



Review Article

Segmental Testicular Infarction: A Clinical Dilemma

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Abstract

Segmental testicular infarction is an uncommon entity often diagnosed during scrotal imaging for acute scrotum. It is due to a partial ischemic process resulting in hyalinization and fibrosis of the infarcted area. Various factors have been implicated for this entity such as infection, trauma, hematological disorders and microangiopathy. It frequently presents as acute scrotum and sonographically simulates a small intra-testicular neoplasm. Hence it is of paramount importance to distinguish segmental testicular infarction from intra-testicular neoplasm. Although high resolution scrotal ultrasonography can identify such lesions, scrotal MR imaging can accurately differentiate segmental testicular infarction from testicular tumor, thus preventing unnecessary orchiectomy in most of the cases.

Keywords

Testis; Segmental Infarction; Ultrasonography; MRI; Orchiectomy

Introduction

The human testis receives its blood supply directly from the aorta. The testicular parenchyma receives approximately 9 ml of blood per gram of tissue per minute. The individual testicular arteries penetrate the tunica albuginea and travel within the septa that contain seminiferous tubules. Interruption of the main testicular artery leads to global testicular ischemia and similarly, involvement of the intra-testicular centrifugal arteries cause segmental testicular infarction.

Segmental testicular infarction is defined as any focal area of altered reflectivity, with or without focal enlargement with absent or diminished flow on color Doppler confirmed histology or on follow-up exclusion of lesion progression.

It is an extremely rare entity, mostly detected on scrotal imaging. These lesions are mostly wedge shaped with vertex at the testicular mediastinum. However, round lesions may also be encountered [1], mostly involving superior hemisphere of the testis. Many a times it is difficult sonographically to distinguish segmental testicular infarction from small intra-testicular germ cell neoplasia with negligible vascularity [2].

Segmental testicular infarction is detected in adults, mostly in their third decade, but also reported to occur in extremes of age such as in children [3] and elderly diabetic male. Very rarely it may

occur in neonates following birth trauma [4]. Segmental testicular infarction is a unilateral pathology but bilateral involvement is also reported to occur, which is extremely rare [5].

The exact etiology of segmental infarction of testis is not clear. Mostly reported to be idiopathic in origin [6]. Several authors have implicated various local and systemic pathology as possible etiological factors for the development of segmental infarction within the testis.

Acute epididymitis [7-9], spermatic cord torsion [10], inguinal trauma such as varicocele [11] and herniorrhaphy [12-14], cholesterol embolisation [15], protein S deficiency [16] and sickle cell trait [17] are all implicated in the pathogenesis of segmental testicular infarction. Of late diabetic microangiopathy [18] and human chorionic gonadotropin [19] are identified as possible etiological factors in the development of focal testicular infarction. The other etiological factors attributed to this pathology are vasculitis, fibroplasias of spermatic artery tunica media, polyarteritis nodosa, polycythemia, thromboangitis obliterans and strenuous exercise [6].

Pathophysiology

The arterial distribution within the testis are from centripetal arteries which when interrupted by any cause, predisposes to partial infarct, predominantly in the superior hemisphere of the testis.

Alteration in testicular blood supply can involve either arterial or venous flow, resulting in to ischemic or hemorrhagic infarct. Hemorrhagic infarct are rare and usually occurs secondary to inguinal hernia surgery which may block the venous outflow leading to venous congestion and hemorrhagic necrosis of testis [20]. Venous infarction may also occur in cases with severe epididymo-orchitis where local inflammatory edema occludes the venous drainage. Hypercoagulable state may also precipitate venous infarction [21].

Inflammation of adjacent epididymis may lead to obstruction of the testicular blood supply resulting in focal or global ischemia of the testis. Ledwidge [22] has described superior hemispheric infarction of the testis following torsion and distortion in a 22 years old boy with bell clapper deformity [22]. Similarly hematological disorders cause congestion and thrombosis of venous and arterial blood supply to the testis [16]. It is postulated that human chorionic gonadotrophins, stimulate leydig cells to release Prostaglandin denovo which induces intra-testicular artery to contract in frontal lower area and such obstruction for more than 12 hours induces focal necrosis [19].

Aquino [23] has observed specific histologic characteristics of the lesion corresponding to the age of the infarct. Presence of coagulative necrosis, RBC's, fibrin extravasation with mild or no thickening of the basement membrane of seminiferous tubule is considered indicative of acute infarct while presence of early maturation arrest in seminiferous tubule with thickening of basement membrane is considered as sub-acute infarct. A chronic infarct was defined to have foci of sclerosed seminiferous tubule with hyalinized interstitial fibrosis [23].

