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Case Report

A case with primary signet ring cell adenocarcinoma of the prostate and review of the literature

Keywords: carcinoma, hypertension, pancytopenia, physical examination, duodenoscopy, signet ring cell, adenocarcinoma, prostates

Abbreviations: NA, not applicate; H, hormone; R, radiotherapy; RP, radical prostatectomy; CT, chemotherapy

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De La Pinta C, Martin M
Department of Radiation Oncology, Hospital Ramon Y Cajal, Spain

Correspondence: De La Pinta C, Department of Radiation Oncology, Hospital Ramn Y Cajal, Spain, Email cdelapinta88@gmail.com

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Case report

Primary signet ring cell carcinoma (SRCC) constitutes approximately 3-4% of the stomach cancers; it is very rare variant of prostate carcinoma. It is estimated to occur in 2.5% of cases of prostate adenocarcinoma.[1] Prostate SRCC is diagnosed by eliminating of other organs such as colon and stomach, pancreas, breast, thyroid and bladder. Prognosis is worse than the adenocarcinoma. Only 69 cases have been reported in the English literature since 1979.[2] We recently diagnosed a case of primary SRCC of the prostate by this we review previous reports. Based on our case, we present the pathologic, clinical and therapeutic aspects of this rare entity.

In July 2016 an 83-year-old male was referred to our institution for evaluation of a serum prostate antigen (PSA) level of 8, 79 ng/ml. The patient had personal history of hypertension and pancytopenia. He had personal or family history of malignancy. Levels of serum prostatic acid phosphatase (PAP) and carcinoembryonic antigen (CEA) were normal. Physical examination, including digital rectal exam, demonstrated invasion of unilateral seminal vesicles. A prostate needle biopsy was carried out. Histologically, biopsy demonstrated an adenocarcinoma with predominantly of the signet ring cell component, Gleason 8 (4+4) in left prostate lobe (65%). Duodenoscopy and colonoscopy were performed for evaluation of the gastrointestinal and no evidence of tumour was found. Overall, this tumour was regarded as primary SRCC of the prostate. Radionuclide bone scan and computed tomography body were negative for metastatic disease. The patient was diagnosed with primary SRCC of the prostate (T3bN0M0).

In multidisciplinary committee we decided androgen blockade. One month later of blockade, his serum PSA level was reduced to 0.1ng/mL. He was alive with no evidence of disease 8 months after blockade. As in common prostatic adenocarcinoma, the tumor retained hormonal dependency, showing a dramatic response to androgen deprivation therapy.

Discussion

The majority of prostatic cancers are acinar adenocarcinomas. Histological variants of prostatic carcinoma have been variably defined. One approach is to consider two groups of variants. The first group comprises histological variants of acinar adenocarcinoma (atrophic, pseudohyperplastic, foamy, colloid, signet ring, oncocytic and lymphoepithelioma-like carcinomas) and the second group non-acinar carcinoma variants (5-10%).[3]

The term "signet ring cell" is used to describe cells that have their nuclei displaced by an intracytoplasmic vacuole. These cells are more frequently seen focally within otherwise high-grade prostate cancers and should be considered as part of the Gleason scoring.[3] But, Guerin and colleagues[1] suggested that SRCC should be classified as a variant of high-grade adenocarcinoma rather than a separate histologic classification. The largest series to date has shown poor survival. Clinically, primary SRCC of the prostate presents with the same classic obstructive and irritative symptoms or asymptomatic as other common adenocarcinomas, the diagnosis of primary prostatic SRCC of the prostate is certainly a histologic diagnosis that can be done using needle biopsy, endoscopic resection specimens or radical prostatectomy. To diagnose primary SRCC of the prostate, tumour should contain more than 25% of these cells. In our patient, the gastrointestinal workup showed no evidence of tumour. Therefore, we assumed that the prostate was the primary source of the tumour. Some series show carcinoembryonic antigen (CEA) positive in 20% of the cases (10) whereas PSA and prostate specific acid phosphatase (PSAP) were positive in 87% of the cases[4] and positive staining with periodic acid-schiff stain (PAS) was positive in 60%, with alcian blue 60% and with mucicarmin 50%. Clinically, SRCC of the prostate is usually diagnosed in an advanced stage of disease through elevated PSA levels. However, some cases with SRCC have high carcinoembryonic antigen immunoreactivity, while PSA is negative.

