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Current Perspectives on Hematospermia: A Review

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ABSTRACT: Hematospermia is a disconcerting symptom that produces extreme anxiety in sexually active male patients. To understand the pathophysiology of hematospermia, the anatomy of the ejaculatory system and neurophysiology of emission and ejaculation is essential. Emission and ejaculation must be present for hematospermia to occur. Hematospermia may be the result of inflammation, infection, ductal obstruction or cysts, neoplasms, vascular abnormalities, and systemic or iatrogenic factors. Most patients promptly consult a urologist after an episode of hematospermia. History and physical examination are often unrevealing and the judicious use of imaging modalities, such as transrectal ultrasound, MRI, and rigid or flexible endoscopy, may be diagnostic. Unless the specific etiology is defined, most cases are managed expectantly. We review the etiology of hematospermia and an algorithm is provided for the diagnosis and management.

Key words: Accessory sex glands, blood, ejaculation, genital diseases, hemospermia, semen.

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Hemospermia or hematospermia is defined as the presence of fresh or altered blood in the ejaculate and may be derived from pathology involving the accessory sexual glands or their ducts, urethra, or bladder. It is a symptom related to sexual function and its presence is dependent on the processes of emission and ejaculation. Hematospermia may clinically present with infertility, hematuria, or lower urinary tract symptoms. Occasionally, it may be a sole manifestation of underlying urologic disease. This alarming symptom persuades patients to seek prompt medical evaluation with the concern of cancer, venereal disease, or potential threat to sexual function. The profound anxiety that hematospermia provokes in patients is supported by the acute onset of symptoms. Jones reported that 77.5% of men with hematospermia had experienced only one or two episodes prior to visiting a urologist (Jones, 1991). Although the incidence of hematospermia is unknown, a busy urologist may see more than one case per month (Ganabathi et al, 1992).

Historical Perspectives

In his 1894 review of the topic, Hugues noted that Hippocrates, Pares, Morgagni, Velpeau, Fournier, and Guyon had all seen cases of hematospermia (Dermarquary, 1865; Guelliot, 1882; Hugues, 1894; Keersmecker, 1899) A number of etiologies and pathophysiologic mechanisms have been proposed to explain these symptoms (Shropshire, 1912; Young and Davis, 1926; Ashkavand, 1935; Herbst and Merricks, 1940; Pelouse, 1940; Parker, 1942; Arakawa, 1944; Huggins and McDonald, 1945; Magid and Hejtmancik, 1957; Masungaga, 1968; Ross, 1969; Leary and Aguilo, 1975). Although many consider hematospermia "idiopathic" and of little importance, it is now clear that many of these cases were incompletely investigated due to a lack of modern diagnostic technology (Leary, 1975; Yu et al, 1977; Papp and Molnar, 1981; Papp et al, 1994). With growing use of transrectal ultrasound, MRI, flexible endoscopy, and a better understanding of pathophysiology, it should be possible to reduce the number of idiopathic cases.

The following reviews the anatomic and physiologic considerations important in the diagnosis of hematospermia. An algorithm for clinical and radiologic evaluation is presented, and management is discussed.

Anatomy

The male ejaculatory apparatus consists of the testes, vas deferens, and a group of accessory sex glands, secretions of which admix in the prostatic urethra resulting in the ejaculate. The vas deferens originates at the caudal end of the epididymis and travels upwards in the spermatic cord located in the inguinal canal. It courses behind the bladder and enters the rectovesical septum. As the vas deferens approach one another, they become twisted and dilated and appear similar to seminal vesicles. The vas delivers the small-volume sperm component of the ejaculate.

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Seminal vesicles are paired hollow sacculated structures located posterior to the bladder, in front of the rectum in the recto-vesical pouch. They measure approximately 5 cm in length, and the upper bulbous portion is covered by peritoneum. They are located immediately lateral and inferior to the ureter and lateral to the vas deferens. The seminal vesicles are less than 1.5 cm in transverse diameter, and dilatation greater than this may indicate ejaculatory duct obstruction (Littrup et al, 1988).

