Alveolar Soft Part Sarcoma (ASPS)
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ABSTRACT

Alveolar soft part sarcoma (ASPS) is a rare, poor prognosis neoplasm of unknown histogenesis with a distinctive histology, specific molecular characteristics, and unique clinical behaviors. ASPS generally develop in younger patients. Unlike other soft tissue sarcomas, ASPS also metastasizes to the brain. While surgery can improve outcomes even in the setting of metastatic disease, traditional chemotherapeutic agents and/or radiotherapy have failed to demonstrate significant survival advantages. This article provides an overview of the clinical manifestations, diagnosis, radiographic features, and treatment of ASPS.

BACKGROUND

Alveolar soft part sarcoma (ASPS) is a distinct histologic soft tissue sarcoma subtype.\(^1\) ASPS is an uncommon tumor, typically occurring in adolescent and younger adult patients. It accounts for 0.5 to 1% of all soft tissue sarcomas. Despite a relatively indolent tumor growth pattern, up to 79% of the patients develop metastatic disease with a high proportion being resistant to conventional chemotherapeutic regimens. The development of therapeutically resistant metastasis contributes to increased mortality.

Credit for the original description of ASPS traditionally goes to Christopherson, then a fellow in surgical pathology at Memorial Sloan Kettering Cancer Center. With the publication of a study of 12 cases in 1952, Christopherson et al. established the descriptive term “alveolar soft part sarcoma” for a unique soft-tissue tumor.\(^1\) This tumor was first defined histologically by the presence of cells arranged in nests (“alveoli”) separated by delicate partitions of connective tissue containing sinusoidal vascular channels lined by flattened endothelium.\(^1\) Prior to Christopherson’s publication, the entity ASPS had been described by a variety of other names, including “malignant myoblastoma”, “granular cell myoblastoma” and “malignant granular cell myoblastoma.”\(^2-6\) While Christopherson et al. did not describe the intracytoplasmic crystalline structures that have become one of the hallmarks of ASPS, they did quote an unpublished letter from Dr. Pierre Masson, who noted the intracytoplasmic crystals.\(^1\) Therefore, credit for the intracytoplasmic crystals belongs to Dr. Masson, who later in 1956 published a study on the ultrastructural appearance of these structures.\(^7\) Unknown to Christopherson and colleagues, ASPS had been described by Smetana and Scott one year earlier, as malignant tumors of non-chromaffin paraganglia.\(^8\) They chose this term because the tumors resembled non-physiologically active paraganglia, postulating that primitive paraganglia-like structures may perhaps normally occur in the somatic soft tissues (this hypothesis was later discredited). Smetana and Scott also independently observed the intracytoplasmic crystals of ASPS, describing them as rod-shaped, coarse, basophilic bodies of unknown nature.\(^8\)

CLINICAL MANIFESTATIONS

ASPS usually presents as a soft, painless, slow-growing mass that rarely causes functional impairment. In adults, the lower extremities are the most common location for this lesion, although it has been described in a variety of locations including the female genital tract, mediastinum, breast, urinary bladder, gastrointestinal tract, and bone.\(^9-13\) In children, ASPS most
often occurs in the head and neck region. These tumors are extremely vascular, and occasionally present as a pulsatile mass with an associated bruit. Because of the relative lack of symptoms, in many patients the tumor is easily overlooked and metastasis to the lung or other sites may be the first disease manifestation. The most common metastatic sites are lung, bone, central nervous system, and liver. Metastasis has been reported as long as 15 years after initial resection of the tumor. Unlike other soft tissue sarcomas, ASPS also metastasizes to the brain, and are described as a common feature of metastatic ASPS (Figure 1).

A review of 70 ASPS patients treated at the University of Texas MD Anderson Cancer Center revealed that brain metastases were almost always detected in conjunction with metastatic disease at other sites.

