

Review

Solitary Fibrous Tumor of the Adrenal Gland: A Comprehensive Review

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Abstract

Solitary fibrous tumors (SFTs) are rare mesenchymal neoplasms characterized by a broad spectrum of anatomical localizations and variable biological behavior. Although initially described in the pleura, they can arise almost ubiquitously, including in endocrine glands. Nevertheless, adrenal involvement represents an exceptionally rare localization. Most adrenal SFTs (ASFTs) are non-functioning tumors detected incidentally. Radiological findings are generally non-specific or indeterminate and may overlap with those of other adrenal neoplasms, including malignancy, often leading to surgical resection to achieve pathological characterization. Definitive diagnosis relies on histopathological evaluation, where nuclear STAT6 expression—reflecting the pathognomonic *NAB2-STAT6* gene fusion—serves as the diagnostic gold standard. Complete surgical resection is considered the treatment of choice. Although most ASFTs appear to have an indolent course, their biological behavior remains unpredictable, and long-term follow-up is advisable.

Keywords: solitary fibrous tumor; SFT; adrenal gland; adrenal incidentaloma

1. Introduction

Solitary fibrous tumors (SFTs) are rare mesenchymal neoplasms characterized by variable anatomical localization and heterogeneous biological behavior. Formerly classified as hemangiopericytomas, they comprise a histologic spectrum of fibroblastic mesenchymal neoplasms that arise from serosal membranes and deep soft tissues [1]. Initially described as pleural tumors in the early twentieth century, these lesions were historically considered primarily thoracic neoplasms [2]. Subsequent studies demonstrated that SFTs may arise in virtually any anatomical site, including soft tissues, visceral organs, the central nervous system, and endocrine glands [3–5]. Nevertheless, the adrenal gland represents an exceptionally rare site of origin, with only a few cases reported in the literature worldwide, mainly as anecdotal reports or isolated cases among surgical series of adrenal and genitourinary system neoplasms. Consequently, much of the current knowledge about adrenal SFTs (ASFTs) is derived indirectly from similar tumors occurring at other anatomic sites. Since ASFTs are typically asymptomatic and non-functioning, they present as adrenal masses discovered incidentally during imaging studies. Current clinical practice guidelines issued by the European Society of Endocrinology (ESE) in collaboration with the European Network for the Study of Adrenal Tumors (ENSAT) recommend a systematic



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evaluation of adrenal incidentalomas aimed at excluding both hormonal hypersecretion and malignancy [6]. Within this diagnostic framework, ASFTs are often encountered as non-functioning, atypical adrenal lesions with indeterminate imaging characteristics, posing a diagnostic challenge for endocrinologists, radiologists, and pathologists. Definitive diagnosis relies on surgical resection with histopathological evaluation supported by immunohistochemistry, since histomorphology may overlap with other adrenal spindle cell tumors. Specifically, nuclear STAT6 expression reflecting the NAB2–STAT6 gene fusion is considered the most specific marker for establishing a differential diagnosis [7]. Complete surgical excision is considered the treatment of choice. Although most ASFTs appear to have an indolent course, their biological behavior remains unpredictable, and long-term follow-up is advisable [8].

The aim of this review is to summarize the currently available evidence regarding ASFTs, focusing on epidemiology, clinical presentation, diagnostic work-up, pathological and molecular features, therapeutic management, and clinical outcomes.

2. Materials and Methods

A literature review was conducted to identify published reports describing ASFTs.

A systematic search of the biomedical literature was performed using the PubMed, Google Scholar, and Scopus databases from 1994 to January 2026. The following keywords were used in various combinations: solitary fibrous tumor, hemangiopericytoma, adrenal gland and adrenal tumor.

Additional studies were identified through manual screening of reference lists of relevant publications. Reference management was conducted using Mendeley Reference Manager (Elsevier, Amsterdam, The Netherlands).

Because ASFTs are extremely rare and the available evidence consists primarily of single case reports, the collected data were analyzed using a descriptive narrative approach rather than quantitative meta-analysis. PRISMA-based methodology was not applied due to the descriptive nature of the review. This narrative review was structured in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA) guidelines.