The dilated portion of the vas deferens is termed the ampulla. As the ampulla converge towards the midline the ducts again narrows to be joined by the ducts from the ipsilateral seminal vesicles to become the ejaculatory ducts that measure approximately 4.5 cm in length and are the direct continuation of the seminal vesicles. The ejaculatory ducts enter the prostate posteriorly and obliquely at its base traversing the substance of the prostate for a length of 1–2 cm where they open into the prostatic urethra antero-lateral to the prostatic utricle (Nguyen et al, 1996). The prostatic utricle is a midline structure measuring less than 6 mm. It is a Mullerian duct remnant of endodermal origin that fails to communicate with other structures. The openings of the ejaculatory ducts are of small calibre and are collapsed, making them difficult to see or catheterize during endoscopic examination.

The prostate is a fibromuscular gland surrounding the urethra at the base of the bladder. The segment of the urethra that traverses the gland between the proximal internal and distal external sphincteric mechanism is called the prostatic urethra. The glandular portion of the prostate is surrounded by smooth and skeletal muscle, contractions of which propel the prostatic secretions via the prostatic ducts that open at the base of the prostatic urethra.

The bulbourethral or Cowpers' glands are paired pea-sized glands situated inferior to the prostate on either side posterior to the membranous urethra. The ducts of these glands pierce the urogenital diaphragm and drain into the bulb of the urethra. These gland are located below the prostatic apex and above the bulb of the penis. It is therefore essential that MRI or transrectal ultrasound imaging proceed below the apex of the prostate to view these glands completely in cases of hematospermia.

Physiology of Emission and Ejaculation

The male sexual response consists of four distinct stages: erection, emission, ejaculation, and detumescence. Emission and ejaculation are two components responsible for deposition and expulsion of semen from the prostatic urethra, respectively. The clinical expression of hematospermia is dependent on these processes and may occur in the absence of erection.

Emission begins as coordinated sequential contractions originating in the efferent ducts of the testis, the tail of the epididymis, and the convoluted portion of vas deferens. Contractions proceed in an integrated manner and propel the sperm into the prostatic urethra where it is mixed with fluid expressed from the prostate and seminal vesicles by a similar mechanism. Prostatic fluid is the first contribution to the ejaculate, followed by the sperm-rich fraction from the ampulla and the vas. Finally, the seminal vesicular fluid is deposited into the prostatic urethra. During emission, the bladder neck and external urethral sphincter are closed, creating a spindle-shaped compartment to contain the deposited seminal fluid. Physiologic closure of the bladder neck is essential for antegrade ejaculation that prevents retrograde ejaculation. Hematospermia in patients with retrograde ejaculation may present as hematuria. As many as 20% of patients with hematospermia will have simultaneous hematuria that may be partially secondary to this mechanism (Fletcher et al, 1981). Hematospermia is often an event in patients following neurophysiologic dysfunction, such as in spinal cord injury, diabetes mellitus, multiple sclerosis, transverse myelitis, retroperitoneal lymphadenectomy, or psychogenic etiology.

The initial emission phase is followed by the ejaculatory event that consists of relaxation of the external sphincter followed by rhythmic contractions of the prostate and the bulbospongiosus muscles propelling the semen in an antegrade manner out the external urethral meatus (Shafik, 1995). During these contractions, intermittent closure of the external meatus prevents retrograde flow of semen into the prostatic urethra.

The process of emission is mediated by the sympathetic nervous system via adrenergic mechanisms. Signals for emission arise in the spinal cord at the T10–L2 level. The pathways for these efferent neural signals travel from the L1 paravertebral ganglion via a complicated variety of pathways, including the hypogastric nerves, the sympathetic nerves of the lumbosacral trunk, and the spermatic nerve. Ejaculation is mediated by both parasympathetic (S 2–3–4) and somatic nervous systems via the pudendal nerve (S 2–3–4). Hematospermia can occur in the absence of erection; however, emission and ejaculation are necessary. The neural efferent signal for ejaculation originates at the S2–S4 cord level and travels via a somatic pathway along the motor division of the pudendal nerve. Although these signals are reported to originate at the spinal cord level they are modulated by inhibitory and excitatory stimuli from higher brain centers, including the anterior thalamic, preoptic, hypothalamic, and forebrain nuclei (Junemann et al, 1987; Lue, 1991; Lue and Tanagho, 1987; Malloy and Malkowicz, 1987; Creed et al, 1991; Andersson and Wagner, 1995; Burnett, 1995).

