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ADENOMATOID TUMORS OF THE EPIDIDYMIS

Richard D. Sautter

Submitted in Partial Fulfillment for the Degree of Doctor of Medicine College of Medicine, University of Nebraska February 27, 1953 Omaha, Nebraska

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INTRODUCTION

Since Sakaguchi (1) first reported a tumor of this type as an adenofibromyoma in 1916, it has been known by a variety of names, such as mesothelioma, adenoma, fibromyoma, mycademfibroma, adenomatoid leiemyoma, adenocarcinoma Grade I, scirrhous carcinoma Grade I, and mixed lieomyoma and lymphangioma. The tumor was first designated as 'adenomatoid' by Golden and Ash (2) in 1945. This inconsistency of designation arises from the disparity of opinion as to genesis of the tumor and type of cell present. Since the first report in 1916, there have been a little less than one hundred cases reported.

While this is a rare lesion, its importance lies in the differentiation from other primary tumors of the epididymis and surrounding structures. The adenomatoid tumor, while it may appear microscopically malignant, is clinically completely benign, and the majority of other tumors found in this region are malignant. Confusion of this benign lesion with a malignancy could obviously have far-reaching consequences concerning the patient's future, possibly causing needless mental anquish and even material less.

This paper contains the review of sixty-six cases of this type of tumor, approximately two-thirds of all reported cases. There is also a report of four new cases. The purpose is thereby to remind the physician once again of the existence and importance of the diagnoses of this lesion.

LITERATURE AND CASE REPORTS

In the following Table I, there is a tabular presentation of sixty-six cases of adenomatoid tumor of the epididymis, which was obtained from a review of the literature.

In Table II, there is a presentation in similiar manner of four new cases of this tumor, obtained from a review of the files of Dr. J. R. Schenken, Pathologist, Nebraska Methodist Hospital, Omaha, Nebraska.

In both of the tables, the legend is as follows:

¥	p ositiv e	
-	negative	
0	no report	t

											Ð						**************************************
Case Number	Year Reported	Author	Author's Designation	Color	Age	Tumefaction	Pain	Incidental	Trauma	V. D.	Increase in Size	Duration	Tenderness	Hydrocele	Right	Left	Pre-Operative Diagnosis
1	1916	Sakaguchi	Adenofibromyoma	W	32	-	-	-	0	-	0	Long	0	-	0	0	0
2	1917	Stout	Fibronyoma	0	57	-	-	*	*	*	-	0	-	-	-	*	Testicular tumor metastatic to bile duct
3	1924	Hinman & Gibson	Scirrhous Ca Grade I	M	7 8	*	-	-	-	-	-	ll mos.	-	-	-	*	Fibroma, Gunma
4	1 935	Fischer (Spivack)	Myoadenofibroma	0	37	*	-	-	-	-	*	6 yrs.	*	-	-	*	0
5	1936	Thompson	Adenocarcinoma Grade I	0	56	*	-	-	-	-	*	6 yrs.	*	*	-	*	Bilateral hydro- cele
6	1936	Thompson	Adenocarcinoma Grade I	0	27	*	-	-	-	-	*	7 yrs.	*	0	*	-	Tuberculous Epididymitis
7	1936	Thompson	Adenocarcinoma Grade I	0	3●	*	-	-	-	-	*	10 yrs.	-		-	*	Testicular Tumor
8	1936	Thompson	Adenocarcinoma Grade I	0	29	-	-	*	-	-	-	0	*	0	*		Chronic Epididymitis
9	1939	Charache	Lymphangioma	M	43	*	-	-	-	-	*	10 yrs.	*	*	-	*	Hydrocele Tumor of Epididynis
10	1941	Halpert	Mixed Leiomyoma & Lympangioma	C	60	-	-	*	-		-	2 mos.	0	0	-	*	Epididymitis

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TABLE I

Treatment	Location		Size in cms.	Shape	Color	Encapsulated?	Inflammation ?	Muscle Fibers?	Lymphocytic Infil.	, , ,	Comments
Epididymectomy	Caput Major		0	Globular	Grey White	-	-	•	0		0
Excision	Caput Minor	1.0		0	0	0	0	0	0	Patient	jaundiced
Epididymectomy	Caput Minor	9 x 5		0	White Grey	0	0	*	0	Basemen present	t membrane
Epididymectomy	Caput Minor	Hazel	Nut	0	0	*	0	0	0	Cyst on Major	Globus
Excision	Caput Major	0.7		0	Bluish White	*	0	0	0		0
Epididymectomy	Caput Minor	2.5		0	0	*	0	0	0		0
Excision	Caput Minor	2.0		0	Grey White	*	Ũ	0	0		0
Epididymectomy	Caput Minor		0	0	0	*	0	0	0	Incapac pain	itating
Excision	Caput Minor	3.0		0	0	0	0	0	0	Alive & years l	well 3 ater
Epididymectomy	0	3 x 2	x 1.5	Globular	G rey White	0	0	0	0		0

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Case Number	Year Reported	Author	Author's Designa ti on	Color	Age	Tumefaction	Pain	Incid ental	Trauma	V. D.	Increase in Size	Duration	Tenderness	Hydrocele	Right	Left	Pre-Operative Diagnosis
11	1941	Blumer & Edwards	Adenoma	0	54	*		-	-	-	-	38 yrs.	-	*	0	0	Hydrocele
12	1941	Gordon- Taylor & Ommaney- Davis	Adenoma-	0	0	*	-	-	-	-	•-	F ew wks.	-	-	-	*	Testicular Tumor
13	1 943	Evans	Mesothelioma	0	53	0	0	0	0	0	0	3 yrs.		0	0	0	0
14	1 943	Evans	Mesothelioma	0	36	-	*	-	0	0	*	17 yrs.	*	0	*	-	0
15	1943	Evans	Mesothelioma	0	30	0	0	0	0	0	0	Sev. yrs.	-	0	0	0	0
16	1 943	Evans	Mesothelioma	0	55	0	0	0	0	0	0	4 yrs.	*	*	0	0	0
17	1 943	Ma l ioff & Helpern	Mixed Leiomyoma & Lymphangioma	W	57	-	*	-	*	-	*	4 wks.	*	-	*		Traumatic epid dymitis; tumor Epididymis
18	1945	Robinson	0	0	35	*		-	-	-	*	5 yrs.	-	-	-	*	0
19	1945	Robinson	0	0	28	*	*	-	-	-	*	4 yrs.	*	-	-	*	0