3. Epidemiology and Risk Factors

SFTs are rare neoplasms with an estimated incidence of less than one case per million individuals per year. Although the thoracic district represents the classic site of origin, extra-pleural locations are increasingly recognized [8]. Adrenal involvement represents an extremely uncommon occurrence, with most evidence derived from anecdotal case reports or isolated cases among surgical series of adrenal and genitourinary system neoplasms [9–28]. Much of the current knowledge of ASFTs is indirectly derived from similar tumors occurring at other anatomic locations [29]. These primarily include the pleura, meninges, and pelvic or retroperitoneal soft tissues [8]. ASFTs occur predominantly in adults, typically during the fifth to seventh decades of life, with a mean age of 48 years [23]. However, one pediatric case has been described [17]. While no sex predilection has been observed for extra-adrenal SFTs, a slight male predominance has been suggested in the adrenal localization (M:F ratio, 1.5:1), although the small number of cases prevents definitive epidemiological conclusions [23]. To date, no specific environmental or genetic risk factors have been identified, and most tumors occur sporadically and in isolated form. A case of ASFT diagnosed during ultrasonographic monitoring of pregnancy raised the possibility that hormonal factors might influence tumor development and growth, as progesterone and oxytocin receptors were detected in the tumor specimen [10]. However, the clinical significance of these findings remains uncertain, as no imaging prior to gestation was available to document tumor absence or interval growth.

Table 1 summarizes the demographic, clinical, and pathological characteristics of the cases of ASFT reported in the literature to date.

Table 1. Published cases of adrenal solitary fibrous tumors (ASFTs) and related findings.

Author (Year)	Age/Sex	Side	Size (cm)	Clinical Presentation	Imaging Modality	Functional Status	Treatment	Cell Type	IHC (CD34/STAT6)	Risk Class (Demicco [30])	Outcome (Follow-Up)
Prévot S. (1996) [9]	42/F	R	NR	AI	US	NFAT	Surgery	Round	CD34+	NA	SD (5 years before surgery)
Bongiovanni M. (2000) [10]	23/F	L	9	AI	US	Pregnancy-associated elevated UFC	Surgery	Spindle	CD34+	NA	NR
Paeng MH. (2002) [11]	41/F	L	12.5	AI	CT	NFAT	Surgery	Spindle	CD34+	NA	NR
Ho YH (2010) [12]	71/M	R	15.5	AI	US/CT	NFAT	Surgery	Spindle	CD34+	NA	NR
Treglia G. (2014) [13]	33/M	R	3	FUO	¹⁸ FDG-PET/CT	NFAT	Surgery	Spindle	CD34+	NA	NR
Toniato A. (2014) [14]	54/M	Bilateral	R 15/L 4	Hypertension	US/MRI	NFAT	Surgery	Spindle	CD34+	NA	NED (18 months)
Kuribayashi S. (2019) [15]	62/F	L	10	Back pain and hypertension	CT	Synchronous contralateral PHEO	Surgery	Spindle	CD34+, STAT6+	NA	NED (24 months)
Yonli DS (2019) [16]	52/M	R	12	Lumbar pain	US/CT	NFAT	Surgery	Spindle	CD34+	Low	NED (36 months)
Gebresellassie HW (2019) [17]	13/F	R	19	Abdominal mass with pain	US/CT	NFAT	Surgery	Spindle	NR	Low	NR
Huisman SE (2021) [18]	77/M	R	8.5	AI	CT	NFAT	Surgery	Spindle/ovoid	STAT6+, CD34+	Intermediate	NR
Ambardjieva M. (2021) [19]	28/M	L	7	Abdominal and back pain; hypertension	CT/MRI	Elevated serum cortisol	Surgery	Spindle/oval	NR	Low	NED (24 months)
Campista-Jacquez (2021) [20]	71/M	L	22	NICHTI	CT	DPS (IGF2)	Surgery	NR	STAT6+, CD34+	NA	NR
Casademunt-Gras E. (2023) [21]	48/F	L	10	Lower limb paresthesia	MRI	NFAT	Surgery	Spindle	STAT6+, CD34+	Low	NR
Whaley RD. (2023) [22]	70/M	L	NR	AI	NR	NFAT	Surgery	Spindle	NR	NA	NR
Jha S. (2023) [23]	49 (mean); 7/9 M	Various	5.1 (mean)	5/9 AI	9/9 CT	NFAT (all)	Surgery (all)	Spindle (all)	STAT6+, CD34+ (all)	8/9 low (1 intermediate)	NED (median 14 months)
Shi C. (2024) [24]	47/F	L	6.9	Palpable abdominal mass	CT	NFAT	Surgery	Spindle	STAT6+, CD34+	Low	NED (8 months)
Amankwah S. (2025) [25]	35/F	R	3.7	Abdominal pain	CT	NFAT	Surgery	Spindle	STAT6+, CD34+	Low	NED (6 months)