Kim HK [24] described the following histological features in 19 years old boy with segmental testicular infarction. They found well demarcated area of coagulation necrosis beneath the tunica albuginea,

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along with thrombosis of a muscular artery and several arterial branches. There was loss of tunica media resulting into aneurysm of the artery beneath the tunica along with neovascularisation of the adventitia adjacent to the aneurysm with adventitial inflammation [24].

Anomalies of the intra-testicular centripetal arteries or bifurcation of the testicular artery or presence of inconsistent anterior epididymal artery predisposes to development of segmental testicular infarction, particularly in the areas where collateral vessels are lacking, especially at the superior testicular pole. It is proposed that predominant vascular insult results in a rounded lesion in relation to venous obstruction which may further produce mass effect, whereas a wedge shaped lesion most commonly occurs due to arterial obstruction by thrombosis or microemboli [25].

Clinical Presentation

Acute scrotum is the most common clinical presentation. However, it may present with chronic orchalgia or testicular mass [26]. Physical examination findings are not specific or may even be normal except during acute presentation. A nodule or induration may be palpable over the testis on careful examination [6]. It can be clinically confused with testicular neoplasm in presence of firm nontender testicular mass [27]. Analyses of testicular tumor markers are routinely done to exclude testicular neoplasm in cases with intra-testicular pseudotumor detected on scrotal imaging.

Imaging

Although imaging is not conclusive, it may provide sufficient clue to plan for testis preserving modalities. An avascular intra-testicular lesion on sonography which may be round or wedge shaped is considered as segmental infarction of the testis. The gray scale ultrasound usually detect a round or triangular shaped, well defined, hypo echoic intra-testicular mass with minimal or no blood flow on color Doppler examination with heterogeneity and enlargement of epididymis [9]. After comparing imaging characteristic on gray scale, Colour Doppler and contrast enhanced images of 20 men with acute segmental testicular infarction, Bertolotto [28] has opined that detection of lobular morphologic characteristics together with presence of perilesional rim enhancement on contrast enhanced ultrasonography can predict segmental testicular infarct almost with certainty [28]. They were of the opinion that reduction in size of the lesion and changes in vascular features during follow-up leads to a firm diagnosis of Segmental Testicular Infarction.

Peritesticular rim enhancement is attributed to inflammatory changes around the infarcted areas or mass effect secondary to intralesional edema that displaces the surrounding testicular tissue [21].

Many a times, it is difficult to distinguish sonographically between segmental testicular infarction and intra-testicular tumor for which radical orchiectomy was performed irrespective of the pathology. With the advent of scrotal MRI it is now possible to accurately characterize segmental testicular infarction in doubtful situations and prevent unnecessary orchiectomy [29]. Echogenicity and discreteness of the lesion on sonography seems to reliably predict the age of the infarct [23].

The typical MRI features of segmental testicular infarction is, a lesion with low intensity central area and high signal intensity in the peripheral area suggesting ischemia [6]. A slight retraction of the tunica albuginea adjacent to the focal infarct is an additional imaging sign that helps to differentiate it from the neoplasm [1]. Bilagi [10]

has described characteristic ultrasonographic features for segmental testicular infarction and stated that they are not always wedge shaped, and reduced or absent vascularity is the key importance. In a systematic review of 24 patients with focal testicular infarct they found that 50% were of low reflectivity, 45.8% has mixed reflectivity and only in one patient the lesion was of high reflectivity on sonography. They emphasize that awareness of such sonographic features of focal testicular infarct will allow for conservative management avoiding unnecessary orchiectomy [10].

Patel and Patel has reported typical MRI features of intra-testicular germ cell neoplasia which shows more rapid enhancement and high peak enhancement compared to contralateral and surrounding ipsilateral normal testicular parenchyma. In contrast, areas of focal testicular ischemia show practically no enhancement on gadolinium enhanced MRI sequences. Knowledge of such imaging characteristics can accurately differentiate benign from intra-testicular neoplasm [30].

Utilizing multimodality modern imaging techniques Parenti [25] reviewed the accuracy of diagnosing Segmental Testicular Infarction amongst 798 patients, adopting certain clinico-radiologic criteria. They compared Color Doppler Ultrasound (CDUS), Contrast Enhanced Ultrasound (CUES) and MRI based on size, morphology, echogenicity, signal intensity, vascularity and pattern of contrast enhancement of the intra-testicular lesion. Color Doppler Ultrasound detected unisegmental or plurisegmental (43%) hypoechoic areas in all 14 patients with no sign of microcalcifications, poor vascularity and no mass effect or signs of infiltration of vascular structure or scrotal tunics.