Pre-Operative Diagnosis	Treatment	Locati on	Size in cms.	Shape	Color	Encapsulated ?	Inflammation ?	Muscle Fibers ?	Lymphocytic Infil.		Comments
cele	Exci si on	Caput Minor	3•5 x 3	Sphere	Yellow White	*	0	*	*		0
cular	^U rchiectomy	Caput Minor	0	Round	0	*	0	0	0		0
0	Excision	Caput Minor	1.7	Globular	0	0	0	0	0	1 Year - Recurren	- No Nce
0	Excision & Orchiectomy	0	2.0	Globular	0	0	*	0	0	ll Mos. Recurrer	- No nce
0	Excision	0	2.0	Oval	0	*	0	0	0	No Recur	rence
0	Excision	0	1.0	Round	0	0	0	0	0	Patient	Well
atic epidi- is; tumor o dymis	Excision f Orchiectomy	Caput Major	0.18	0	0	*	0	*	*		0
0	Epididymectomy	Caput Minor	2•5	Sphere	G rey B rown	0	0	0	*		0
0	Epididymectomy Orchiectomy	Caput Minor	2.5	Sphere	Grey Brown	0	0	0	*		0

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11-11-11-1	the spectrum in	Case Number	Year Reported	Author	Author's Designation		Color	Age	Tumefaction	Pain	Incidental	Trauma	V. D.	Increase in Size	Duration	Tenderness	Hydrocele	Right	Left	Pre-Operative Diagnosis
All a		20	1945	Golden & Ash	Adenomatoid Tumor		M.	29	-	-	*	-	-	-	0	-	-	-	-	0
F		21	1945	Golden & Ash	Adenoma toid Tumor		W	23	*	-		-	-	*	6 mos.	*	•	*	-	0
E		22	1 945	Golden & Ash	Adenomatoid Tumor		W	28	*	-	-	*	-	*	5 yrs.	-	-	*	-	0
		23	1945	Golden & Ash	Adenomatoid Tumpr		W	32		-	*	-	-	-	0	-	*	-	-	Hydrocele
14	_	24	1945	Golden & Ash	Adenomatoid Tumor		W	26	*	-	-	*	-	-	3 yrs.	-	-	-	*	0
and the second second		25	1945	Golden & Ash	Adenomatoid Tumor		W	44	*	-	-	-	-	*	l0 yrs.	*	-		*	0
a to the		26	1945	Golden & Ash	Adenomatoid Tumor	÷	W	21	*	*	-	-	-	-	0	*	-	*	-	0
KR JPCI		27	1 945	Golden & Ash	Adenomatoid Tumor		W	26	*	*	-	-	÷	*	8 mos.	*	-	-	*	0
1		28	1945	Golden & Ash	Adenomatoid Tumor		W	33	0	0	0	-	-	-	0	-	-	*	-	. 0
		29	1945	Golden & Ash	Adenomatoid Tumor		W	34	0	0	0	-	-	-	3 yrs.	-	-	0	Ø	- 0
1 de	•			1 1 M																
100		14																		

Treatment	Location	Size in cms.	Shape	Color	Inflammation ? Muscle Fibers ? Lymphocytic Infil.	
0	Caput Minor	1.5	0	White)
0	0	2.6 x 1.3 x 0.8	Dumb- bell	0	⊧000 C)
0	0	2.5	: 0	Grey Pink	⊧_ _ ≭ ()
0	0	1.2	Sphere	Grey	Found at tion	opera-
0	Caput Major	0	0	0)
0	Caput Minor	1.2 x 1.6	Sphere	White	-000)
0	Caput Minor	1.0	. O	White	000 * 0)
0	Caput Minor	1.5 x 1.2	0	White	*=** ()
0	Region of Right Testic	Bean le	Bean	0		D
0	Caput Minor	0	0	0	00** 0)

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Case Number	Year Reported	Author	Author's Designation	Color	Age	Tumefaction	Pain	Incidenta1	Trauma	V. D.	Increase in Size	Duration	Tenderness	Hydrocele	Right	Left	Pre-Operative Diagnosis
30	1945	Golden & Ash	Adenomatoid Tumor	C	68			*	-	-	0	0	0	-	,	*	0
31	1945	Golden & Ash	Adenomatoid Tumor	C	22	*	*	-	•	-	0~	2 wks.	*	-	-	*	0
32	1945	Golden & Ash	Adenomatoid Tumor	W	38	*		۲	-	-	*	5 yrs.	*	-	0	0	0
33	1946	Morehead	Angiomatoid Tumor	W	62	-	-	*		-	-	0	-	-	0	0	0
34	1946	Sworn Marshall Edwards	Adenomatoid Tumor	0	34	*	-	-	-	-	*	Sev. yrs.	-	0	*		0
35	1946	Sworn Marshall Edwards	Adenomatoid Tumor	0	43	*	-	-			*	Sev. yrs.	-	*	-	*	Spermatocele
36	1946	Bothe Cristol Devers	Fibroma	0	42	*	4	-	*	-	-	13 yrs.	-	-	-	*	0
37	1946	Codnere Flynn	Adenomatoid Tumor	W	26	*	đ	-	÷	-	*	10 yrs.	-	-	-	*	Adenomatoid Tumor
38	1946	Codnere Flynn	Adenomatoid Tumor	W	33	*	-	-	-	-	0	7 yrs.		0	0	0	Neoplasm of Epididymis

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Treatment	Loca ti on	Size in cms.	Shape	Color	Encapsulated 7	Inflammation 7	Muscle Fibers?	Lymphocytic Infil.	Comments
0	Caput Minor	0	0	Yellow	*	0	*	0	0
0	Caput Major	0.5	Oval	Pink Grey	*	0	0	0	Round Cell In filtration
0	Caput Minor	1.0	0	0	*	0	*	*	0
Epididymeetomy	Caput Minor	1.5 x 1	0	0	*	-	*	*	Eosinophils & Plasma Cells Present
Resection	Caput Minor	2•2 x 2•0	0	Yellow White	*	0	*	*	0
Resection	Caput Minor	1.7 x 1.25	0.	Yellow White	*	0	*	*	0
Orchiectomy	Caput Minor	5.0	Oval	0	*	0	*	*	0
Epididymectomy	Caput Major	1.8 x 1.2	Globular	G rey White	0	0	0	0	0
Epididymectomy	Caput Major	2.5 x 1.0	Hemi- sphere	G rey White	*	0	0	*	0

