CLINICAL PATHWAY IMPROVES IMPLEMENTATION OF EVIDENCE-BASED STRATEGIES FOR THE MANAGEMENT OF ANDROGEN DEPRIVATION THERAPY–INDUCED SIDE EFFECTS IN PROSTATE CANCER PATIENTS

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Background

Androgen deprivation therapy (ADT) is one of the cornerstones in the management of locally advanced and metastatic prostate cancer (PCa) but it also negatively influences bone, metabolic, cardiovascular health and body composition. These adverse effects are detrimental to patients' health and quality of life (QoL). Evidence-based recommendations are available for the management of ADT-induced side effects. Clinical pathways are widely used to structure and standardize evidence-based care processes and to optimize adherence to guidelines. A clinical pathway is defined as "a complex intervention for the mutual decision making and organization of care processes for a well-defined group of patients during a well-defined period"[1].

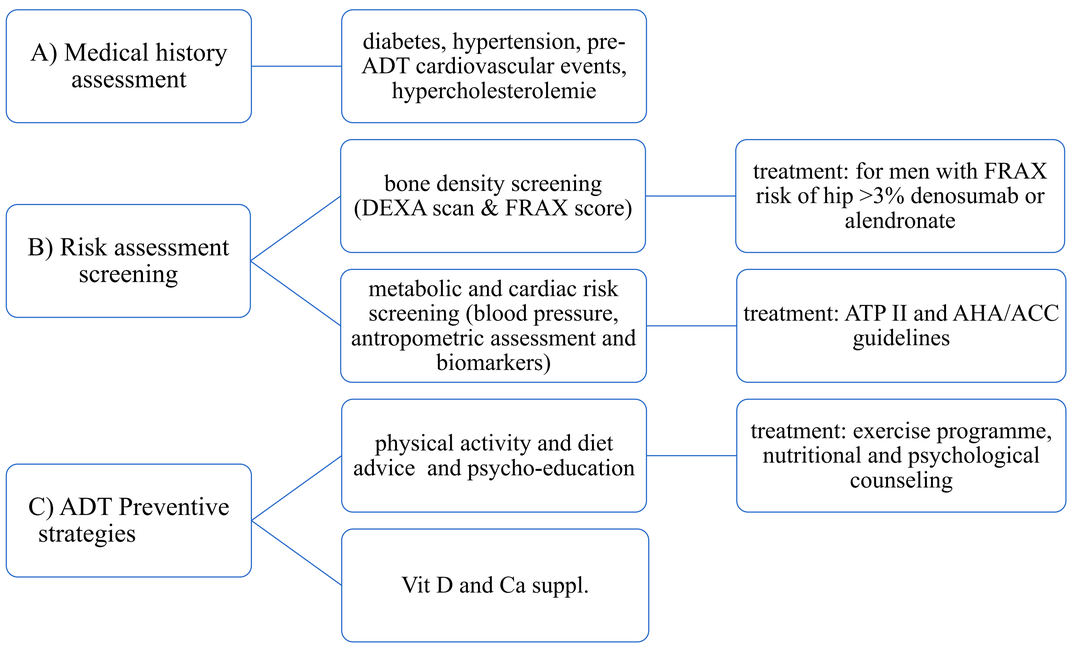
Objective

To assess the effects of a PCa clinical pathway on the implementation of evidence-based strategies for the management of ADT-induced side effects.

Materials & Methods

Patients receiving ADT during the year 2014 were allocated to the control group (no clinical pathway) and patients receiving ADT in 2015 to the intervention group (clinical pathway). The clinical pathway is based on evidence-based strategies[2,3] (cfr. Figure 1).

Figure 1:Clinical pathway in 2015 at Ghent University Hospital



Descriptive statistics were used for patient characteristics. The Chi-square test and Mann-Whitney U test were used to compare groups. Because of multiple comparisons, p=0.003 was considered statistically significant (adjusted with the Bonferroni method).