We reviewed previous English reports and found 69 reported cases of primary SRCC of the prostate. Table 1 shows the results of special treatment of more important cases. Because of its rarity, no recommended treatment has been established. Conventional therapy for prostate cancer has generally been used, involving variable combinations of hormonal therapy, radiation and surgery. Nevertheless, on the basis of the available cases, the effectiveness of hormonal therapy is unpredictable. To date, no definite explanation for this variability has been given however; Lilleby et al.[12] reported a case of SRCC treated with Neoadjuvant hormonal therapy and radiotherapy with a favorable response at 12 months of follow-up. Moreover, Kanematsu and Hiura[6] reported a case of primary SRCC with an undetectable PSA level 3 years after a radical prostatectomy and preoperative androgen blockade. Akagashi et al.[8] further reported a case of an undetectable PSA level 20 months after treatment with

complete androgen blockade. No single treatment modality is ideal for treating SRCC, but we consider that more aggressive multimodal treatment should be considered. Androgen deprivation therapy in Yamamoto study was not effective and therefore that systemic chemotherapy (due to possible gastrointestinal tract origin) and irradiation therapy should be recommended.[20,21] In our case, treatment response to androgen blockade was good.

Table 1 Results of special treatment of more important cases

	Stage	Age	PSA/CEA	treatment	Follow up months	Status
Yoshimura et al.[5]	T3N0M0	65	+/NA	H+R	100	Alive CR
Kanematsu et al.[6]	T2N0M0	76	237/-	H+RP	36	Alive
Ishizu K[7]	E-IV ParaAO lymph nodes	67	46,2/NA	H	2	Alive CR
Akagashi K[8]	T4N0M0	72	+/NA	H	20	Alive CR
Fujita [9]	T2aN0M0	75	9,3/-	H+RP	12	Alive
Uemura M[10]	Local stage	76	28/NA	RP	13	Alive
Derouiche[11]	T2	85	9,1/-	H	18	Alive
Lilleby [12]	T3bN0M0	70	27/-	H+R	12	Alive
Matsuoka Y[13]	cT4N1M1c	62	364.7/NA	H	15	Died
	cT1	58	NA	R	Median 30	24
	T4M1	82	NA	H		5
	T4M1	68	NA	H		12
	T3M1	65	NA	H		24
Warner et al.[14]	T3a	66	NA	RP		108
	cT2a	67	1,9	H+R		Alive at 48
	T3b	79	5,9	CP		4
	T3b	50	NA	RP+R		Alive at 36
	T2b	59	4,8	RP+H		Alive at 12
Hashimoto Y[15]	T3N2M2	61	0,19	CT	16	Died
Bonetti LR[16]	-	70	+	H+R	11	Died
Kwon[17]	T2bN0M1b	61	14,7	H Progression at 9m docetaxel	11	Died
Celik et al.[18]	E-IV Bone metastases	66	6658	H+docetaxel	22	Died
Kim et al.[19]	T3bN0M0	56	0,64	RP+CT Progression	24	Died

NA, not applicate; H, hormone; R, radiotherapy; RP, radical prostatectomy; CT, chemotherapy

Prostate specific antigen values and treatment modalities were not determinants of survival. Many authors reported the poor prognosis of prostate SRCC, with less treatment response and poor prognosis when compared to the classical type of the prostate adenocarcinoma. It is difficult to define an optimum treatment strategy for SRCC of the prostate from the existing data, early diagnosis and local treatment of the prostate when there is no evidence of metastatic disease might improve prognosis. Fujita et al.[9] reported that, in primary SRCC of the prostate, the survival rate is about 55% at 3 years and decreases to 12% at 5 years. The same study revealed that prognosis is only related to the stage of the disease at diagnosis and more of these patients present with stage IV.

Conclusion

We conclude that signet-ring-cell carcinoma of the prostate is a variant of poorly differentiated adenocarcinoma of the prostate (high grade).[22] Therefore, some authors have hypothesized that prostatic SRCC is not always an aggressive disease.[8] The present case of prostatic SRCC responded well to medical therapy, however, tumors can recur after a long period of time. We recommend early, careful assessment for appropriate diagnosis.

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Conflicts of interests

The authors declare that there is no conflict of interest.

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