Historical perspective of prostatitis

The prostate gland was described in the anatomical studies of Herophilus about 350 BC and was rediscovered in the 16th century by the Venetian physician Nicola Massa. At the same time the physician Riolanus noted that bladder obstruction can be caused by swelling of the prostate gland. Prostatitis as a clinical entity/syndrome was first described in 1815 by Legneau, who noted that inflammation of the prostate gland could be a complication of urethritis (von Lackum 1928).

The first accurate description of the pathology of prostatitis in written form was presented by Verdies in 1838, and this was later confirmed and updated by Hugh Young, Gereghty and Stevens in 1903 and published in 1906 (Young et al. 1906, von Lackum 1928).

The time between the 1880 and 1928 was a period of searching for bacteria and confirming the postulates of Koch and Wirchow related to the mechanisms of bacterial infections acting in the human body (Hitchens & Brown 1913, von Lackum 1927).

Between 1900 and 1930, the basic chemistry of prostatic fluid was studied and the role of possible pathogen bacteria cultured from the prostate gland and from prostate secretion was defined and von Lackum´s theory of possible retrograde secondary infection ascending from the urethra to the prostatic ducts was clinically demonstrated by several physicians (Young et al. 1906, Hitchens & Brown 1913, von Lackum 1927, Nickel 1930).

The period between 1930 and 1960 was marked by an active search for aetiological factors related to prostatitis syndrome, factors that could be responsible for its chronicity and the histological changes that take place, fibrosis and scarring together with late functional disorders of the prostate gland. Despite of all these new findings, there were many internists and psychoanalysists who denied the existence of chronic prostatitis processes, and the latter group termed such symptoms ”anal/rectal psychoses” (Cumming & Chittenden 1938). Ritter and Lippow (1938) searched for an explanation for the natural history of the pathological processes involved in prostatitis, and they also described the histological findings during acute phase of inflammation as hyperaemia, oedema, cellular infiltration of polymorphonuclear leukocytes and round cells in the prostate tissue. Grant (1938) noted that the infection was mostly located in the ducts and acini of the prostate. On the other hand, Kretschmer (1937) drew conclusions from 1000 cases that pointed to polyaetiological aspects of the disease, and also reflected on the sexual life of patients.

There was a period in the 1940s and 1950s when it was generally accepted that an important offending organism in cases of acute infectious prostatitis was gonococcus.

Henline (1943) reported that untreated gonococcal urethritis was a possible aetiological factor in over 20–30% of cases of chronic prostatitis symptoms, but Kretschmer (1937) found gonococcus to be an aetiological reason for prostatitis in only 2.4% cases.

Genitourinary tuberculosis was regarded as a specific aetiological factor between 1900 and 1960. Tuberculosis of the prostate gland as only affected genital organ was found in 12% cases in an autopsy series, and the majority of patients with upper genitourinary tuberculosis were also found to have signs of prostatic irritation (Moore 1937, Meares 1998).

In the late 1950s it was recognized and noted, especially by Campbell (1957), that chronic prostatitis may be present in the prostate gland without any clinical symptoms, and it was also realized that a disease such as prostatitis can be congestive and non-bacterial.

The active clinical research period of the 1970s and 1980s, was motivated by the findings of Meares and Stamey (1968), which pointed to a new direction in the diagnosis of prostatitis, and those of Drach et al. (1978), which provided a new classification of the disease. This was followed by a period of collecting clinical experience and basic knowledge on prostatitis at large, using new laboratory methods for bacterial cultures, microscopy and immunofluorescence imaging.

From the 1990s onwards a young generation of disappointed urologists who did not accept all the written data from past concerning prostatitis as a forgotten ”light urological ambulatory pathology” and ”not needing special urological care” started a new era in prostatitis research (Nickel 1999, Nickel et al. 1999b).

Aare Mehik, MD. Department of Surgery/Division of Urology Oulu University Hospital Kajaanintie 50 Oulu 90220 Finland Tel: 358-8-315-2011 Fax: 358-8-315-2447, E-mail: amehik@cc.oulu.fi Artikeln finns i en foskningsrapport på Uleåborgs Universitets hemsida http://herkules.oulu.fi/isbn9514265068/html/index.html.

The Swedish Chronic Prostatitis Network http://home.swipnet.se/isop