Results

Table 1: Baseline patient characteristics

Patient characteristics		Control group n=126	Intervention group n=132	p-value
Age at inclusion, years (range)				0.980
		69.3 (49-87)	69.4 (48-87)	
PSA at diagnosis (ng/ml)				0.420
	Mean (range)	30.9 (1.13-426)	27.3 (1.13-304)	
	Median (IQR)	14.0 (7.4-30.0)	14.2 (7.0-28)	
Gleason at diagnosis, n (%)				0.479
	2-6	10 (7.9%)	13 (9.8%)	
	7	34 (27.0%)	45 (34.1%)	
	8-10	75 (59.5%)	71 (53.8%)	
	Unknown	7 (5.6%)	3 (2.3%)	
Tumor stage at diagnosis, n (%)				0.669
	p/c T1	9 (7.1%))	9 (6.8%)	
	p/c T2	29 (23.0%)	42 (31.8%)	
	p/c T3	66 (52.4%)	66 (50.0%)	
	p/c T4	9 (7.1%)	10 (7.6%)	
	Unknown	13 (10.3%)	5 (3.8%)	
Prostatectomy, n(%)				0.153
	No	87 (69.0%)	79 (59.8)%)	
	Yes	39 (31.0%)	53 (40.2%)	
Lymphadenectomy				0.892
	No	36 (28.6%)	39 (29.5%)	
	Yes	90 (71.4%)	93 (70.5%)	
Radiotherapy				0.862
	No	7 (5.6%)	8 (6.1%)	
	Yes	119 (94.2%)	124 (93.9%)	
Setting ADT at inclusion				0.473
	Localized	79 (62.7%)	77 (58.3%)	
	Palliative (non)-metastatic	47 (37.3%)	55 (41.7%)	

Data are represented as mean or number (%)