Etiologies and Pathophysiology

Hematospermia may be attributed to a variety of pathologic processes (Marshall and Fuller, 1983; Pryor, 1985). In the past, these processes have been divided into functional, pathologic, and idiopathic causes (Khan, 1983). Age-dependent classification also has its advocates because in men under 40 years of age, hematospermia tends to be of benign causes while in older men, malignant etiologies must be sought. These distinctions, however, provide little insight into the pathophysiologic mechanisms involved in the development of hematospermia and are not pragmatic with current improved diagnostic capabilities. A more precise system of organization categorizes the causes of hematospermia by the pathophysiologic mechanisms as follows: (1) inflammation and infections, (2) ductal obstruction and cysts, (3) tumors (4) vascular abnormalities, and (5) systemic and (6) iatrogenic factors (see Table 1).

Inflammation and Infections

Inflammatory processes that cause mucosal irritation, hyperemia, and edema of the involved duct or gland may lead to bleeding and the clinical presence of hematospermia (Colpi et al, 1988; Soler et al, 1989). The inflammation may be a result of traumatic chemical or infectious causes. Traumatic causes include overuse or disuse such as haemorrhage exvacuo, a phenomenon in which a distended seminal vesicle, due to prolonged sexual abstinence, finally evacuates, or bleeding caused by nonobstructing calculi of the prostate or seminal vesicle (Yada, 1963; Widdison and Feneley, 1989; Worischeck and Parra, 1994). Inflammatory etiologies include viral, bacterial, mycobacterial, and parasitic infections such as bilharziasis or schistosomiasis (Becquet, 1966; Pedro et al, 1973; Dunzendorfer and Feller, 1981; Koment and Poor, 1983; Abdel Ruzic and el-Morsy, 1990; Corachan et al, 1994).

Ductal Obstruction and Cysts

Ductal obstruction and cyst formation within the accessory sex glands may also present as hematospermia (Mayersak and Viviano, 1992; Wang et al, 1993). The mechanism responsible for these cases is probably dilation and distention, resulting in rupture of mucosal blood vessels. Multiple cases of ejaculatory duct obstruction, wolffian duct cysts, seminal vesicle cysts, and utricular cysts have been associated with hematospermia (Heller and Whitesel, 1963; Yada, 1963; Van Poppel et al, 1983; Neustein et al, 1989; Weintraub et al, 1993).

Tumors and Vascular Abnormalities

Tumors and vascular aberrations of the accessory sex glands may present with hematospermia (Tripathi and Dick, 1969). Hematospermia is a rare symptom of prostate cancer (Dumas et al, 1985; Fujisawa et al, 1993). Ectopic prostatic tissue in the urethra and prostatic polyps and proliferative urethritis producing hematospermia have been described (Stein et al, 1980; Navio et al, 1981; Glancy et al, 1983; Baroudy and O'Connel, 1984; Eglen and Pontius, 1984; Fan et al, 1984; Leifert et al, 1985; Chou et al, 1989; Satoh et al, 1989; Sato et al, 1990; Ishigooka et al, 1993). Testicular tumor presents only rarely with hematospermia (Weissbach et al, 1977). It is thought that friable aberrant vessels produced by these angiogenic tumors are responsible for the bleeding. By a similar mechanism, venous varicosities of the seminal vesicles, prostatic urethra and bladder neck may also be the source of bleeding (Cattolica, 1982; Redman and Young, 1987). In adolescents, vascular abnormalities associated with reproductive development such as arterio-venous malformations (AVMs) and hemangiomas of the prostate and seminal vesicles may be responsible but, rarely, those located in the spermatic cord (Chipkevitch, 1989; Ferrari, 1989; Harada et al, 1994).