With a follow up ranging from 20 to 200 days, CDUS could confirm progressive reduction in size of intra-testicular lesion. CEUS, which was performed in 5 patients, confirmed the abnormality detected in CDUS and found to be useful in monitoring patient with Segmental Testicular Infarction. In their study within 5 days of CDUS, the MRI revealed hyperintense lesion on T₁ weighted sequences indicating hemorrhagic infarction in six patients (43%), while remaining eight cases (57%), the lesion appeared isointense or weakly hypointense, while on T₂ weighted sequences, lesion displayed varying signal intensity. Contrast enhanced sequences showed rim enhancement in the periphery part of the lesion. After 2 months of follow up, the lesion appeared smaller in size and hypointense on both T₁ and T₂ weighted images. MRI is typically useful when ultrasound findings are atypical or suggestive of a solid lesion. Appearance of granulation tissue in response to ischemic process leads to rim enhancement on MRI [25]. Because of hyalinization and fibrosis within the infarcted lesion, loss of testicular substances may produce retraction of the tunica albuginea which can be better appreciated on MRI imaging [28].

Based on the principle of change of tissue stiffness in real time, Kantarci et al. recently has proposed Shear Wave elastography as one of the diagnostic modality to accurately predict segmental infarct within the testis assuming that testicular neoplasm are stiffer than the normal testicular parenchyma [31]. They further proposed that segmental ischemia causes increase its water content resulting in to swelling of the tissues which causes the area to appear soft on shear wave elastography.

On the basis of their imaging experiences and follow up, Parenti et al. suggested the following criteria to diagnose segmental testicular infarction in absence of testicular trauma and negative tumor markers. These includes presence of hypo/isoechoic lesion on USG, intra-testicular echostructural changes, with no microcalcification, or

mass effect, markedly reduced CDUS signal, presence of abnormal intra-testicular signal with no contrast enhancement on MRI. At follow up, absence of progression, reduction in size of the lesion with absent or reduced flow further confirms the diagnosis [25].

Treatment

Recent report by Hidalgo and colleague has stressed upon the role of excisional frozen biopsy for atypical intra-testicular sonographic findings to arrive at a definitive diagnosis in order to perform testis sparing surgery [32]. Most authors in the past have recommended surgical intervention in the form of orchiectomy for segmental testicular infarction due to difficulty in differentiating focal testicular infarct from small intra-testicular neoplasm. Liu et al. reported performing partial orchiectomy in a 24 year old male based on the sonographic evidence of segmental infarct which was subsequently confirmed on histology as segmental testicular hemorrhagic infarction (Figure 1) [33].

Appropriate evaluation and interpretation with sonography and MRI makes conservative management a feasible option in cases of segmental testicular infarction presenting as acute scrotum [29].

Conclusion

Segmental testicular infarction is an uncommon entity though not very rare which is often diagnosed on suspicion on scrotal imaging.

In case of suspicion, Color Doppler Ultrasound (CDUS), Contrast Enhanced Ultrasound (CUES) and MRI help to differentiate the lesion from testicular mass and other testicular pseudotumors.

Exact etiology of this condition is not known. However, it may be associated with local or systemic disorder. Accurate characterization of the lesion by Color Doppler Ultrasonography and Contrast enhanced ultrasonography helps to establish a diagnosis with certainty. Availability of the modern, advanced imaging modalities helps to arrive at a definitive diagnosis and avoid unnecessary orchiectomy. Rarely very few cases with unclear morphology or no conclusive MRI findings may require surgical intervention in the form of partial or total orchiectomy. Further, clinicians should be aware of the imaging characteristics of segmental testicular infarction on sonography which may be mistaken for intra-testicular tumor.

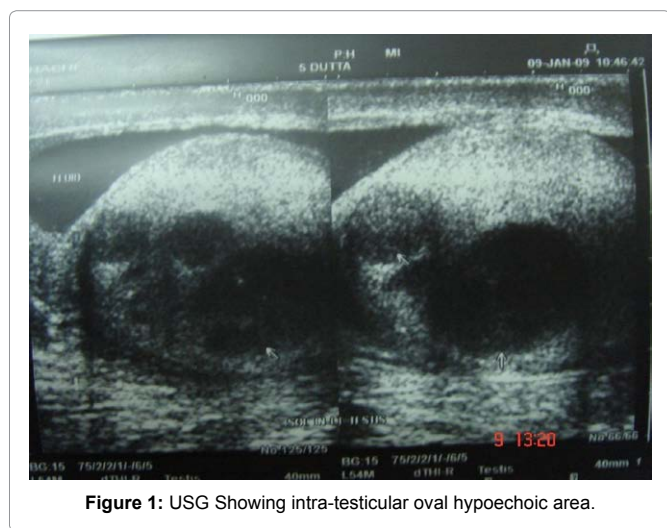


Figure 1: USG Showing intra-testicular oval hypoechoic area.

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