Summary of published cases (25): mean age 48 years; slight male predominance (M:F ratio, 1.5:1); mean tumor size 9.6 cm; no laterality preference; the most common presentation is incidentaloma detected by CT or abdominal pain; the majority of tumors are non-functioning; surgical treatment was performed in all cases; no recurrence reported in most cases during the available follow-up. Notes: Cases are listed in chronological order; Jha et al. reported a series of 9 cases. Due to aggregated data, only the overall results of the series are included. Legend: M = male; F = female; R = right; L = left; US = ultrasound; CT = computed tomography; MRI = magnetic resonance; ¹⁸FDG-PET = 18-fluorodeoxyglucose positron emission tomography; IHC = immunohistochemistry; CD34 = cluster of differentiation 34; STAT6 = signal transducer and activator of transcription 6; AI = adrenal incidentaloma; NFAT = non-functioning adrenal tumor; UFC = urinary free cortisol; FUO = fever of unknown origin; PHEO = pheochromocytoma; DPS = Doeg-Potter syndrome; IGF2 = insulin-like growth factor 2; SD = stable disease; NED = no evidence of disease; NR = not reported; NA = not applicable due to case described prior to the model development or lack of histological data.

4. Clinical Presentation

The majority of ASFTs are discovered incidentally during imaging studies performed for unrelated clinical conditions. This reflects the typically slow growth and indolent clinical course of these tumors [9].

Most patients are asymptomatic at the time of diagnosis. When symptoms occur, they are usually related to the size of the tumor and mechanical compression of adjacent structures [19,21]. Large lesions may produce abdominal discomfort, flank and back pain, or may be detected as palpable masses [17,24].

Tumor size at diagnosis varies widely, ranging from small incidental lesions to large masses up to 20 cm, with a mean size of 9.6 cm. There is no laterality preference, and the contralateral adrenal gland is usually normal [23]. To date, only one case of bilateral ASFT has been described. Additionally, Kuribayashi et al. reported a unique case of ASFT with a

synchronous contralateral pheochromocytoma. Both patients underwent bilateral sparing adrenalectomy [14,15].

ASFTs are non-functioning tumors. However, biochemical abnormalities have occasionally been reported during endocrine evaluation. Two patients described by Bongiovanni et al. and Ambardjieva et al. showed elevated urinary and basal cortisol levels, respectively. However, no further endocrinological assessment, such as the dexamethasone suppression test (DST), was carried out. In the former patient, these findings were interpreted as functional hypercortisolism related to pregnancy. In the latter case, they were attributed to the compressive effect of the tumor, since hormone levels declined after adrenalectomy [10,19]. It must be acknowledged that conditions such as hospitalization and concomitant illness may lead to activation of the hypothalamic–pituitary–adrenal (HPA) axis, resulting in transient biochemical hypercortisolism [31]. These findings may complicate the diagnostic evaluation of adrenal incidentalomas and highlight the importance of careful clinical interpretation of endocrine tests [32].