**DIAGNOSIS**

**Radiological Findings**

Accurate diagnosis and treatment of this unusual tumor requires a high index of clinical suspicion coupled with clinicopathologic correlation via appropriate radiographic studies. If the clinical or radiographic interpretation is equivocal, early biopsy is essential to differentiate alveolar soft part sarcoma from arteriovenous malformation. ASPS tumors appear to be hypervascular on angiography and computed tomographic scan (CT scan), with a dense tumor stain and tortuous, dilated draining veins.

**Diagnosis**

Diagnosis of ASPS requires clinicians from different specialties, such as radiologists (a physician who has specialized training in obtaining and interpreting medical images), pathologists (a physician who interprets and diagnoses the changes caused by disease in tissues and body fluids), surgeon oncologists (a physician that deals with the surgical treatment of cancer), and medical oncologists (a physician who uses chemotherapy to treat cancer).
Figure 2 is a CT scan of a 50 year-old gentleman with a 10 cm diameter ASPS arising from the anterior lower chest wall. The tumor in the image demonstrates a vascular periphery surrounding a core of central necrosis.

Figure 2 (left): CT scan of a 50-year-old gentleman with a large alveolar soft-part sarcoma arising from the anterior lower chest wall (red arrow). The tumor in the image demonstrates a vascular periphery surrounding a core of central necrosis (white arrow). Figure 3 (right): Cross-section from a resected primary ASPS tumor from the right thigh reveals a well-circumscribed mass with focal areas of hemorrhage.

Magnetic resonance imaging typically exhibits high signal intensity of tumor on both T1- and T2-weighted images.\(^\text{17}\) Three-phase bone scans with administration of 26.4 mCi and Tc-99m oxidronate sodium (Tc-99m HDP) can also be used to show the vascularity of the tumor in selected cases.\(^\text{18}\)

**Pathological Findings**

Tumor size usually ranges between 3 and 8 cm, but cases of ASPS up to 20 cm have been reported. Macroscopically, the tumor tissue is pale gray or yellowish in color and has a soft consistency (Figure 3).

Areas of necrosis and hemorrhage are common in larger lesions. Histologic examination reveals ASPS tumors to be composed of well-defined nests of cells separated by delicate fibrovascular septae (Figure 4A). Within these nests there is a prominent lack of cellular cohesion, accounting for the distinctive pseudoalveolar pattern for which this disease is named (19). A variant of ASPS is observed in young patients with lingual ASPS, which has focal prevalence of solid ‘non-alveolar’ growth pattern, without the typical cellular discohesion that is observed in non-lingual ASPS (20). Intravascular extension is present at the periphery of the tumor in almost all cases, and may account for the high rate of metastasis observed in ASPS (Figure 4B).
The cells frequently contain eosinophilic crystalline or rod-shaped inclusions which are faintly visible in hematoxylin-eosin-stained tissue sections. On periodic acid-Schiff (PAS) stains, intracytoplasmic glycogen and characteristic PAS-positive, diastase-resistant rhomboid or rod-shaped crystals may be present (Figure 4C, D). Typical crystalline material is seen in at least 80% of cases, and PAS-positive granules are present in almost all tumors. It has been shown that the crystalline cytoplasmic granules of ASPS contain monocarboxylate transporter 1 and CD147. Ultrastructurally, ASPS cells have numerous mitochondria, a prominent smooth endoplasmic reticulum, glycogen, and a well-developed Golgi apparatus.

Another characteristic ultrastructural feature of ASPS is the membrane bound or free rhomboid, rod-shaped crystals consisting of rigid fibrils (Figure 4D). These tumors also express desmin, an intermediate filament. It has been shown that approximately 50 percent of tumors express desmin (9). It is important to emphasize that desmin expression can be seen in a wide variety of other lesions such as melanoma, Ewing’s sarcoma and angiomatoid malignant fibrous histiocytoma, among others (R).