Case Number	Year Reported	Author	Author's Designation	Color	Age	Tume fa ction	Pain	Incidental	Trauma	V. D.	Increase in Size	Durati on	Tenderness	Hydrocele	Right	Left	Pre⊷Operative Diagnosis
39	1946	Codnere Flynn	Adenomatoid Tumor	W	47		-	*	-	-	-	0	•••	*	*		Hydroc ele Inquinal Herr
40	1947	Patterson Mogg	Mesothelioma	0	0	*	-	-	*	-	*	l yr.	-	-	*	-	0
41	1947	Beneventi	Adenoma toid Leiomyoma	0	3 8	*	-	-	*	*	*	2 yrs.	-		-	*	0
42	1949	Wilson	Adenomat oid Leiomyoma	0	46	*	-	-	-	-	-	3 mos.	-	-	-	*	0
43	1950	Wyatt Khoo	Adenomatoid Tumor	W	56	0	0	0	0	0	0	0	-	0	*	-	Malignant Tes cular Tumor
74	1950	Wyatt Khoo	Adenomatoid Tumor	W	45		-	*	0	0	0	0	0	0	0	0	0
45	1950	Glaser	Lymphangioma	0	37	*	*	-	*	-	*	6 mos.	*	0	*	-	Resolving In- flammation
46	1950	Burros Maycock	Adenomatoid Tumor	W	5 mo	*	-	-	-	-	*	5 mos.	0	0	*	-	0
47	1951	Folk Konwaler	Adenomatoid Tumor	C	38	-	-	*	-	*	*	2 mos.	*	-	*	***	Ca of Testicl Acute Epididy mitis

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	Tres tment	Locati on	Size in oms.	Shape	Color	Encapsulated ?	Inflammation ?	Muscle Fibers ?	Lymphocytic Infil.	Comments
8	Epididymectomy	Caput Minor	0.8 x 0.6 x 0.5	Globular	Grey White	0	0	0	*	0
	Epidid ymeetomy	Caput Major	2.5	Sphere	Cream	*	*	0	*	Eosinothils & Plasma Cells Present
	Excision	Caput Minor	2.0 x 2.0 x 1.5	0	0	0	0	*	0	0
	Exci si on	C a put Minor	2.0	0	White	*	0	*	*	0
i-	Epididymectomy Orchiectomy	Caput Minor	3.5	Round	White Brown	0	0	*	*	Round Cell Infil- tration Basement Membrane
	0	Caput Minor	1.0	0	White	0	0	*	*	Found At Autopsy Round Cell Infil- tration Basement Membrane
	Excision	Caput Minor	Pea	Pea	0	0	Ò	*	*	Calcification Present
	Epididymectomy Orchiectomy	0	2 x 1 x 1.5	Globular	Grey	0	0	*	0	Youngest Patient Recorded
) x	Epididymectomy Orchiectomy	Caput Major	1.0	0	Yellow White	-	0	0	*	Neutrophils & Eo- sinothils & Plasma

8a

Case Number	Year Reported	Author	Author's Designation	Color	Age	Tumefaction	Pain	Incidental	Trauma	V. D.	Increase in Size	Durati on	Tenderne ss	Hydrocele	Right	Left	Pre-Operative Diagnosis
48	1951	Meeter Schwartz	Adenomatoid Tumor	0	32	*	-	-	-	0	*	3 yrs.	*	-	*		0
49	1951	Meeter Schwartz	Adenomatoid Tumor	0	60	*	-	-	0	0	*	5 yrs.	-	0	*	-	0
50	1951	Longo	Adenomatoid Tumor	0	31	*		-	0	0	0	6 yrs.	-	*	-	*	Spermatoce
51	1951	Longo	Adenomatoid Tumor	0	49	-	-	*	0	0	0	0			-	*	Tumor, Tul culous Ep: dymitis
52	195 1	Longo	Adenomatoid Tumor	0	30	*	-	-	0	0	0	10 yrs.	-	-	-	*	Testicula: Tumor
53	1951	Longe	Adenomatoid Tumor	0	44	*		—	0	0	0	5-6 yrs.	-	-	ŧ	-	Tuberculo: Epididymi [†]
54	1951	Longo	Adenomatoid Tumor	0	29	-	-	*	0	0	0	0	-	-	*	-	Tuberculou Epididymi Spermatoce
55	1951	Longo	Adenomatoid Tumor	0	54	*	-	-	0	0	0	4 yrs.	*	-	*	-	Testicula: Tumor
56	1951	Longo	Adenomatoid Tumor	0	46	*	-	-	0	0	0	1.5 yrs.	*	-	*	-	Epididymi 4

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	Treatment	Loostion	Size in cms.	Shape .	Color	Encapsulated i	Inflammation ?	Muscle Fibers?	Lymphocytic Infil.	Comments	
	Epididymectomy	0	5	Cylindri. cal	- G rey	*	0	*	*	Alive & well 4 years later.	
	Epididyme ctomy	Caput Minor	2.5 x 2.0 x 1.7	0	G rey Pink	*	0	0	*	Alive & well 1 year later. As- sociated inquina hernia	
le	Excision	Caput Minor	2 x 1.5	0	Grey White	*	0	*	*	Alive & well 26 years later.	
er- di-	Excision	0	2 x 1.5	0	Grey White	*	0	*	*	Died 3 years lat Cause unknown.	er
	Excision	Caput Minor	2 x 2	0	Yellow	*	0	*	*	0	
s is	Epididymectomy	Caput Minor	1.5 x 1.5	0	White Grey	*	0	*	*	Alive & well 17 years later.	
s is le	Orchiectomy	Caput Minor	1 x 1	0	Yellow	*	0	*	*	Alive & well 15 years later.	
	Orchiectomy	Caput Minor	1 x 1	0	Yellow	*	0	*	*	Alive & well 14 years later.	
is	Excision	Caput Minor	1 x 1	0	Grey White	*	0	*	*	Alive & well 11 years later.	

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Case Number	Year Reported	Author	Author's Designation	Color	Age	Tumefaction	Pain	Incid ental	Trauma	V. D.	Increase in Size	Duration	Tenderness	Hydrocele	Right	Left	Pre-Operative Diagnosis
57 3	1951	Longo	Adenoma toid Tumor	0	49	*	-	-	*	0	0	16 yrs.	*	*	*	-	Tuberculo Epididymi Hematoma
58 :	1951	Longo	Adenomatoid Tumor	0	50	*	-	-	0	0	0	7 yrs.	*	-	*	-	Benign Ne plasm of didymis
59	1951	Longo	Adenomatoid Tumor	0	42	*	-	-	0	0	0	2 yrs.	*	-	-	*	Benign Ne plesm of didymis
60	1951	Longo	Adenomatoid Tumor	0	34	-	-	*	0	0	0	0	-	-		*	Benign Ne plasm of didymis
61	1951	Longo	Adenomatoid Tumor	0	58	-	-	*	0	0	0	2 yrs.	-	-	-	*	Cyst, Epi dymitis
62	1951	Longo	Adenomatoid Tumor	0	60	-	-	*	0	0	0	0	-	*	*	-	Tuberculc Epididymi
63	1951	Longo	Adenomatoid Tumor	0	61	*	-	-	0	0	0	l.5 yrs.	-	-	-	*	Testicula Tumor
64	1951	Longo	Adenomatoid Tumor	0	56	*	-	-	0	0	0	6 yrs.	*	*	*	-	Spermatoc
65	1951	Longo	Adenomatoid Tumor	0	27	*	-	-	0	0	0	7 yrs.	*	-	*	-	Tuberculc Epididymj
66	1951	Longo	Adenomatoid Tumor	0	42	*	-	-	0	0	0	4 yrs.	*	*	-	*	Tuberculc Epididymi