Although uncommon, SFTs may be associated with paraneoplastic manifestations. Characteristic syndromes involve digital clubbing and hypertrophic pulmonary osteoarthropathy (HPO; Pierre–Marie–Bamberger syndrome) due to vascular–endothelial growth factor (VEGF) overexpression and non-islet cell tumor hypoglycemia (NICTH) related to insulin-like growth factor-II (IGF-2) overproduction (Doege–Potter syndrome) [33,34]. While the latter has been well documented in pleural SFTs, clinical presentation due to paraneoplastic syndromes is exceptional in ASFTs, with only one reported case of refractory hypoglycemia [20]. Other paraneoplastic manifestations occasionally described in ASFTs include unexplained fever and non-endocrine hypertension [13,14,19].

5. Diagnosis

5.1. Imaging

Radiological imaging plays an essential role in the detection and initial characterization of ASFTs, although imaging findings are generally non-specific and cannot reliably distinguish SFTs from other adrenal neoplasms.

On computed tomography (CT), ASFTs typically appear as well-defined solid masses without infiltrative margins. These lesions often demonstrate heterogeneous attenuation and moderate to high baseline density, reflecting a high density of collagen fibers [35]. The Hounsfield Unit (HU) values for ASFTs on CT scans can vary significantly depending on tumor composition, such as fibrous tissue content and vascular density [25]. Although specific HU ranges for ASFTs remain poorly defined, the available literature on retroperitoneal SFTs indicates that unenhanced attenuation values typically range from 30 to 50 HU [36]. Following contrast administration, tumors usually exhibit intense and heterogeneous enhancement consistent with a rich capillary network. Large tumors may contain areas of necrosis, hemorrhage, cystic degeneration, and calcifications [8].

On magnetic resonance imaging (MRI), signal characteristics are variable. Tumors are generally iso- or hypointense on T1-weighted images and display heterogeneous signal intensity on T2-weighted sequences. Highly fibrotic lesions may appear relatively hypointense on T2 scans due to dense collagen deposition. Dynamic contrast-enhanced sequences frequently demonstrate heterogeneous or progressive enhancement patterns consistent with the vascular architecture of these tumors [35].

Data on 18-fluorodeoxyglucose-positron emission tomography (¹⁸F-FDG PET) patterns of ASFTs are extremely limited. The only lesion detected by this imaging modality was described by Treglia et al., who concluded that a benign ASFT could be considered a possible false-positive ¹⁸F-FDG PET/CT finding for malignancy in the evaluation of adrenal incidentalomas [13]. However, while ¹⁸F-FDG PET is a useful tool in the diagnosis and

management of indeterminate adrenal incidentalomas, its value in the characterization of SFTs is more limited as uptake patterns appear variable and do not reliably distinguish indolent SFTs from aggressive forms [6,37]. Additionally, the role of other nuclear tracers in the evaluation of SFTs is currently unknown since only anecdotal reports of tumor detection with Carbon-11 (^{11}C -methionine) and Gallium-68 Prostate-Specific Membrane Antigen (^{68}Ga -PSMA) PET/CT scans have been described [38,39].

Within the diagnostic algorithm recommended by the ESE and the ENSAT, ASFTs are usually classified among indeterminate adrenal masses [6]. As a consequence, ASFTs pose significant diagnostic challenges from a clinical perspective, sometimes closely mimicking malignancy, and often requiring surgical resection for definitive diagnosis.

Diagnostic evaluation may be further hindered since some retroperitoneal SFTs might displace and hide the adrenal gland at imaging. Such lesions may mimic a primary adrenal tumor, prompting en bloc resection, which eventually can reveal a tumor arising from periadrenal soft tissues separately from the gland for specimen analysis [40].

5.2. Pathological and Histological Features

Definitive diagnosis of SFT relies on histopathological examination [8].

Macroscopically, these tumors are usually well circumscribed and firm, sometimes encapsulated. The cut surface typically appears whitish or tan because of abundant collagen deposition. Larger lesions may exhibit hemorrhage, necrosis, or cystic degeneration [1].

Microscopically, SFTs show a characteristic but heterogeneous morphology, encompassing a spectrum of histologic patterns ranging from spindle to ovoid or round-epithelioid neoplastic cells. The classic histological findings consist of spindle-shaped cells arranged in a so-called “patternless” architecture, with alternating hypercellular and hypocellular areas embedded within a collagen-rich stroma. One of the most distinctive histological features is the presence of branching, thin-walled vessels forming a “staghorn” vascular pattern. Tumor cells typically display oval nuclei, inconspicuous nucleoli, and scant cytoplasm, with low-to-moderate nuclear atypia. Mitotic count (Ki-67) is generally low, although increased proliferative activity may be observed in more aggressive forms [1].