ASPS often represents a diagnostic challenge. Due to the epithelioid appearance of the neoplastic cells and their pseudoalveolar growth pattern, ASPS may resemble a wide variety of neoplastic conditions, such as metastatic renal cell carcinoma, paraganglioma, granular cell tumor, and melanoma (21). In the majority of cases the clinical presentation, together with the demonstrations of PAS-positive diastase-resistant crystals, will be sufficient to make a diagnosis. Occasionally, the intracytoplasmic crystals are not present. In these instances, the presence of well-developed Golgi complexes containing many small granules in and around them is a helpful finding in the proper clinical context (21). Immunohistochemical analysis is a useful tool in establishing the differential diagnosis. For example, renal cell carcinoma can be differentiated by...
from ASPS by its strong cytokeratin expression (9). Additionally, renal cell carcinoma lacks intracytoplasmic crystals or Golgi complexes with small dense core granules. ASPS lingual tumors often demonstrate very small nests of cells, closely mimicking true paragangliomas (20). These two entities can be differentiated in that the paragangliomas present strong reactivity for chromogranin and synaptophysin and are negative for desmin (9).

Granular cell tumors differ from ASPS by the presence of numerous lysosomes with particulate content and the absence of crystals (22). Melanoma can be distinguished from ASPS by the presence of premelanosomes and the lack of crystals (9).

**Molecular Analysis**

ASPS is characterized by a tumor-specific translocation: der(17)t(X;17)(p11;25). This translocation causes the fusion of the transcription factor TEF3 located on Xp11.22 with a novel gene at 17q25, named ASPL, also known as ASPSCR1 (Figure 5).

![Figure 5: Alveolar Soft Part Sarcoma (ASPS) Translocation. The translocation between chromosome 17 (blue) and X (orange) characteristic of ASPS is seen in panel A. In the exonic gene diagrams (B), the TFE3 gene (Xp11.2; orange) contributes either exons 3-8 or 4-8. Abbreviations: UT, untranslated region; AD, activation domain; bHLH, basic helix-loop-helix domain; UBX, UBX homology domain; LZ, leucine zipper domain.](image-url)
Panel A: Non-reciprocal fusion events are uncommon in most sarcomas, but predominate in ASPS as illustrated in the (A; upper right).

Exons 3-8 or 4-8: The breakpoint in ASPSCR1 (17q25; blue), previously termed ASPL, is invariant. This results in type 1 and type 2 fusion transcripts as depicted. Type 2 contains the activation domain of TFE3 while type 1 does not, but both form novel transcription factors incorporating the DNA binding domain of TFE3 with the activation domain of ASPSCR1 and are functional. Differences in function of the protein or clinical outcomes are not described; type 1 fusion events may be more common than type 2, but only a small number of cases are reported.

This characteristic translocation between chromosomes X and 17 results in a functional transcription factor with altered target gene activation, implicating transcriptional deregulation in the pathogenesis of this tumor (23). The translocation creates a novel ASPL-TFE3 fusion protein that appears to act as an aberrant transcription factor, inducing unregulated transcription of TFE3-regulated genes.

It has been demonstrated that tumors having the ASPL-TFE3 translocation express TFE3 (Figure 6) (24). As a consequence, detection of TFE3 nuclear expression is useful in establishing the diagnosis of ASPS.

Figure 6: An ASPS demonstrating uniform and strong nuclear reactivity for TFE3, confirming the presence of an ASPL-TFE3 fusion protein.