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	Treatment	Location	Size in cms	Shape	Color	Encapsula ted ?	Inflammation ?	Muscle Fibers ?	Lymphocytic Infil.	,		Comments	
us tis	Excision	C a put Minor	2.5 x 2.5	0	G rey White	*	0	*	*	Alive years	å la	Well ter.	8
o- Epi-	Epididymectomy-	-Caput Minor	3 x 3	0	Grey White	*	0	*	*	Alive year 1	å Lat	Well ;er	1
o- Epi-	Excisi on	Caput Major	2 x 1.5	0	Yellow	*	0	*	*	Alive years	å ls	Well	4
e- Epi-	Orchiectomy	Caput Major	2 x 1.5	0	G rey White	*	0	*	*	Alive years	å ls	Well	4
.di-	Excision	0	1.5 x 1.5	0	White	*	0	*	*	Alive years	å 1a	Well ter	4
us .ti s	Orchiectomy	Caput Minor	3 x 3	0	Yellow	*	0	*	*	Alive years	& 18	Well ter	2
(1°	Orchiectomy	0	3•5 x 3	0	White	*	0	*	*	Alive year	å lete	Well pr	1
ele:	Excisi on	0	0.7 x 0.7	0	0	*	0	*	*			0	
us .tis	Excision	Caput Minor	2.5 x 2.5	0	White	*	0	*	*	Alive years	å le	Well ter	20
us tis	Epididymectomy	Caput Minor	3 x 2	0	White Grey	*	0	*	*	Al iwe years	& 10	Well ater	26

10a

Case Number	Year Reported	Author	Author's Designation	Color	Age	Tumefaction	Pain	Incidental	Trauma	V. D.	Increase in Siz	Durati on	Tenderness	Hudrocele		Right	Left	Pre-Operative Diagnosis
1	1953	Schenken	Adenomatoid Tumor	0	0	0	0	0	0	0	0	0	0	0)	*		0
2	1953	Schenken	Adenomatoid Tumor	W	59	*	-	-	*	-	*	18 yrs.	*	*		*	-	Testicula: Tumor
3	1953	Schenken	Adenomatoid Tumor	W	41	*	-	-	-		*	l yr.	-	-		0	0	Tumor of t Epididymia
4	1953	Schenken	Adenomatoid	W	32	*	-	-		-	*	4-5 Vrs.	*	0)	*	_	Testicular Tumor

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ABLE	ea tanent	cation	ze in ôms.	• • •	lor	capsulated?	flammation 7	scle Fibers?	mphocytic Infil. ?	9 · · ·	mnents	
		Lo	ŝ	Shinks	CO	ů H H	In	Mu	Ly	•	CO	
	Excision	Caput Minor	1.5	Round	Pearly Grey	*	-	*	*		0	
,	Excision	Caput Minor	3.0	0	Grey White	*	0	0	0	Patient Well to	Alive Date	\$
he	Excision	0	1.2	Nodular Irregu- la r	0	0	0	*	*		0	
•	Orchiectomy	Caput Minor	2.5	Globular	White Grey	*	0	*	*	Alive & years la	Well ater	15

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CLINICAL FINDINGS

The specific diagnosis of this lesion is impossible without microscopic examination. There are, however, certain findings in the history and physical examination that form somewhat of a pattern.

Chief Complaint

The chief complaint in all cases in this series has been either pain, tumefaction, or a combination of both. The combination of both pain and tumefaction as the presenting complaint was found in about eight per cent of the sixty cases reporting a chief complaint. Tumefaction is the most common complaint in thirtyeight cases, or about sixty-three and one-half per cent. In only two of the cases reviewed was pain a presenting complaint; however, in one of these the pain was incapacitating. The remaining twenty-five per cent of the lesions were discovered on routine examination, or examination for other complaints. There was one case found at autopsy.

Age

This tumor has been found at both extremes of life. The age ranging from an infant of five months, to a retired coffee importer of seventy-eight. The average age in this series was approximately forty-two years.

Race

This tumor has been reported almost exclusively among the white race. However, there have been two cases reported in negroes and one case reported in a Chinese.

Duration

The average duration in this series was six and two-tenths years. The extremes were from thirty-eight years to two weeks.

Size

The intra-scrotal tumor mass may range from five tenths centimeters to nine by five centimeters in diameter, averaging about two and six-tenths centimeters in its greatest diameter. While this is a slow-growing tumor, in about sixty-two per cent of the thirty-nine cases reporting, there was a history of slow progressive increase in size between the period of discovery and treatment.

Relation of Duration to Size

There is some relationship between the duration and the size of the tumor, although it does not seem too significant. The tumor of longest known duration attained a size of three and one-half centimeters, which is a relatively large tumor of this type. In

contrast to this, the largest tumor, nine by five centimeters, had a <u>know</u> duration of only eleven months, while one of the smallest tumors of the series, seven-tenths of a centimeter, was known to the patient for six years. Palpation

Usually the finding of an intra-scrotal tumor mass is the only significant abnormality. This mass is almost always well-circumscribed and very hard to palpation. Tenderness is present in about thirty-nine of the sixtytwo cases reporting; however, considering the region, this would be a hard finding to evaluate.

Shape

The shape of the tumor has been reported as being globular, hemispheric, oval, cylindrical, round, and dumbbell shaped. In this series, the globular form prevails.

Location

There is no predelection for either side of the body, for of the cases reporting, there are twenty-six right-sided lesions, and twenty-nine left sided lesions. As of this date, there have been no bilateral cases reported. The tumor is reported in three locations, the head of the epididymis (globus major), the tail of the

epididymis (globus minor), and the less specific location, the region of the testicle. Of the fifty-two cases reporting, about eighty-eight per cent are found in the region of the tail of the epididymis.

Hydrocele

A relatively frequent associated finding with this tumor is a hydrocele. In fifty-six of the cases reporting, a hydrocele was found in twelve cases, or in about twenty-two per cent of the cases; two of these cases were bilateral.

Pre-Operative Diagnosis

In the case of a lesion such as this, a pre-operative diagnosis is made on the basis of the clinical findings. In this series this tumor has been diagnosed preoperatively as:

> Tuberculous epididymitis..8 cases Testicular Tumor......8 cases Epididymal Tumor.....7 cases Chronic epididymitis....6 cases Hydrocele.....5 cases Spermatocele....4 cases Hematoma....1 case Cyst....1 case Fibroma....1 case Gumma....1 case Resolving Inflammation...1 case Adenomatoid Tumor....1 case

There were only thirty-seven cases in the sixty-six that reported a pre-operative diagnosis.