Figure 2A: Medical history ADT management

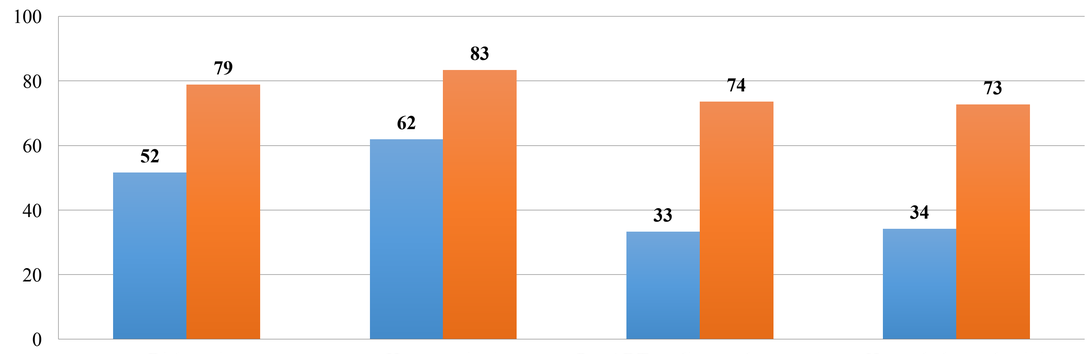


Figure 2B: Risk assessment screening ADT management

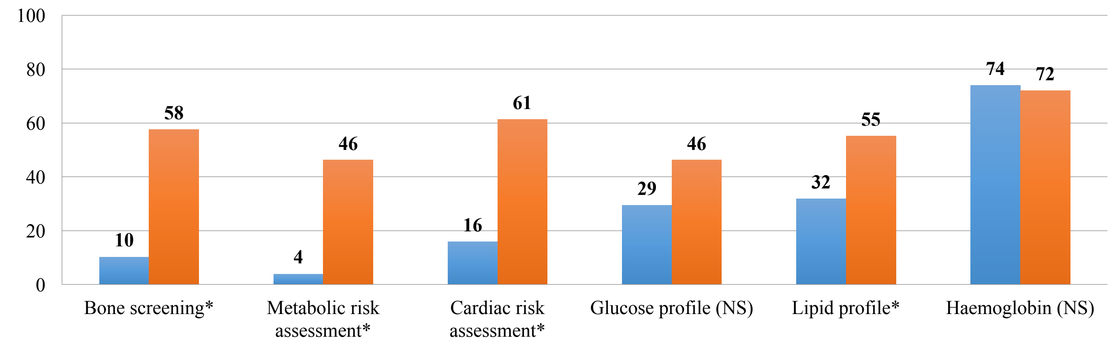
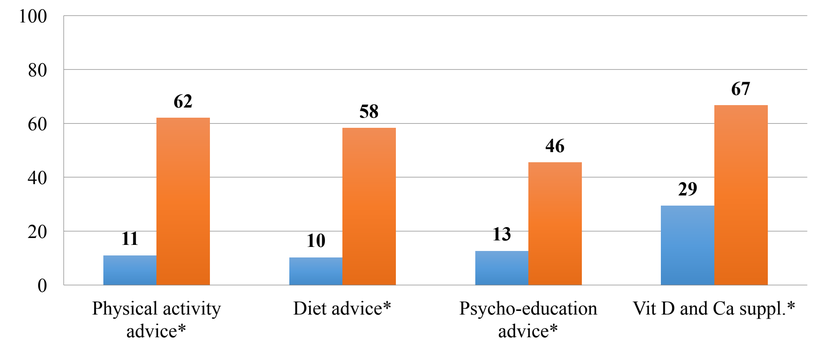


Figure 2C: ADT preventive strategies



Legend figure 2:

Control group

Intervention group

An asterix (*) indicates a significant improvement in score between groups and all p-values are adjest for multiple testing.

NS= not significant

Data are represented in %.

Table 2: Comorbidity risk factors intervention group

Co-morbidity		Number of available risk assessments	Patients affected in intervention group
BMI		111 (84.1%)	
	Underweight (<18.5 kg/m^2))		1 (0,9%)
	Normal weight (18.5-24.49 kg/m^2))		29 (26.1%)
	Overweight (25-29.99 kg/m^2))		59 (53.2%)
	Obese (30-39.99 kg/m^2))		22 (19.8%)
Hypertension		108 (81.8%)	
	No		46 (42.6%)
	Yes		62 (57.4%)
Hypercholesterolaemia		96 (72.7%)	
	No		49 (51.0%)
	yes		47 (49.0%)
Triglycerides		61 (46.2%)	
(within 3 months off inclusion)	Normal < 150 mg/dL		45 (73.8)
	Borderline high (150 – 199 mg/dL)		11 (18.0%)
	High (200-499 mg/dL)		5 (8.2%)
Diabetes		103 (78.0%)	
	No		83 (80.6%)
	Yes		20 (19.4%)
(within 3 months off inclusion)		56 (42.4%)	
	Fasting glucose (80-100 mg/dL)		22 (56.4%)
	Fasting glucose (100-120 mg/dL)		11 (28.2%)
	Fasting glucose (> 120 mg/dL)		6 (15.4%)
Bone mineral density		73 (55.3%)	
	Normal bone density (T-score between +1 and – 1)		43 (58.9%)
	Osteopenia (T-score between -1 and -2.5)		21 (28.8%)
	Osteoporosis (T-score <-2.5)		9 (12.3%)
Pre-ADT cardiovascular event		96 (70.4%)	
	No		51 (53.1%)
	Yes		45 (46.9%)

Data are represented as number (%)

Conclusion

A **clinical pathway** for PCa patients quickly **improved** the implementation of **evidence-based strategies** for the management of **ADT-induced side** effects and therefore improves quality of care. A clinical pathway may be one of the solutions to bridge the gap between evidence-based guidelines and daily clinical practice.

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[1]Vanhaecht K, Panella M, Van Zelm R, Sermeus W. An overview on the history and concept of care pathways as complex interventions. International Journal of Care Pathways. 2010;14(3):117-23.
[2]Ahmadi H, Daneshmand S. Androgen deprivation therapy: evidence-based management of side effects. BJU international. 2013;111(4):543-8.
[3]Nguyen PL, Alibhai SM, Basaria S, D'Amico AV, Kantoff PW, Keating NL, et al. Adverse effects of androgen deprivation therapy and strategies to mitigate them. European urology. 2014;67(5):825-36.