Systemic Factors

The pathologic basis of bleeding due to systemic conditions is less obvious and may be multifactorial. Hematologic disorders such as von Willibrands disease, hemophilia, and acquired anticoagulable states secondary to liver disease may be responsible (Yu et al, 1977; Ganabathi et al, 1992; Lemesh, 1993). Hypertension has also been associated with hematospermia (Hamburger et al, 1980; Iversen, 1987). Hematospermia may even be a rare and sole symptom of lymphoma (Geoghegan and Bonavia, 1990).

Iatrogenic

The iatrogenic causes of hematospermia are currently the most commonly seen etiologies. These causes of bleeding are often evident from the patient's history. With its increased use for prostate cancer screening, transrectal prostate biopsy has become the single most common cause of hematospermia (Gustafsson et al, 1990). Other less common causes include radiation therapy of prostate cancer, brachytherapy, high intensity focus ultrasound therapy (HIFU), intraprostatic injection of medication, ureteral stent migration, and urethral foreign bodies (Baert et al, 1983; Jimenez-Cruz et al, 1988; Kumon et al, 1993; Madersbacher et al, 1993; Bihrle et al, 1994). In addition, external trauma to the perineum, sex glands, pelvic fractures, and unskilled urethral instrumentation may all result in hematospermia.

Evaluation

History

The approach to evaluation of a patient with hematospermia should be systematic and deliberate to avoid exclu-

Table 1. *Causes of hematospermia*

Inflammatory and Infections	Systemic Factors
Prostatitis	Hypertension
Seminal vesiculitis	Hemophilia
Urethritis	Purpura
Epididymo-orchitis	Leukemia
Calculi of seminal vesicles or prostate	Scurvy
Calculi of the urethra, bladder, or ureter	Amyloidosis of seminal vesicles
Sexually transmitted disease: gonorrhea, syphilis	Lymphoma
Tuberculosis	Cirrhosis of the liver
Bilharziasis (schistosomiasis)	**Iatrogenic**
Cysts and Ductal Obstruction	Prostate biopsy
Dilation of the seminal vesicles	Prostatic injections
Ejaculatory duct cyst	Post-vasectomy
Ejaculatory duct obstruction	Post-orchiectomy
Diverticula of the seminal vesicles	Trauma to perineum/genitalia/pelvis
Urethral stricture	Post-hemorrhoidal sclerotherapy
Utricular cysts	High frequency ultrasound
Prostatic cysts	Post-prostate cryosurgery or brachy therapy
	Ureteral stents

Tumors

Benign

Granulations or papillary adenomata or adenomatous polyps
Condylomata acuminata
Benign prostatic hyperplasia
Tumors of the spermatic cord or prostatic utricle
Leiomyoma of the seminal vesicle

Malignant

Carcinoma of the seminal vesicle
Testicular cancer
Carcinoma of the prostate
Sarcoma of the prostate or seminal vesicles
Intraductal carcinoma

Vascular Abnormalities

Abnormal veins in the prostatic urethra
Hemangioma of the urethra
Semino-vesico-venous fistula
Arterio-venous malformation

sion of potentially treatable causes (see Fig. 1). As with all clinical evaluation, the physician should start with a thorough history. The amount of bleeding, duration of symptoms, and types of symptoms, such as weight loss, fever, local pain, voiding complaints, and sexual symptoms should be ascertained (Tolley and Castro, 1975). It is extremely important to question the patient on how the hematospermia was observed to determine the possibility of the sexual partner being the source of bleeding. Spurious hematospermia caused by uterine or cervical carcinoma may present as postcoital bleeding and masquerade as hematospermia. Likewise, anorectal pathology may falsely present as apparent hematospermia in patients engaged in anal intercourse. As an accurate means to exclude the partners as sources of bleeding we recommend that the patient perform a "condom test," where the ejaculate is collected and analyzed and blood on the external surface of the condom can be easily identified. Hematospermia must further be distinguished from urethral bleeding and melanospermia (Lowell and Lewis, 1966; Smith et al, 1973). Surgical history or urethral instrumentation, such as prostate biopsy or sclerosis of hemorrhoids, history of exposure to tuberculosis, travel to endemic areas where TB or schistosomiasis are found, and the use of medications, especially anticoagulants or aspirin, should be included as part of a detailed history.