Although it would be folly to lay much, if any,

importance to the pre-operative diagnosis, it does serve to point out the infrequency of consideration of this lesion. This neglect becomes quite significant when you consider that this lesion makes up fifty-three per cent of all primary neoplasms of the epididymis, as reported by Longo (3) in 1951.

PATHOLOGY

Gross

Grossly the adenomatoid tumor is a small tumor averaging about 2.6 centimeters in its greatest diameter. As has been previously stated the extremes in this series range from 0.5 centimeters to 9 by 5 centimeters. It is found about seven times more frequently in the region of the lower pole of the epididymis as any other location. The tumor is usually globular, but is reported as being oval, cylindrical, round, dumbbell shaped, and hemispheric. It is not invasive, but discrete and in most cases, easily shelled out of surrounding tissue. Forty authors report the tumor as being encapsulated, against two that report it as not being encap-The tumor is firm to feel and on cut section sulated. as Longo (3) states, often has the appearance of an uterine fibroid. Some of the tumors reported by Lee (4) bulged when sectioned. One tumor reported by Golden and Ash (2) had a finely nodular cut surface. Typically it is grayish-white in color, but has been reported as cream, pinkish gray, white and light brown, yellowish white, bluish white, and white. Usually the tumor has a homogenous appearance with no areas of degeneration. However, Falk and Konwaler (5) report seeing areas of necrosis, and Glaser (6) reports finding calcification.



Gross photograph showing firm homogeneous pearly gray tumer nodule in the epididymis, with sharp demarcation from the testis.

PLATE I

PATHOLOGY

Microscopic

In the microscopic structure of this tumor there is a good deal of variation between the proportional amounts of fibrous tissue and gland-like spaces. The fibrous tissue itself varies from a loose collagenous meshwork to a dense, sometimes hyalinized fibrous stroma. This stroma has acidophilic staining properties. At the periphery of the stroma muscle fibers are often encountered. Longo (3) states that muscle fibers are always present; however, in this series only thirty-three authors reported this finding. It is generally agreed that these muscle fibers are victims of incarceration by the expanding tumor rather than the proliferation of primary elements.

The gland-like spaces are found dispersed among the fibrous. cords. They are quite variable and may run in many directions, even in a single microscopic field. The spaces vary in structure from almost solid cords of low cuboidal epthelium to very large greatly dilated spaces. The large dilated spaces are lined with flattened epithelial cells. Although there is usually a preponderance of one type of these spaces, both types can always be found.

PLATE II



Photomicrograph showing cuboidal epithelicid cells lining spaces resembling glandular structures. It is this type of microscopic structure that may be confused with an aden ocarcinoma. The cells of these gland-like spaces are characteristic and peculiar to this tumor. Most of these cells are composed of vacuoles of various sizes. Some of the vacuoles are exceedingly large. Cells with the extremely large vacuoles have a signet ring appearance, due to the peripherally placed nucleus. Vacuoles are most frequently found in flattened cells which are sharply demarcated. The non-vacuolated cells are usually found in the cords of cuboidal and low columnar cells. The non-vacuolated cells have a finely granular acidophilic cytoplasm. These cell types are regarded as the primary units of structure.

It has been speculated that the gland-like spaces just described are formed by fusion of the large vacuoles. This is supported by the finding of shreds of cytoplasm still present along the free border of the spaces. In the markedly vacuolated cells, only very thin cytoplasmic strands connect the cells, but still they are connected.

The lumina of the gland-like spaces contain no elements of blood, lymph or any material that lends itself to any known stain. Infrequently mononuclear cells are seen in the lumen and are considered products of desquamation. The question of glycogen, fat, and





Photomicrograph showing numerous irregular spaces lined with flattened epithelioid cells showing large vacuoles. In some of the vacuoles there are desquamated monounclear cells. There are cytoplasmic shreds present along the free border of the spaces in several ofthe vacuoles. mucin content, was partially settled by Lee (4) when he stained fresh unfixed tissue with Bauer-Feulgen's glycogen stain, Sudan IV fat stain, and Mayer's mucicarmine stain with the Dresbach modification. The stains for fat and glycogen were negative. The stain for mucin was very weak and inconstantly found; this is regarded as highly questionable. It is of interest that Lee (4), who considers this tumor a mesothelioma, used the Mallory-Heidenhain stain, with negative results. This stain, when positive, colors the cells of the tubules in the kidney red.

Scattered foci of lymphocytes are typically seen near the periphery of the tumor. They can also be seen in the interstitial tissue, but not nearly as frequently. An occassional monocyte is not considered uncommon, but plasma cells, neutrophils and eosinophils are not typically seen. Falk and Konwaler (5) report seeing these non-typical cells in an adenomatoid tumor. Patterson and Mogg (7) report finding eosinophils and plasma cells, but no neutrophils. Wyatt and Khoo (8) report round cell infiltration, as does Golden and Ash (2) in one of the cases they report.

The majority of authors do not think there is a true basement membrane present in this tumor. However,

PLATE IV



Photomicrograph showing characteristic lymphocytic accumulations among the gland-like spaces in an adenomateid tumer of the opididymis. Wyatt and Khoo (8) have reported finding a basement membrane present in two of their cases; Hinman and Gibson (9) report this finding in one case.

Although no brush border could be demostrated by Golden and Ash (2) by the phosphotungstic acid hematoxylin stain, Longo (3) presented in his article a photomicrograph clearly showing a brush border. At present, therefore, it must be considered an inconstant finding.

PATHOGENESIS

Introduction

The fact the histogenesis of this tumor is in question, is evidenced by the wide variety of names applied to it. There are, in fact, four main theories concerning its pathogenesis, they are: Epithelial, Endothelial, Mesothelial, and Mesonephric. For clarity, each of these theories will be discussed separately.

PATHOGENESIS

Epithelial Theory

The authors holding this theory have given the tumor various names according to what they considered the most important microscopic finding. Among the terms used are: adenoma, adenofibromyoma, adenomyoma, fibromyoadenoma, adenocarcinoma Grade I, and finally adenomatoid tumor of the epididymis.

Adenoma is the name given the tumor by Blumer and Edwards (10), and Gordon-Taylor and Ommaney-Davis (11). For because of the gland-like spaces, they consider the tumor principally a glandular tissue, and essentially benign.

There are those authors who consider this tumor chiefly an admixture of fibrous tissue and smooth muscle, while still not disregarding the presence of the gland-like spaces. Among these authors are: Sakaguchi (1), adenofibromyoma; Fischer (12), myoadenofibroma; Wilson (13), adenomatoid leiomyoma. There are other authors that recognize the fibrous tissue and smooth muscle as being the chief tissue, but do not regard the gland-like spaces as being of epithelial origin.