TREATMENT
Radical resection is the therapy of choice for localized disease (14, 25). Local recurrence rates have been reported to range from 11-50% (10, 11). Achievement of complete microscopic R0 resection is critical in localized alveolar soft part sarcoma, but incomplete excision due to lack of appreciation of the correct diagnosis is all too often encountered. Despite the occurrence of metastases in up to 79% of patients, 5-year overall survival rates range from 45 to 88% (10, 11, 26-28). No significant survival advantages have been achieved by utilizing conventional chemotherapy, radiation or excision for patients who have evidence of metastasis at the time of original diagnosis as compared to patients who are not treated (10, 11). In our institution, patients with metastatic ASPS are evaluated individually. Due to the resistance of ASPS to conventional chemotherapy, we propose to observe these patients or to consider the enrollment in clinical trials using novel therapies as potential treatment. As has been previously reported, few patients who subsequently developed lung metastases treated by metastasectomy (with or without radiation) had an increase in median survival as compared to those not treated by excision (218 months versus 63.5 months) (10). However, since this observation was based on small numbers of
patients, further verification of this finding is required. Salvati et al. reported a study including three ASPS patients who underwent surgical removal of ASPS brain metastases followed by radiotherapy and/or chemotherapy. Of the three patients with ASPS, two patients were alive at 15 and 20 months, whereas the third patient was alive at 24 month follow-up (29). Due to the limited number of patients, it is challenging to make a conclusive statement regarding the role of resection of ASPS brain metastasis. We consider metastasectomy in those patients that have good performance status and operable tumors that can be resected completely with acceptable morbidity.

New Targeted Therapies
While surgery may improve outcomes even in the setting of metastatic disease, traditional chemotherapeutic agents and/or radiotherapy have failed to confer significant survival advantages (30, 31). Chemotherapeutic regimens used for the treatment of other soft tissue sarcomas generally lack efficacy in ASPS. Recently, systemic cancer therapeutics have focused on utilization of molecularly targeted treatments rather than, or in addition to, non-specific cytotoxic agents. This change in medical management has prompted the search for and identification of commonly expressed deregulated targets amenable to pharmaceutical inhibition. For example, we have evaluated the expression of potential targets in ASPS utilizing a focused ASPS tissue microarray (32). Equipped with this unique bioresource, we have demonstrated that the c-Met receptor and its downstream effectors (e.g., AKT and ERK) are activated in ASPS.

The gene that encodes for c-Met receptor (MET) was recently identified as an ASPL–TFE3 transcriptional target (33). This gene is up-regulated by TFE3, resulting in the increased production of c-Met protein. Activation of the c-Met receptor and downstream signaling has been shown to promote angiogenesis (growth of blood vessels), proliferation, survival, cell motility and invasiveness in tumor cells, and might contribute to the malignant progression in ASPS. Therefore, evaluation of c-Met warrants further attention.

c-Met is a tyrosine kinase receptor. Both c-Met and its ligand, hepatocyte growth factor (HGF), are each required for normal mammalian development and have been shown to be particularly important in cell migration, morphogenic differentiation, and organization of three-dimensional tubular structures as well as cell growth and angiogenesis. Both c-Met and HGF have been shown to be deregulated in a number of major human cancers. New drugs targeted against c-Met and HGF are currently being investigated in vitro and in vivo, with promising results. These drugs function at a variety of steps including c-Met expression at the RNA or protein level, the ligand-receptor interaction, and tyrosine kinase function (R).

A Phase II clinical trial evaluated the use of ARQ 197, a novel c-Met inhibitor, for the treatment of ASPS patients. Preliminary data of this trial was presented at ASCO 2009, indicating that a total of 28 patients—of whom 17 had ASPS—were treated with ARQ 197. Fifteen patients with ASPS demonstrated stable disease for durations up to 29+ weeks. An overall response rate of 5% and a disease control rate (CR+PR+SD) of 80% were demonstrated among 20 patients who were evaluable for efficacy. Furthermore, activation of ERK and AKT pathways might also occur as a result of stimulation by factors other than activated c-Met receptor. Currently, a Phase II study is being conducted to evaluate the use of KRX-0401 (perifosine), an AKT inhibitor, in ASPS patients.