Physical Examination

Physical exam should be directed towards exclusion of local and systemic pathology. The patient's blood pressure and temperature is noted and the abdomen is carefully examined to exclude enlargement of the liver or the spleen or the presence of pelvic masses. The groin, perineum, and external genitalia are examined with particular

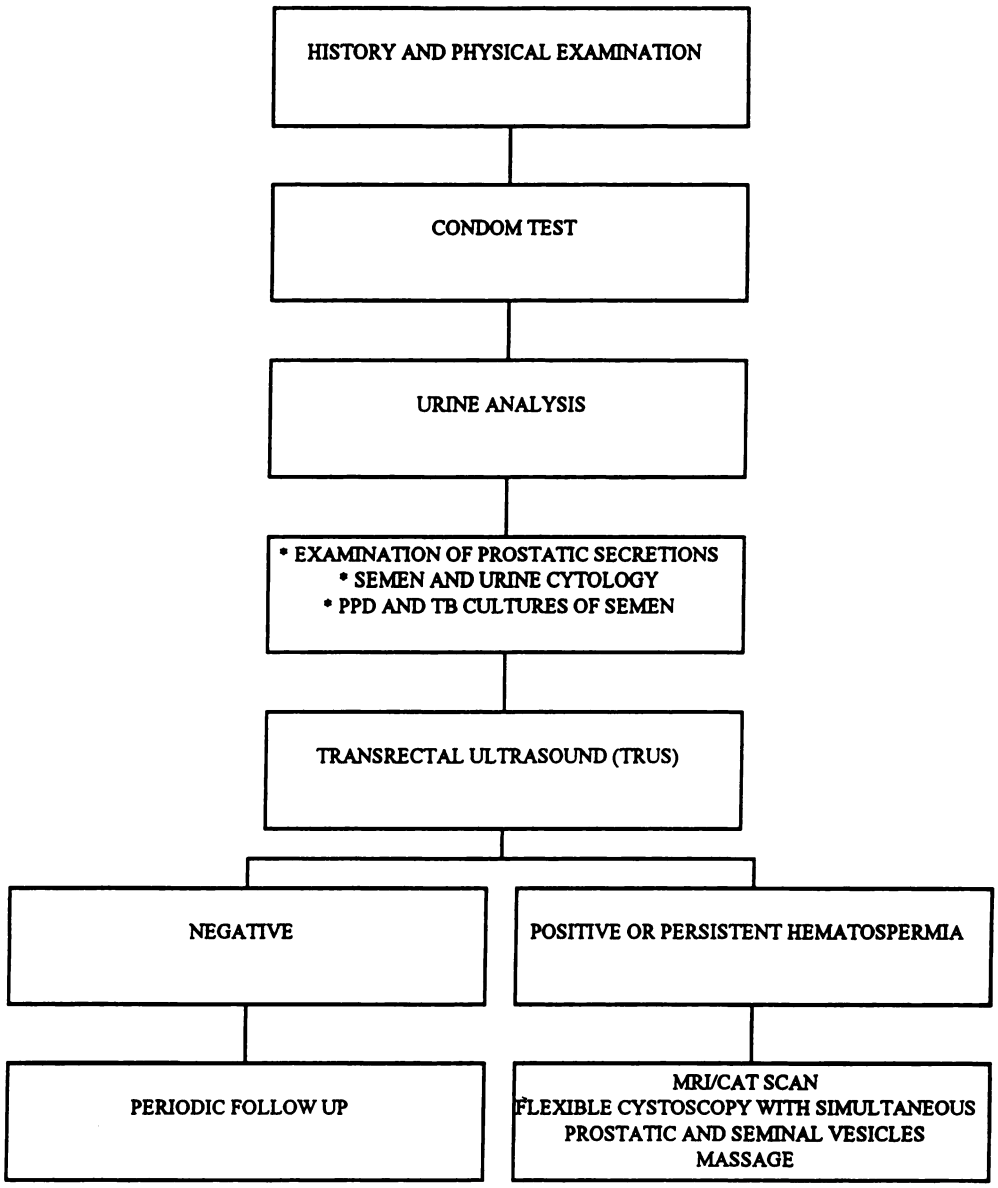

FIG. 1. Algorithm for evaluation of hematospermia.

attention to skin lesions, urethral meatus, presence of hypospadias, testes, and spermatic cord. A rectal examination is performed on all patients to rule out rectal, prostate, and seminal vesicule cysts or masses. The urethral meatus is re-examined after rectal examination for the presence of bloody discharge. In patients with absence of rectum following abdomino-perineal resection, a transperineal ultrasound or MRI may be necessary to scan the genital glands and their ductal drainage systems.

