Primary hyperaldosteronism is a traditional term of rare form of renin-angiotensin-independent mineralocorticoid excess syndrome resulting from an autonomous secretion of the hormone by an adenoma, carcinoma or hyperplasia of the glomerular zone of the adrenal glands. Metabolic defects, usually inherited, are other cause of aldosterone excess in patients with primary hyperaldosteronism. Primary hyperaldosteronism is known in medical literature as Conn’s syndrome. The symptoms of this clinical condition are skeletal muscle weakness or intermittent paralysis, polyuria, hypertension and cardiac arrhythmias. The symptoms are associated with hyperkalemia, sodium retention and alkalosis.

The first description of primary hyperaldosteronism is commonly attributed to Jerome W. Conn who in 1955 reported a 34-year-old female patient with weakness and periodic paralysis of the lower extremities and frequently recurrent numbness with cramping of her hands lasting for 7 years prior to hospital admission. Jerome W. Conn presented the case at his presidential address to the Central Society for Clinical Research on October 29, 1955 [1]. In the same year, the report was published in the Journal of Laboratory and Clinical Medicine [2]. Later, Jerome W. Conn published other papers and reviews on primary hyperaldosteronism [3].

Conn’s description is considered the first report on primary hyperaldosteronism in medical literature although more than two years earlier, two cases of malignant hypertension caused by tumors of adrenal gland cortex had been published by a Polish internist Michał Lityński [4]. He reported two male patients who died due to malignant hypertension and renal insufficiency caused by hypertension, and in whom autopsy revealed a tumor in the adrenal glands. The first 48-year-old man had a tumor in both adrenal glands (tumor diameter 24 mm, 30 mm respectively) while in the second one, 44-year-old individual a large tumor of the right adrenal gland was discovered at autopsy (the gland measured 6 x 5 x 1.5 cm). Microscopic evaluation of the tumors revealed that they consisted of large cells surrounded by blood vessels. The cells had foamy cytoplasm and small nuclei, and resembled those of the glomerular zone of the adrenal gland. Clinically both patients had symptoms of severe malignant hypertension and in the last stage of the disease symptoms of renal insufficiency. Blood pressure in both was 240/150 mmHg. In conclusion Michał Lityński wrote: “One can suppose that in both cases hypertension was caused by adrenocortical tumors, bilateral in one case, and localized in the right adrenal gland in the other case. In the structure of the tumors, proliferation of the cells resembling those of the glomerular zone was found; which secretes mineralocorticoids. This finding suggests that in both cases overproduction of the hormones had took place”. The paper of Lityński was received by the editorial office on July 29, 1952.

Only a few biographical details of Michał Lityński are known and his life and achievements need further medico-historical studies. He was born on June 14, 1906 in Łódź. He studied medicine at the Warsaw University Center for Military Medical Education in 1925–1931 as a cadet. After an internship in Warsaw from November 1932 until March 1936, he took the post of military physician in Toruń. From 1936, he was the head of the internal medicine ward in the Center for Military Education Hospital in Warsaw, the so-called Ujazdowski Hospital. After the outbreak of the Second World War, he was a commander of the military hospital in Garwolin, and later returned to the Ujazdowski Hospital in Warsaw. There he was the head of the 6th ward of internal medicine. Michał Lityński was an active participant of the Home Army resistance movement. He harbored wounded soldiers of the underground army and persons of Jewish origin who escaped from the
ghetto in the hospital. For his activity, Lityński was awarded the medal "The Just Man among the World Nations" in 1986 [5]. During the Warsaw uprising, he was a commander of the medical post at Central Warsaw, later he worked with partisans in the Mariańska Forest. After the war, he worked as a physician in Gdańsk and from 1946 in Warsaw. Michael Lityński was a physician in the Wola Hospital and head of the ward at the Tuberculosis Institute.

He published a dozen research papers, including studies on amyloidosis, diabetes, protein metabolism as well as papers on history of medicine and war memoirs. He died on March 5, 1989 in Warsaw [6].

Jerome W. Conn was born on September 24, 1907 in New York. He graduated with honors from the University of Michigan School of Medicine in 1932. All his professional life he was associated with the university. He was the Director of the Division of Endocrinology and Metabolism from 1943 to 1973, and since 1950, he was a Professor of Internal Medicine. Beside his studies on primary hyperaldosteronism, he investigated obesity and regulation of human adaptation to high temperature. Jerome W. Conn died on June 11, 1994 in Naples, Florida.

The description by Michał Lityński was forgotten and almost unknown even in Polish medical literature. In 1983, the Lityński’s paper was mentioned in the book of Walentyna Hartwig [8] and in 1984 by Franciszek Kokot [9]. A few years later, Tadeusz Marcinkowski proposed to rename the syndrome as “Lityński-Conn syndrome” [10]. In 1991, “Lancet” published a letter indicating the priority of Michał Lityński in discovery of primary hyperaldosteronism [11], and subsequently Norman Kaplan has included a note on the first description of the disease by Michał Lityński in a new edition of his "Hypertension". Lityński’s name also appeared in reviews on the history of endocrinology.

REFERENCES