ASFTs must be differentiated from multiple other benign and malignant spindle or round/epithelioid cell neoplasms of the adrenal glands (perineuriomas, schwannomas, leiomyomas, sarcomas, sarcomatoid adrenal carcinomas, and metastatic spindle-cell neoplasms). Because of this wide differential spectrum, a careful integration of morphological, immunohistochemical, and molecular findings is essential for establishing differential diagnosis [41].

5.3. Immunohistochemistry and Molecular Features

Immunohistochemistry plays a fundamental role in the diagnosis of SFTs. Conventional markers frequently expressed in SFTs include Cluster of Differentiation 34 (CD34), Cluster of Differentiation 99 (CD99), B-cell lymphoma 2 (BCL-2), and vimentin, in the absence of actin, desmin, S100 protein, or epithelial markers. However, these markers lack specificity because they may be expressed in other spindle-cell neoplasms [1].

The most reliable diagnostic marker is nuclear STAT6 expression, which is almost pathognomonic, with a sensitivity and specificity close to 100% for SFT [7]. STAT6 overexpression reflects the presence of the *NAB2-STAT6* gene fusion, which results from an intrachromosomal inversion involving chromosome 12. The discovery of the *NAB2-STAT6* gene fusion represented a major breakthrough in the understanding of SFT pathogenesis. This fusion protein leads to aberrant transcriptional activity, driving tumorigenesis. Although the *NAB2-STAT6* fusion is considered the molecular hallmark of SFT, it does not appear to correlate directly with tumor aggressiveness [42,43].

Additional genomic alterations have been investigated as potential prognostic markers. In extra-adrenal SFT, TERT promoter mutations and TP53 alterations have been associated with more aggressive behavior and tumor progression in some studies [8]. However, the prognostic significance of these alterations remains currently unknown in ASFTs. To date, no cases of ASFTs with associated expression of these biomarkers have been reported. Further studies are required to determine whether integrating molecular features can improve current risk stratification systems for adrenal lesions [44].

5.4. Fine Needle Aspiration and Biopsy

The role of fine-needle aspiration (FNA) or core needle biopsy (CNB) in the diagnosis of SFTs remains controversial, particularly in the context of adrenal lesions [45].

Generally, cytological diagnosis of SFT is challenging. Although cases of SFT diagnosed by FNA have been reported in multiple anatomical sites, cytological specimens are often limited by low cellularity or by cytomorphological features overlapping with other spindle-cell neoplasms, lacking distinctive architectural characteristics [46,47]. As a result, cytology alone is frequently insufficient for definitive diagnosis.

Ancillary immunohistochemical studies are therefore required. In this context, STAT6 immunostaining has emerged as a valuable diagnostic tool. The diagnostic performance of STAT6 in cytological specimens has been evaluated by Tani et al., who demonstrated that STAT6 immunocytochemistry on FNA samples may achieve a sensitivity comparable to that observed in surgical specimens, supporting its potential role in the preoperative diagnosis of SFT [48].

Conversely, CNB provides intact tissue fragments that allow for both architectural evaluation and extensive immunohistochemical profiling, making it inherently superior to FNA for spindle-cell lesions [49].

Despite these advances, the application of FNA and CNB to adrenal lesions remains limited. According to current clinical practice guidelines issued by the ESE/ENSAT, adrenal biopsy should be considered only under strict conditions, namely when: (i) the lesion is hormonally inactive, with particular exclusion of pheochromocytoma; (ii) imaging does not conclusively demonstrate a benign lesion; and (iii) histological characterization would directly influence clinical management [6]. This conservative approach is dictated by the risk of potential neoplastic seeding and, more critically, the risk of inducing lethal cardiovascular complications (such as hypertensive adrenal crises) if a subclinical pheochromocytoma is inadvertently punctured. Within this framework, biopsy is generally not recommended in the routine evaluation of adrenal incidentalomas, which limits the opportunity for preoperative diagnosis [6]. Consistently, diagnostic FNA was attempted only in one case of bilateral ASFT. In the case described by Toniato et al., FNA of the right adrenal lesion yielded non-specific findings, including a poorly cellular hematic specimen with lymphocytes and necrosis, and was ultimately inconclusive [14].