Laboratory Studies and Imaging

The laboratory assessment of hematospermia should be tailored to the individual patient. In general, urine analysis and urine culture are obtained. In the appropriate clinical setting, analysis of urine and ejaculate for acid fast bacilli (AFB) or parasites (schistosomiasis) may be necessary. Blood urea nitrogen, creatinine, platelet count, prothrombin, and partial thromboplastin times may be necessary when a bleeding diathesis is suspected. A serum PSA should be obtained in those individuals over 50 years of age or in patients over 40 years of age with a family history of prostate cancer. This is especially true for African Americans, who are at a higher risk for prostate cancer.

A significant number of cases will remain "idiopathic"

after the initial assessment. It is with these individuals that the judicious use of imaging modalities, such as ultrasound, MRI, direct flexible endoscopic evaluation of the prostatic urethra and bladder neck, and bimanual examination of the bladder with partial distention, may be appropriate. Individuals with hematospermia associated with hematuria, infertility, painful ejaculation, pyospermia, and lower urinary tract symptoms demand comprehensive urologic evaluation, as do individuals over the age of 40 in whom more serious etiologies must be considered (Murphy and Weiss, 1985; Sampalmieri et al, 1992).

Some have advocated the use of plane x-rays and intravenous urograms (IVU) to search for calcification of the prostate, seminal vesicles, and bladder (Ganabathi et al, 1992). However, Fletcher et al have found that IVU was helpful in the evaluation of only 6% of cases of hematospermia (Fletcher et al, 1981). The use of CT scanning, transrectal ultrasound, and MRI imaging have greatly improved the ability to determine the etiology of hematospermia. These non-invasive imaging modalities have given physicians visual access to the specific anatomic sites that in the past have been invisible to all but indirect endoscopic inspection or limited surgical exploration (Bacaro et al, 1992; Fuse et al, 1992; Mirowitz, 1992; Gualdi et al, 1993; Kavoussi et al, 1993; Ramchandani et al, 1993; Roy et al, 1993; Stricker et al, 1993; Amano et al, 1994; Honig, 1994; Cornud et al, 1995; McDermott et al, 1995; Ruiz Rubio et al, 1995). CT scanning was the first of these techniques available for non-invasive imaging of the prostate and seminal vesicles. The limited resolution, risk of x-ray exposure, and technical complexity gave way to the widespread use of endocavitary (endorectal) ultrasound for accessory sex-gland imaging (Schwartz et al, 1988).

Endorectal ultrasound has gained widespread popularity in the evaluation of prostate cancer (Gluck et al, 1989). Relatively inexpensive and detailed real-time sagittal and coronal images may be obtained with good resolution, without preparation or exposure to radiation. Transrectal ultrasound has been used to evaluate cysts of the seminal vesicle prostate and ejaculatory ducts (Yada, 1963; Littrup et al, 1988; Tzai et al, 1989; Soh et al, 1995). It is excellent for detecting the presence of stones in these structures. Soft-tissue masses, such as polyps and tumors, may be accurately delineated and measurements obtained. There are numerous reports regarding its sensitivity in the diagnosis of hematospermia (Yada, 1963; Littrup et al, 1988; Maeda et al, 1993; Worischeck and Parra, 1994; Soh et al, 1995). Etherington et al (1990) has demonstrated ultrasound abnormalities in 83% of patients with hematospermia. Ultrasound is considered to be the primary screening modality for patients with hematospermia and should be considered an extension of the physical examination.

