest number of cases (12 cases) followed by pyocele, torsion and epididymo orchitis. (Table 1)

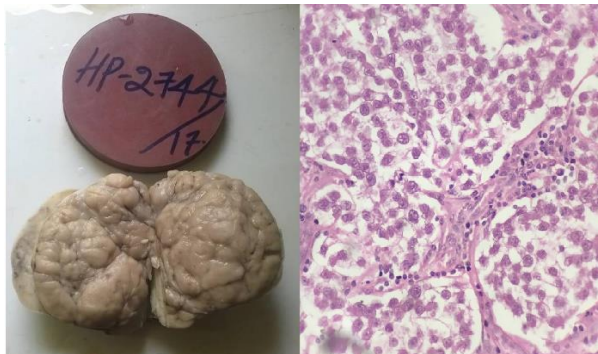
Among the neoplastic lesions youngest age group was 17 years and oldest was 54. Among the non neoplastic lesions youngest was 19 years and oldest was 62. Age group between 31- 40 years constituted highest number of cases.(Table 2)

**Table 1: Showing different testicular lesions**

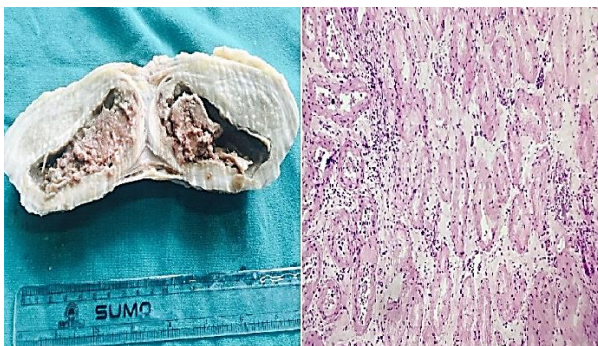
Sl No	Lesion	Number of cases
1	Seminoma	10
2	Embryonal cell carcinoma	02
3	Mixed germ cell tumour	02
4	Sertoli cell only syndrome	01
5	Maturation arrest with atrophic changes	12
6	Pyocele	06
7	Torsion	06
8	Epididymo orchitis	01
Total		40

**Table 2: Showing different age groups**

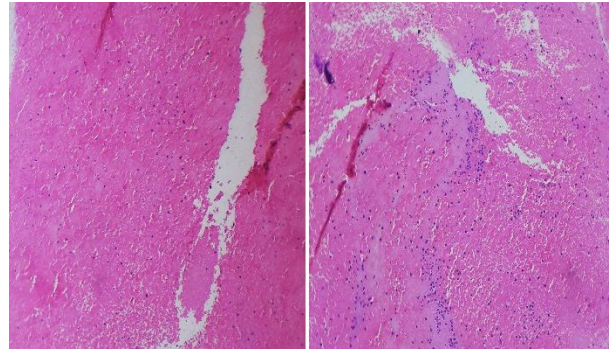
Age group	No of cases
1-10 yrs	00
11-20 yrs	04
21-30 yrs	06
31- 40 yrs	18
41- 50 yrs	05
51- 60 yrs	03
61-70 yrs	04
Total	40



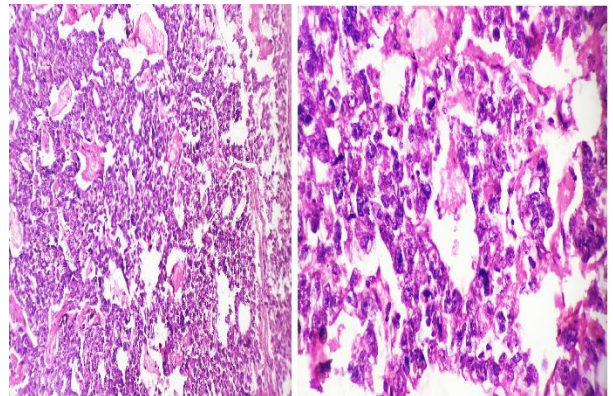
**Fig. 1: Gross showing tumour tissue which is enlarged compared to normal. Microscopy showing tumour cells arranged in monotonous population with delicate fibrous septa in between infiltrated by lymphocytes.(H&E, x 40)**



**Fig. 2: Gross showing testis which appears to be atrophied. Microscopy showing atrophied tubules with thickened basement membrane.(H&E, x40)**