Thompson (14) regards the gland-like structures as the most important structures and designates the tumor

as an adenocarcinoma, Grade I, indicating a low degree of malignancy. Thompson (14) is the only author to definitely report this tumor as malignant. This. however, was reported in 1936, and the history of this tumor since that time has shown that this diagnosis is not well substantiated, for there is no record of invasion or metastases from such a lesion. Of considerable interest in this matter is Dr. Ewing's statement concerning such a tumor sent to him by Hinman and Gibson (9) in 1924. The following is a personal communication from Dr. Ewing to the latter. "Structurally, your tumor of the epididymis is an adenocarcinoma, being composed of widely scattered acini lined by flat or often cuboidal epitheloid cells in which nuclei and nucleoli are promiment. Many of the acini are widely dilated and the lining cells flattened so that these portions recall a lymphangioma. The general structure recalls that of the rete testis, but the tumor seems to have been entirely separate from the testis, so that an origin from the rete is not easily adjusted. That it has some malignancy is shown by the infiltration of the skin, but I should not regard it as very malignant. I am quite unable to reach any conclusion regarding the origin of the tumor. Some years ago I saw a very similiar growth removed from the

cord, and I concluded that it was a lymphangioma, but this conclusion can hardly be regarded as satisfactory. The occurrence of such a tumor suggests to me a search for aberrant canals of embryonal origin in its locality which might give rise to such a tumor. This region abounds in peculiar structures of undetermined nature, which might give rise to tumors. Of aberrant canals from the rete I know nothing. The possibility of an origin from lymph canals is perhaps worth considering, but lacks definite support." (9)

Dr. Bloodgood was consulted by Hinman and Gibson (9) about this same tumor, and the following is his personal communication. "The tumor is composed of acini lined by cells of the epithelial type; the cells sometimes proliferating, and in a few instances solid. There is no definite basement membrane, and this adenomatous tumor extendsup to the epidermis of the scrotum. This growth of adenomatous tissue growing up to the epidermis favors malignancy. The complete absence of cells in many places in the basement membrane and here and there nest of cells as we see in cancer favor malignancy. I have never seen a tumor of this kind in the epididymis, but if this were in the breast, and the tumor extended to the skin, I would treat it as cancer.

If this tumor would recur or metastasize, it would settle the diagnosis. If it does not, then we do not know. I also notice here and there papillary growths so that we have cystic adenoma, papillary cystadenoma, and cancer." (9)

Neither of these noted authors place a definite diagnosis for the tumor, nor do they have a definite statement as to the origin. However, Ewing gives considerable import of the consideration of "aberrant canals of embryonal origin in this region." (9)

Epididymyomata is the name given the tumor by Baille (15). He reports seeing in one of his specimens every stage of transition from the gland-like structures or acinar groupings to the adult epididymis. As his designation implies, he feels that this tumor arises from the adult epididymis. Many authors report seeing normal adult tubules of the epididymis near the periphery of the tumor, but there has been no other report of the transitional states.

Golden and Ash (2) consider the cohesiveness and presence of vacuolation in the neoplastic cells good evidence of epithelial origin. They regard the smooth muscle as inclusion of pre-existing muscle involved in an expanding tumor. However, having no definite con-

victions as to the origin of the tumor, they proposed the term, "Adenomatoid." They consider this term morphologically correct and genetically neutral. Since the time this term was proposed in 1945, the majority of tumors of this type have been reported under its heading.

Lee (4) feels that while the cells do have an epithelial-like appearance, they do not resemble the type epithelium which lines the epididymis, fallopian tubes, or any other structures normally seen in the ovary or uterus. He also points out that epitheliallike formations also arise from both mesothelium and endothelium. On the basis of microscopic finding alone, he feels that it is impossible to settle the question of histogenesis, which in the view of history seems to be correct.

PATHOGENESIS

Endothelial Theory

Authors supporting this theory usually designate this tumor as a lymphangioma; among these authors are Rigano-Irrera (16), Scalvi (17), Charache (18), and Glaser (6).

Halper, and Malioff (19) and Helpern (20) do not consider the tubules the chief microscopic feature of the tumor, but rather the fibrous tissue and the smooth muscle. However, they regard the tubules as endothelial structures and designate the tumors as mixed leiomyoma and lymphangioma.

The chief microscopic evidence supporting this theory is the presence in the tumor of tubules lined with flat enothelial-like cells, surrounded or embedded in reticular atroma. Although the cells are cuboidal and tall columnar, they are regarded as products of metaplastic conversion from the cells of endothelial lined spaces. Such conversion is seen in inflammatory and neoplastic processes. The authors contend that the primary process is that of mechanical blockage, due to trauma or inflammation with a resulting epididymitis. This is followed by blockage of the lymphatics which results in cellular proliferation distal to the block. Since most authors regard trauma and infection

as incidental findings, this theory is not widely accepted. In reviewing the series presented in this paper, there were fifty-three cases mentioning trauma, only nine or seventeen per cent of these had a positive history, and in considering the vulnerability of this region to injury, this does not seem to be a significant factor. There were three cases of veneral infection in the thirty-nine mentioning this factor, two leutic infection and one case of gonorrhea. Here again, the incidence does not warrant significance.

The contention that the cells resemble endothelial cells is disputed by Golden and Ash (2), whe contend the cells are cytologically different from endothelial cells seen elsewhere, even those seen in angioma.

Morehead (21) sees a striking resemblance between the tumor formation and the microscopic characteristics of lymphatic vessels. He recognizes the fact that the cells have the appearnce of epithelial elements, and explains it thus: Embryonic tissue destined to form vascular channels first appears as solid areas of cells in the embryo called blood islands. Small vacuolated areas appear in these islands and these coalesce to form spaces, thus forming channels. The central cells then become blood cells, and the peripheral cells endo-

thelium. However, before the cells have definite endothelial characteristics, they structurally resemble epithelium. Morehead (21) offers this as an explanation as to why the cells he considers to be endothelial appear like epithelial cells. The stages are no doubt similar, for this is a common way to embryologically form channels.

The fact that no one has ever succeeded in staining any material in the lumen of the tubules does not favor this theory, for in the lumen of lymphangiomas there is lymph or lymph-like fluid.

Some feel that the fact there are lymphocytes present in clumps about the tumor lends evidence to this theory. It is the contention that these clumps are formed from cells that migrate from the island forming the lymphatics and develop separate from the tubules.

Others feel that this tumor resembles the lymphangiomas that develop in other parts of the body and which regress by sclerosing. However, this type of lymphangioma is usually found in childhood and infancy. Therefore, one would expect a preponderance of cases in the younger age group. This is not the case, for

while there is one case in an infant of five months, the next youngest patient is fifteen years, and the average for the series is forty-one and eight-tenth years.

PATHOGENESI S

Mesothelial Theory

There are three authors that hold to this theory, namely Masson and Riopelle (22), Evans (23), and Lee (4).