Contemporary imaging of the accessory sex glands by magnetic resonance imaging (MRI) usually defines the etiology of hematospermia by detailed anatomic study of the accessory glands and their ducts. Initially, its expense restricted its use to all but the most unusual cases; however, this imaging technique has proved its worth in the diagnosis of hematospermia. The resolution of MRI has led to it being the scientifically preferred method of staging for prostate cancer. The same attribute is useful in determining the diagnosis and etiology of hematospermia (Maeda et al, 1993; Weintraub et al, 1993). The addition of contrast gadolinium further improves resolution, as does the use of endorectal surface coils (Schnall et al, 1992; Weintraub et al, 1993). MRI is especially useful in evaluation of those cases not diagnosed by ultrasound. In the future, MR angiography (MRA) may provide additional information for localization of bleeding in cases of refractory or remittent hematomspermia (Maeda et al, 1993).

In selected cases of hematospermia, invasive studies such as direct rigid and flexible endoscopy may be used to confirm and treat findings from non-invasive studies (Stein et al, 1980). These minimally invasive methods are useful as a final means to evaluate cases in which the etiology is obscure after non-invasive testing. With direct visualization, urethral polyps, papillary urethritis, prostatic cysts, urethral foreign bodies, stones, and vascular abnormalities can be ascertained (Ishigooka et al, 1993). Rigid cystoscopy allows direct examination of the prostatic urethra, bladder neck, and vesical cavity, but it still has a number of limitations and sources of intermittent bleeding may be difficult to assess. Flexible endoscopic instruments have the unique ability to "retroflex" and allow a complete visualization of the bladder neck, especially to locate vesical varicosities radiating and extending into the prostatic urethra that may easily bleed in patients taking aspirin. At times distended veins in the bladder neck or prostatic urethra may only become apparent during erection. Pharmacologic erection and simultaneous inspection with flexible instruments may be necessary in refractory and remittent cases of hematospermia. Simultaneous cystoscopic evaluation of the prostatic or ejaculatory ducts and massage of the prostate and seminal vesicles may be useful for localizing bleeding.

Other invasive studies, such as ultrasound or CT guided aspiration with contrast instillation, have been used in selected cases. Ultrasound guided aspiration of the seminal vesicles has been used to confirm the presence of ejaculatory duct obstruction. Jarrow has demonstrated that the presence of sperm in the aspirate of the seminal vesicles has a high correlation with ejaculatory duct obstruction (Jarrow, 1994). Contrast vasography is of limited use in

the evaluation of hematospermia but may be indispensable when the seminal vesicles are distended and the preceding investigation is negative.

Treatment

Treatment of hematospermia is dependent on the underlying pathology. In most cases, bleeding is slight and self limited and may be managed expectantly. This is especially true in cases of hematospermia following prostate biopsy or urethral instrumentation where calm reassurance will suffice. With infectious causes, use of appropriate antimicrobial or antiparasitic agents is indicated. When infection is clinically suspected but urine and semen cultures are negative, empiric treatment for chlamydia or bacteroides may be appropriate. Cystic lesions of the seminal vesicles, prostate, ejaculatory duct, or embryologic remnants may be treated by CT or ultrasound guided aspiration (Fuse et al, 1988; Abe et al, 1989; Wang et al, 1993). Transurethral unroofing of cysts or ductal obstruction, as well as endourologic and laparoscopic excision of seminal vesicle cysts, can be employed (Razvi and Denstedt, 1994; Kavoussi et al, 1993). Lesions, such as prostatic varicosities, polyps, ectopic prostatic tissues, and ejaculatory duct obstruction, can be managed by transurethral resection, fulguration, and dilatation (Fan et al, 1984; Suzuki et al, 1987; Weintraub et al, 1993).

Conclusion

Although many consider hematospermia to be a benign condition, all efforts should be made to clearly determine the etiology of bleeding, as advances in non-invasive imaging techniques have greatly facilitated the diagnosis of hematospermia. A thorough history and physical examination, accompanied by specific radiologic tests, focused examination of biological fluids, screening transrectal ultrasound, MRI imaging, and observation with flexible instrumentation will reduce the number of "idiopathic" cases of hematospermia, identify serious pathology, and enable treatment early in the course of the disease.

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