Taken together, these observations suggest that FNA and CNB combined with STAT6 immunocytochemistry may represent a promising diagnostic approach in selected cases, but their role in ASFTs remains uncertain. Surgical resection, therefore, continues to represent the mainstay for both definitive diagnosis and treatment in most patients.

6. Management

Complete surgical resection represents the cornerstone of treatment for SFTs, regardless of anatomical location. Since SFT is a ubiquitous tumor, technical aspects vary among different sites. For localized disease, surgery alone is usually sufficient [8].

Adrenalectomy is typically performed in accordance with international guidelines for adrenal incidentalomas because ASFTs cannot be reliably distinguished from potentially

malignant adrenal tumors based on imaging findings alone [6]. Surgical excision therefore provides both definitive diagnosis and curative treatment when complete resection is achieved [50].

The role of adjuvant therapy remains under investigation. Radiotherapy and systemic therapies have been investigated mainly in locally advanced or metastatic SFTs arising in other anatomical sites, with variable results [51,52]. Targeted therapies directed against angiogenic pathways (anti-VEGF) and tyrosine-kinase inhibitors (TKIs) have shown some activity in aggressive or unresectable disease, consistent with the hypervascular nature of these tumors [8].

However, given the rarity of ASFTs and the achievement of complete surgical remission with no recurrence in all cases described hitherto, the role of these therapies in ASFTs is unknown, and no standardized adjuvant treatment strategies are currently defined.

7. Risk Stratification, Prognosis and Follow-Up

The majority of SFTs behave in an indolent fashion following surgical resection [53]. Nevertheless, these tumors are characterized by unpredictable long-term biological behavior, and a subset may recur or metastasize even many years after initial treatment. Reported rates of recurrence and metastatic disease range between approximately 5% and 25% in large series of extrathoracic SFTs. For this reason, most authors recommend long-term radiological surveillance, particularly in patients with intermediate- or high-risk tumors [8].

Several prognostic models and risk classification systems have been developed to predict tumor behavior. The most widely used model was proposed by Demicco et al. and is currently the standard applied in the World Health Organization (WHO) classification of soft tissue and bone tumors [1] (Table 2). Based on four clinical-histopathological variables (patient's age, tumor size, mitotic activity and presence of tumor necrosis), tumors are categorized into low- (0–3 points), intermediate- (4–5 points) and high-risk (6–7 points) groups for metastatic disease. Low-risk tumors rarely metastasize, whereas intermediate-risk lesions carry an estimated 10% risk of metastasis at ten years. High-risk tumors have a substantially greater metastatic potential, with reported rates approaching 75% at five years [30].

When this model is applied to the ASFTs reported in the literature, the majority of tumors fall into the low-risk category (Table 1), suggesting a generally favorable behavior, despite a mean tumor size of approximately 10 cm. This is primarily due to the typically low mitotic activity and absence of necrosis, which explains the discrepancy between the large tumor volume and the favorable clinical outcomes observed to date. Consistently, recurrence or malignancy was not reported in any case of ASFT during longitudinal follow-up. In the case reported by Prevot et al., the mass discovered incidentally in a 42-year-old woman behaved in an innocuous manner, remaining unchanged for more than 5 years before the patient agreed to surgical intervention [9]. No recurrent or metastatic disease was reported at 18, 24, and 36 months in the cases described by Toniato et al., Ambardjieva et al., and Yonli et al., respectively [14,16,19]. In a recent case series of 9 ASFTs, all patients were alive, and no signs of recurrence or metastasis were detected at a median follow-up of 14 months [23].

Nevertheless, the rarity of this clinical entity and the relatively short follow-up periods available in most reports preclude definitive conclusions regarding long-term prognosis. Consequently, radiological follow-up strategies have not been standardized for ASFTs. In the literature, post-operative monitoring has been primarily performed using periodic CT or, alternatively, MRI, according to clinical judgment. Periodic imaging remains appropriate and advisable given the potential for late recurrence [8].