**Fig. 3: Microscopy showing areas of hemorrhage and necrosis.(H)**



**Fig. 4: Section showing polygonal to ovoid tumour cells arranged in diffuse pattern**

### Discussion

Testicular neoplasms span an amazing gamut of anatomic types. They are divided into two major categories; germ cell tumours and non-germinal tumours derived from stroma or sex cord. Approximately 95% arise from germ cells. Most of these germinal tumours are highly aggressive cancers that are capable of rapid, wide dissemination, although with current therapy, most can be cured. Non-germinal tumours, in contrast, are generally benign, but some elaborate steroids, leading to interesting endocrinologic syndromes. Approximately 8000 cases of testicular tumours are diagnosed per year in the United States, resulting in about 400 deaths per year. For unexplained reasons, there is a worldwide increase in the incidence of these tumours. In the 15 to 34 year age group, when these neoplasms have a peak incidence, they constitute the most common tumour of men and cause approximately 10% of all cancer deaths.<sup>(3,4)</sup>

Testicular germ cell tumours may be divided into two categories on the basis of whether they are composed of a single histologic pattern or more than one. Tumours with a single histologic pattern constitute about 40% of all testicular neoplasms. In approximately 60% of the tumours, there is a admixture of two or more of the histologic patterns. Most tumours in this group originate from intratubular germ cell neoplasia (ITGCN). Untreated intratubular germ cell neoplasia

progresses to invasive germ cell tumour in approximately 50% of cases over 5 years of follow up. Thus its significance is similar to carcinoma in situ in other organs. If ITGCN is identified, it is treated by low dose radiotherapy, which destroys the germ cells yet maintains the androgen production of the Leydig cells.

ITGCN is widely believed to be the precursor of most invasive germ cell tumours. Although Wilms in 1896 described the presence of atypical intratubular cells adjacent to invasive carcinoma, it was Skakkebaek, who in 1972, first reported that the atypical germ cells within the seminiferous tubules actually represented the precursor of the invasive testicular germ cell neoplasm. This in-situ phase of germ cell tumour occurs in 0.5 -1% of infertile patients with severe oligospermia, 2-8% of those with cryptorchidism and 5% of patients with a history of testicular cancer. There is a 15-20% risk for development of ITGCN in the contralateral testis in patients with a history of undescended testis and testicular carcinoma. Patients with dysgenetic gonads and testicular feminization syndrome also have an increased incidence of ITGCN. Approximately 50% of patients with ITGCN will progress to invasive carcinoma within 5 years, and 90% or more if orchidectomy is not performed. Most patients with ITGCN develop seminoma and in fact, the terms seminoma in situ and carcinoma in situ have been used interchangeably for ITGCN.

Testicular swelling constituted > 75% in the present study and it was 81.5% and 60% in studies by W. Duncan et al<sup>(5)</sup> and Deotra A<sup>(6)</sup> et al respectively and which was correlating with our study. Relative frequency of testicular torsion is 22% in present study and which were 10.1%, 13.1% and 48% in studies done by Srinivasn A et al,<sup>(7)</sup> Rizvi SA et al<sup>(8)</sup> and Rampaul M S.<sup>(9)</sup>

Seminomas are the most common type of germinal tumour and the type most likely to produce a uniform population of cells. Seminomas produce bulky masses, sometimes 10 times the size of the normal testis (Fig. 1). The typical seminoma has a homogeneous, grey white, lobulated cut surface, usually devoid of hemorrhage or necrosis. In more than half of cases the entire testis is replaced. Microscopically, the typical seminoma presents sheets of uniform cells divided into poorly demarcated lobules by delicate fibrous septa (Fig. 1). A discrete or diffuse granulomatous reaction, with or without multinucleate giant cells, often occurs in seminoma. In metastatic disease, the granulomatous inflammation may predominate, making diagnosis extremely difficult. Immunohistochemistry with PLAP is very helpful in this situation. Scattered syncytiotrophoblastic giant cells are present in 10-20% of cases. Other forms of giant cells that may be present in seminoma include mulberry cells and the previously mentioned Langhans – type giant cells associated with

granulomata. Extensive calcification and ossification may also occur in seminoma.

Atrophic testis was the most common lesion which was closely correlating with the study done by Hemavathi Reddy et al,<sup>(10)</sup> Srinivasn A et al,<sup>(7)</sup> and Rizvi SA et al<sup>(8)</sup> respectively. Atrophy of the testis may be caused by a number of causes like old age, inflammation, cryptorchidism, malnutrition, hypopituitarism, prolonged administration of female hormones and irradiation. Histopathologically shows atrophied tubules with thickened basement membrane (Fig. 2).