Due to the prominent vascularity of ASPS, we examined the expression of proangiogenic genes via angiogenesis oligomicroarrays (28), demonstrating that eighteen angiogenesis-related genes could be identified as up-regulated in ASPS. Complementary to these findings, several groups have demonstrated the feasibility of using an antiangiogenic approach in the treatment of ASPS using in vivo preclinical ASPS models (34, 35) to assess antiangiogenic approaches such as bevacizumab. Moreover, Cediranib (AZD2171), a VEGF/KIT tyrosine kinase inhibitor that blocks the elaboration of new blood vessels, has recently been identified as having antitumor activity in
early phase clinical trials that included some ASPS patients; a Phase II study evaluating the use of Cediranib for the treatment of patients with ASPS is currently active but no longer accruing patients.

Another approach recently receiving attention is the use of tyrosine receptor inhibitors where the underlying rationale is based on the high levels of activated tyrosine kinase receptors (TKR) such as PDGFR, EGFR, MET family and RET observed in ASPS (36). In a study of five cases, four patients with advanced ASPS were treated with sunitinib malate, a tyrosine kinase receptor inhibitor with direct antitumor and antiangiogenic activity targeting PDGFR, KIT, FTL3, VEGFR and RET (36). Among the four patients who were evaluated for response, two patients showed partial response, one patient had stable disease, whereas the other progressed. Although this data is very preliminary, the observations are promising and suggest some effectiveness of sunitinib malate in ASPS. Several reports have demonstrated the potential use of TKR inhibitors such as Nexavar (Sorafenib) in ASPS patients.

**SURVEILLANCE**

*Follow up for patients with Localized Disease*

Patients should be followed for many years by an experienced oncologist, both for the risk of recurrence and for the risk of side effects from therapy. ASPS can recur or grow even after many years out from diagnosis and even in cases where surgery has rendered the patient apparently "disease free." Long-term follow-up including evaluation of the original sites of disease and the lungs is advisable. The individual recommendations for the schedule and type of surveillance scans will vary according to the patient, and should consider the small but not negligible risks of repeated exposure to radiation. ASPS recurrences may be amenable to surgery (37).

*Follow up for patients with Metastatic Disease*

Although ASPS may spread to a remarkable variety of tissues, the lung remains the principal site requiring surveillance. Surveillance should consist of a history and physical exam performed by a clinician, and chest imaging (38). There is little evidence to support a recommendation for routine intracranial imaging in patients who present with ASPS (11). Intracranial imaging should be considered when pulmonary metastases are documented or if neurologic symptoms are present. Long-term survival is possible even with metastatic disease. Whether the favorable outcome of metastasectomy in a small number of patients with ASPS is due to the surgical therapy or due to the indolent nature of the disease is undetermined. Therefore, it is difficult to comment on the role of surgery in patients with isolated metastatic ASPS. We recommend to evaluate patients with metastatic ASPS individually, and to consider metastasectomy for patients with good performance status and medically operable tumors who have M1 disease that can be resected completely with acceptable morbidity.

**CONCLUSIONS**

In summary, the descriptive term ‘alveolar soft part sarcoma’ refers to a rare soft tissue sarcoma histologic subtype that typically occurs in young patients. It is characterized by a distinctive histological appearance and a specific molecular genetic abnormality; the prognosis is poor. Identification of a specific ASPS chromosomal translocation not only has provided critical information about the pathogenesis of this disease but also has led to rational molecular targeted therapy evaluation. Most series reported in the literature suggest that ASPS chemosensitivity is modest, providing a compelling rationale underlying a major role for surgery in localized disease. The occurrence of distant metastases is quite common in ASPS; however, even the largest currently published ASPS clinical series (10, 11) does not define the optimal treatment for metastatic ASPS disease. On the basis of our experience (11), the benefit of routine systemic chemotherapy on patients with metastatic disease is uncertain.