Masson (22) after reviewing the six cases that he reports. feels the histological tumor pattern and characteristics of the individual cell favors the mesothelial origin. The microscopic evidence that Masson (22) offers deserves more than passing attention. There are four chief characteristics that he feels are significant: (1) Each individual cell possesses a distinct brush border; (2) The presence of a superficial cuticle; (3) The presence of supranuclear diplosomes in the terminal flagellum; (4) The shape of the cell. He feels that these are all native characteristics of the mesothelial cell. He offers more microscopic findings, which he considers similar, quite strikingly so, to the cellular alteration found in inflammatory and neoplastic lesions of the pleuroperitoneal serosa; these are considered mesotheliomas. These microscopic findings are as follows: (1) Poor delineation of cell borders; (2) The production of mucin, a fact that is disputed by many authorites and which seems very ques-

tionable; (3) The presence of mononuclear cells in the lumen of the gland-like spaces, which are most likely products of desquamation; (4) The cytoplasmic vacuolation.

While Evans supports this theory, he does not base his opinion on microscopic evidence, but on the initimate anatomical relation of the tumor to the linings of the serosa membranes. He has reported the occurence of this tumor on the serous surface of the uterus and fallopian tubes in connection with the peritoneum. He reports seeing the gland-like spaces in direct connection with the cells covering the serosa surface. The only other author reporting this microscopic finding is Lee (4), who found this condition in three cases reviewed. by him. Both authors found this lesion only on the It is, therefore, by no means a constant finduterus. ing, moreover, there have only been four cases reported. The fact that this finding is limited to tumor found on the uterus, robs it of any significance. However, he feels that it is reasonable to assume, because of the anatomical and microscopic evidence, that the tumor is related to the serosa of the tunica vaginalis whose structure is identical to that of the pelvic peritoneum and having a common origin in the mesothelial cells of the serous membrane. He also suggests the possibility,

in view of the fact the tumors are directly related to the genitalia, that histogenic factors operating are related to the potenitalities of the specialized mesothelium of the urogential ridge. However, this ridge in the embryo serves for the development of gonadal structures which are epithelial in nature.

Lee (4) maintains that on the basis of microscopic findings alone, it is impossible to settle the question of histogenesis. He excludes epithelial origin on the basis that no basement membrane can be demostrated, which is disputed by some authors. There are three that report this finding present. A basement membrane is characteristic of epithelial structures with the exception of the thyroid and the excretory passages of the urinary system; however, mesotheliomas and lymphangiomas characteristically do not possess such a membrane. Lee (4) also discounts the fact that signet ring formation is limited to epithelial elements, pointing out that vacuolated cells are found in some lymphangiomas and in an occasional case of peritoneal mesotheliomas. As Morehead (21), he feels that the formation of these channels by vacuolation is not limited to vascular elements alone. He does not attach much histogenic or diagnostic significance to the scattered infiltration of the lymphoid

cells in the stroma of these tumors as this is seen in a variety of neoplasms. He, as many others, considers the muscle fibers present to represent inculsion of a near-by structure by an expanding neoplasm. This is supported by the fact that muscle fibers are found almost always near the periphery. As stated previously. only Lee (4) and Evans (23) report continuity between the cells lining the spaces and those of the serosa: however, this type of tumor is found only in the region of the uterus. The anatomical proximity of the tumor to the tunica vaginalis is significant and Lee (4) feels that it strenghtens the mesothelial theory. However, he is aware that the mesotheliomas arising from the peritoneum, pleura, and pericardium and both microscopically and clinically malignant, while this tumor is benign. He also speculates that further investigation of the urogential ridge would provide more accurate knowledge as to the origin of this lesion.

A very strong arguement against this theory is presented by Longo (3). He discounts this theory on the basis that mesothelial lined spaces are very extensive, and this type of tumor has not been seen in any region except along the embryonic course of the mesonephric elements.

PATHOGENESIS

Mesonephric Theory

Schiller (24), Condere and Flynn (25), Falconer (26), and Longo (3), are the authors supporting this theory.

Schiller (24) was the first to suggest that this tumor was specifically derived from misplaced fetal remnants of the mesonephros. He presented his paper in 1942. It was his contention that the gland-like spaces were glomerulus-like structures. However, Wyatt and Khoo (8), felt that a complete study failed to reveal any common histological fundamentals in both the tumor and mesonephric structures.

Codnere and Flynn (25) think this theory is supported by the tenet that the caput major of the epididymis contains different ductules derived from the cranial group of mesonephric tubules, while the caudal group persists as aberrant ductules and vestigial remnants in the lower portion of the epididymis. This lower part of the epididymis is seven times the most frequent site for the tumor to be found.

Longo (3) would like to reconcile the two most prevalent theories of origin, epithelial and mesothelial, under the term, hamartoma of the mesonephros, for most

recent authors agree that the tumor is not endothelial. Considering the histologic structure in light of the anatomical developmental distribution of the mesonephric ducts, he sees a striking connection. The mesonephric elements are seen along the course of the epididymis, tunica vaginalis, and spermatic cord, which is the very region in which the tumor is found. In the female the mesonephric elements are found along the course of the ovary, the Fallopian tubes, and posterior part of the uterus, and this, again, is the exact region in which this tumor has been found. The very fact that this is a similar region of embryonal growth in both the male and female in light of the constant location of this tumor is very significant. The embryologic evidence which Longo offers is also very significant for as he states, the mesonephros with all of its collecting system contains all elements known to exist in the tumor, including smooth muscle.

This second definite embryologic kidney consists of blood vessels and the wolffian duct associated with a series of tubules. The smooth muscle anlagen is present in this tumor and forms an integral part of all adenomatoid lesions. Since adenomatoid tumors and leiomyomas account for almost all neoplastic lesions of

the epididymis, Longo (3) suggests that the leiomyoma just represents a one-sided development of the same lesion. He also points out that mesothelial-lined spaces are very extensive, and since a tumor of this type is soon only along the embryonic course of the mesonephros, it is not reasonable to assume it to be pure mesothelioma. This strongly suggests the mesonephros as origin of the adenomatoid tumor.

Longo (3) feels that the best term would be hamartoma of the mesonephros which would indicate that the tumor was composed of both benign epithelial and benign mesothelial elements in which there is an abnormal mixing of these normal components.

Falconer (26), while not using the term, hamartoma, considers this tumor to be organoid in nature and to contain both epithelial and mesenchymal elements in the neoplastic pattern. He feels it is most likely derived from an embryonal malformation. The smooth muscle seen in the tumor has an architecture which is highly suggestive to him of urinary bladder wall. Because of the neoplastic nature of the tumor, he feels it developed from a "matrix organ", which in this case would be the epididymis originally formed from the transverse primordial kidney and the wolffian duct.