Table 2. Modified four-variable risk stratification model for the development of metastasis in solitary fibrous tumors (SFTs) developed by Demicco et al. [30].

Risk Factor	Score
Age	
<55	0
≥55	1
Tumor Size (cm)	
<5	0
5 to 10	1
10 to 15	2
≥15	3
Mitotic count (/10 HPF)	
0	0
1–3	1
≥4	2
Tumor Necrosis	
<10%	0
≥10%	1
Risk Class	Total Score
Low	0–3
Intermediate	4–5
High	6–7

Legend: low-risk patients had no risk of metastasis at 5–10 years, intermediate-risk cases had only a 10% risk of metastasis at 5 and 10 years, and patients in the high-risk class had a 73% risk of metastasis at 5 years [30]. HPF = high-power fields.

8. Future Perspective and Conclusions

ASFTs represent an exceptionally rare neoplasm but should be considered in the differential diagnosis of non-functioning adrenal incidentalomas when radiological features are not consistent with those of a typical adenoma. In this context, the combination with paraneoplastic manifestations, such as unexplained fever, non-endocrine hypertension, and non-islet cell hypoglycemia, should raise clinical suspicion.

Clinical and radiological features are non-specific, and definitive diagnosis relies on surgical excision with histopathological evaluation supported by immunohistochemistry and identification of the NAB2–STAT6 molecular alteration.

Complete surgical resection remains the cornerstone of management and is usually associated with favorable outcomes. However, given the unpredictable biological behavior, long-term surveillance is advisable.

Future research on SFTs will likely focus on improving molecular characterization and refining prognostic models. The integration of molecular markers and traditional histopathological parameters may enhance risk stratification and help identify patients who may benefit from closer surveillance or targeted therapies. Furthermore, expanding future patient cohorts will be paramount to define the most accurate and cost-effective imaging modality for long-term surveillance protocols.

Greater awareness of ASFTs among clinicians and radiologists involved in the management of adrenal incidentalomas may facilitate earlier recognition and appropriate therapeutic and follow-up strategies.

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Abbreviations

The following abbreviations are used in this manuscript (listed in alphabetical order):

AI	Adrenal Incidentaloma
ASFT	Adrenal Solitary Fibrous Tumor
BCL-2	B-Cell Lymphoma 2
CD34	Cluster of Differentiation 34
CD99	Cluster of Differentiation 99
¹¹ C-MET	Carbon-11 methionine
CNB	Core Needle Biopsy
CT	Computed Tomography
DPS	Doege–Potter Syndrome
DST	Dexamethasone Suppression Test
ESE	European Society of Endocrinology
ENSAT	European Network for the Study of Adrenal Tumors
¹⁸ F-FDG	18-Fluorodeoxyglucose
FNA	Fine Needle Aspiration
FUO	Fever of Unknown Origin
⁶⁸ Ga-PSMA	Gallium-68 Prostate-Specific Membrane Antigen
HPA	Hypothalamic–Pituitary–Adrenal Axis
HPF	High-Power Fields
HPO	Hypertrophic Pulmonary Osteoarthropathy
HU	Hounsfield Units
IHC	Immunohistochemistry
IGF-2	Insulin-like growth factor-II
MRI	Magnetic Resonance Imaging
NA	Not Applicable
NED	No Evidence of Disease
NFAT	Non-functioning Adrenal Tumor
NICTH	Non-islet Cell Tumor Hypoglycemia
NR	Not Reported
PET	Positron Emission Tomography
PHEO	Pheochromocytoma
STAT6	Signal Transducer and Activator of Transcription
SFT	Solitary Fibrous Tumor
SD	Stable disease
TERT	Telomerase Reverse Transcriptase
TKIs	Tyrosine-kinase inhibitors
TP53	Tumor Protein 53
UFC	Urinary Free Cortisol
US	Ultrasound
VEGF	Vascular–Endothelial Growth Factor
WHO	World Health Organization

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