Twisting of the spermatic cord may cut off the venous drainage and the arterial supply to the testis. Usually, however, the thick walled arteries remain patent, so intense vascular engorgement and venous infarction follow. There are two types of testicular torsion; neonatal torsion occurs either in utero or shortly after birth. It lacks any associated anatomic defect to account for its occurrence. Adult torsion is typically seen in adolescence, presenting as sudden onset of testicular pain. In contrast to neonatal torsion, adult torsion results from a bilateral anatomic defect in which the testis has increased mobility, giving rise to what is termed the bell-clapper abnormality. Histopathologically shows only areas of hemorrhage and focal necrosis (Fig. 3)

Embryonal cell carcinoma occur mostly in the 20-30 year age group. These tumours are more aggressive than seminomas. Grossly the tumour is smaller than seminoma and usually does not replace the entire testis. On cut surfaces, the mass is often variegated, poorly demarcated at the margins, and punctuated by foci of hemorrhage or necrosis. Microscopically, the cells grow in alveolar or tubular patterns, sometimes with papillary convolutions. (Fig. 4) Embryonal carcinoma lack the well-formed glands with basally situated nuclei and apical cytoplasm seen in teratomas. More undifferentiated lesions may present sheets of cells. The neoplastic cells have an epithelial appearance and are large and anaplastic with hyperchromatic nuclei having prominent nucleoli. In contrast to seminoma, the cell borders are usually indistinct, and there is considerable variation in cell and nuclear size and shape. Mitotic figures and tumour giant cells are frequent. Within this background, syncytial cells containing HCG, cells containing AFP, or both may be detected by immunoperoxidase techniques. Most authorities allow for focal AFP positivity within an embryonal carcinoma without classifying the tumour as a mixed tumour. However some purists designate any AFP positivity in an embryonal carcinoma, even if unaccompanied by yolk sac differentiation on the H&E stained section, as focal yolk sac tumour in a mixed tumour.

During foetal development, the testis descends from the posterior abdominal wall and by birth reaches the scrotum. In about 5% of the cases, the descent of one or both testis is arrested anywhere along the

pathway of the descent leading to undescended testis and cryptorchidism.

### **Conclusion**

Our study concluded that non neoplastic lesions of testis are commoner than neoplastic lesions. The incidence of Non-neoplastic lesions of the testis are most common in the 3<sup>rd</sup> decade while malignancy was common in 2<sup>nd</sup> to 3<sup>rd</sup> decade of life according to our study. Out of all non-neoplastic lesions, atrophic testis with maturation arrest was the commonest finding. Germ cell tumors formed the bulk of neoplastic lesions. Amongst them Seminomas followed by Mixed germ cell tumours were the most common ones. Histopathological diagnosis is compulsory to diagnose testicular lesions inspite of new diagnostic modalities.

### **References**

1. Chaurasia BD. Male Reproductive System. In: Human Anatomy, Volume 2. 6<sup>th</sup> ed. New Delhi: CBS Publishers and Distributors; 2013.p.266 -296.
2. Juan R. Male reproductive system. In: Rosai and Ackerman's Surgical Pathology, Volume 1. 10<sup>th</sup> ed. St. Louis: Elsevier; 2011.p.1334-1374.
3. Sabiston. Testis. In: Text book of surgery, volume II. Eighteenth ed. Saunders/ Elsevier Publishers;2007.p.2280-2285.
4. Bergstorm et al. Testicular cancer in nine European countries. *Int J Cancer* 1996;59:33-38.
5. W. Duncan et al. Management of testicular seminoma. *British Journal of cancer*; 1987;55:443-448.
6. Deotra A, Mathur DR, Vyas MC et al. An 18 years study of testicular tumours in Jodhpur, western Rajasthan. *J Post Graduate Med* 1994;40(2):68-70.
7. Srinivasan A, Cinman N, Feber K, Gitlin J, Palmer L. History and physical examination findings predictive of testicular torsion: An attempt to promote clinical diagnosis by house staff. *Journal of Pediatric Urology*. 2011;7(4):470-474.
8. Rizvi SAA, Ahmad I, Siddiqui MA, Zaheer S, Ahmad K. Role of Color Doppler Ultrasonography in Evaluation of Scrotal Swellings. *Urology Journal* 2011;8(1):60-5.
9. Rampaul MS, Hosking SW. Testicular torsion: most delay occurs outside hospital. *Ann R Coll Surg Engl*. 1998 May;80(3):169-72.
10. Hemavathi Reddy et al. Histomorphological analysis of testicular lesions. *Indian Journal of Pathology and Oncology*, October- December 2016;3(4):558-563.