Accurate classification of the misroscopic structure is lacking completely. It is not typically like that of any definite tissue type seen; this, however, can be reconciled in the mesonephric theory. Since the microscopic structure of a neoplasm derived from mesonephric tissue would not be unlike that of this lesion, and in view of the fact the tumor defies comprehensive classification, there is an obvious implication.

TREATMENT

Orchiectomy was performed in about one-fifth of the cases presented in this series; however, at present all authorities agree that simple excision is completely adequate, and there is no reason to sacrifice the testicle. It is the general opinion that upon finding a tumor of the epididymis, it is best to do just a simple excision, unless the tumor is grossly malignant, for as stated by Longo (3) in 1951, about seventy-four per cent of the tumors of this organ are benign in nature. Also, in the case of this specific tumor, the value of an immediate frozen section is questioned because of the difficulty in distinguishing it from malignancies found in this region. Sections of paraffin-blocked tissue with a hematoxylin eosin stain are much easier for the pathologist to interpret and, thereby, offer much greater accuracy. Also, if the lesion is malignant, this region is easily accessible for more extensive treatment.

PROGNOSIS

While the microscopic section of this tumor does suggest that it is malignant, it is completely innocent and has never been known to metastasize. In the series presented, the longest a patient was followed was twenty-six years without any evidence of metastatic lesion, nor was there any evidence of local recurrence. In all cases reported, this tumor has been completely benign; therefore, once the tumor has been excised, it is safe for the physician to assure the patient of a complete cure.

SUMMARY AND DISCUSSION

This tumor was first described by Sakaguchi (1) in 1916. Since then it has been known by a wide variety of names, until 1945 when Golden and Ash (2) proposed the term "adenomatoid." This term was adopted by most authors, for it was morphologically correct and genetically neutral. It is a rather rare lesion; there are less than one hundred cases reported. It is important because it appears microscopically malignant and is clinically completely benign.

The clinical findings are particularly outstanding. The patient is most usually white and about forty years of age; however, this ranges from infancy to extremely old age. The most common presenting complaint is tumefaction, usually not associated with pain. Pain itself is very seldom the chief complaint, but the patient does present with both as chief complaint in about eight per cent of the cases. About one-fourth of all cases are detected on routine physical examination. In about seventeen per cent of the cases, there is a history of trauma; however, considering the vulnerability of this region, this is not significant. History of venereal infection is very uncommon and is likewise not significant. The average period of known duration

is about 6.2 years. During this time about sixty-two per cent of the patients report slow progressive increase in size.

Physical examination usually reveal no significant abnormality other than the intra-scrotal mass. This mass may range from 0.5 centimeters to 9 by 5 centimeters in diameter; but is usually about 2.6 centimeters in its greatest diameter. The mass is very firm to palpation and in about one-half of the cases is tender. There is no precelection for either side of the body, and there is no known case of the tumor being bilateral. It is about seven times as frequent on the lower pole of the epididymis as in any other location. In about twenty per cent of the cases a hydrocele is an associated finding, and on rare occasions this finding is bilateral.

Grossly this is a small tumor of the size range stated above. It is firm and on cut section is most often grayish-white, but has been reported tinged with pink, blue, and yellow. The cut surface is usually smooth, having the appearance of the cut surface of an uterine fibroid. It has, however, been reported as being finely nodular, and at times bulging. It is most often encapsulated.

Microscopically this tumor is an admixture of fibrous tissue and gland-like spaces. The fibrous stroma may vary from hyalinized stroma to the loose collagenous meshwork. This fibrous tissue has acidophilic staining properties. Interjected between the cords of fibrous tissue are the gland-like spaces. These vary from almost solid cords of cuboidal and low columnar cells to widely dilated spaces in which the cells are flattened. Most cells are vacuolated, and the ones that are markedly so have a signet ring appearance due to the eccentrically placed nucleus. The vacuolated cells are found most often in the dilated. gland-like spaces, while the non-vacuolated cells are found in the low columnar and cuboidal regions of the gland-like spaces. The cells just described are regarded as primary units of structure. The lumen of the gland-like spaces contain no material that will lend itself to a known stain; there are, however, an occasional mononuclear cell seen, which in all likelyhood is the product of desquamation. Muscle fibers, victims of incarceration, are seen near the periphery. Also near the periphery are scattered foci of lympho-These may also be seen in the interstitial tiscytes. sue, but not nearly as frequently. No other cell types

are typically seen; there have been, however, a few reports of inflammatory cell infiltration. It is generally agreed there is no true basement membrane, and no connection of the gland-like spaces to the serosa surfaces. There have been, however, reports to the contrary in both instances. The brush border is likewise in question, reportedly not present by one author, and pictured in a photomicrograph by another.

There are four theories concerning the genesis of this tumor: Endothelial, Epithelial, Mesothelial, and Mesonephric. While there is evidence supporting all four of these theories. the genesis is best explained by the Mesonephric Theory. The chief evidence supporting this theory is as follows: (1) This tumor has been discovered only along the embryonic course of the mesonephros, and in both male and female. (2) The mesonephros contains all tissue elements necessary for the production of this tumor. (3) The tumor is seven times as frequent in the region of the globus minor where the aberrant ductules of the mesonephros persist. (4) The microscopic structure which has confused renowned pathologists is consistent with what would be found in a tumor which developed from mesonephric tissue and is not adequately explained by any of the germ layer theories.

The diagnosis is made microscopically. It is best to use sections cut from paraffin blocks and stained with the heamotoxyalin-cosin stain.

Since the majority of lesions of the epididymis are benign, the best treatment is simple excision, unless the lesion is grossly malignant. If after microscopic examination, the tissue is malignant, the region is easily accessible for further surgery.

The prognosis in the case of this neoplasm is most excellent. There has never been metastases, and it does not recur locally after complete excision. The cure is complete.

CONCLUSIONS

The adenomatoid tumor of the epididymis is a rare lesion, and its importance lies in the fact it may be confused with a malignancy which carries with it a death sentence. In this manner it may cause needless mental anguish and even financial loss.

Trauma and infection have no significance in the production of this tumor; it is not inflammatory in origin.

The diagnosis is best made from tissue stained with hematoxylin-eosin, with sections cut from paraffin blocks. The frozen section technique is not adequate.

The lesion is best treated by simple excision. No radical procedure is indicated.

The prognosis is most excellent for there has never been a case with metastases, nor has there ever been local recurrence. The cure is complete.

The genesis is best explained by the Mesonephric Theory, which postulates this tumor develops from aberrant ductules of the mesonephros which persist in the globus minor. The strongest point for this theory is that this lesion has never been found in any location except along the embryonic course of the mesonephros.

Since all the evidence as to genesis is still only circumstantial, and because of the general acceptance of the term, "adenomatoid," it seems wise to retain this term for reasons of uniformity and clarity.

This paper has been presented with the hope of reminding future physicians of this lesion and its importance.

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