However, current treatment recommendations are based on very limited clinical information. In the meantime, new molecular targeted therapies, such as antiangiogenic approaches and
tyrosine kinase inhibitors, comprise the most promising new approaches for the treatment of ASPS, a devastating tumor which unfortunately seems to emerge in patients who otherwise would be just on the cusp of otherwise normal adult life.

**GLOSSARY OF TERMS**

**Angiography:** Medical imaging technique used to visualize the inside of blood vessels.

**Arteriovenous malformation:** Abnormal connection between veins and arteries.

**Basophilic:** It describes the microscopic appearance of cells and tissues, as seen down the microscope, after stained with a basic dye.

**Bruit:** refers to the unusual sound that blood makes when it rushes past an obstruction (called turbulent flow) in an artery when the sound is auscultated with the bell portion of a stethoscope.

**Chromogranin:** Protein found in and released from neuroendocrine cells.

**Clinicopathologic:** Refers to symptoms and pathology of disease.

**Cytokeratin:** Proteins of keratin-containing intermediate filaments found in the intracytoplasmic cytoskeleton of epithelial tissue.

**Endoplasmic reticulum:** A cell organelle, responsible for the production of the protein and lipid components of most of the cell's organelles.

**Eosinophilic:** Eosinophilic means loves eosin, and refers to the staining of certain tissues, cells, or organelles after they have been washed with eosin, a dye.

**Ewing's Sarcoma:** A type of sarcoma that occurs in the bone or close to the bone.

**Fusion protein:** Abnormal proteins created through the joining of two or more genes which originally coded for separate proteins. This abnormal proteins can cause cells to grow uncontrollably and result in cancer.

**Golgi apparatus:** A netlike structure in the cytoplasm of animal cells.

**Hematoxylin-eosin:** This is a popular staining method in histology.

**Histogenesis:** Formation or development of tissues from the undifferentiated cells.

**Histology:** A distinctive structure, composition, and function of tissue.

**Hypervascular:** Containing an excessive number of blood vessels.

**Immunohistochemical analysis:** This is also known as immunohistochemistry, which is a test that can detect the expression of proteins in tissue sections. This technique requires the use of antibodies that can recognize the protein of interest. The proteins of interest are visualized with the use of a marker such as fluorescent dye, enzyme, or colloidal gold.

**Intracytoplasmic:** Inside the cell.

**M1 Disease:** Refers to a patient with a distant metastasis.

**Melanoma:** A dangerous type of skin cancer.

**Metastasectomy:** Surgical removal of metastases.

**Metastatic disease:** Metastatic disease is a cancer that has spread from the part of the body where it started to other parts of the body.

**Mitochondria:** A membrane-enclosed organelle found in most eukaryotic cells.

**Morbidity:** Risk of getting sick.

**Paraganglia:** The paraganglia (or chromaffin bodies) are small groups of chromaphil cells connected with the ganglia of the sympathetic trunk and the ganglia of the celiac, renal, suprarenal, aortic and hypogastric plexuses.

**Radiographic:** Radiography is the use of X-rays to view a cross sectional area of a non-uniformly composed material such as the human body.

**Somatic:** “of the body”

**Synaptophysin:** Synaptophysin also known as the major synaptic vesicle protein p38 is a protein that in humans is encoded by the SYP gene.

**Tissue microarray:** Tissue microarrays (also TMAs) consist of paraffin blocks in which up to 1000 separate tissue cores are assembled in array fashion to allow multiplex histological analysis.

**Transcription factor:** A protein that binds to DNA sequences, thereby controlling the movement (or transcription) of genetic information from DNA to mRNA.

**Translocation:** Translocation is the movement of a gene fragment from one chromosomal location to another, which often alters gene expression and results in the creation of an abnormal protein.

**Editor's Note:** Additional Information about ASPS can be found in the 2005 ESUN article, "Clear Cell Sarcoma and Alveolar Soft Part Sarcoma," by Dr. Goldberg and Dr. Albritton.
REFERENCES

The Electronic Sarcoma Update Newsletter

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