Solitary Fibrous Tumor of Nasal Cavity in Patient With Long-Standing History of Cocaine Inhalation

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• We report a solitary fibrous tumor in the nasal cavity of a 48-year-old woman who presented with a history of bilateral nasal obstruction and long-standing cocaine inhalation. Physical examination revealed a large mass involving the right nasal cavity and extending into the posterior aspect of the left nasal cavity. The computed tomography scan showed opacification of airways. During surgery, the mass was found to involve the entire nasal cavity, with extension to the right maxillary sinus, posterior nasal airways, and left nasal cavity. The mass was completely excised. Pathologic examination revealed a polypoid mass 3.7 × 3.0 × 1.2 cm. This tumor was composed of spindle cells that were cytologically bland in a background of ropey and nodular collagen, giving a "patternless" pattern. Immunohistochemically, the neoplastic cells stained for CD34 and vimentin but not for S100 protein, keratin, desmin, HMB-45, and c-Kit. This immunohistochemical pattern confirmed the diagnosis of solitary fibrous tumor. Although solitary fibrous tumors are usually found in the pleura, they can occur in various other locations, such as the orbit, nasal cavity, paranasal sinuses, mediastinum, breast, vagina, meninges, and soft tissues. This case is of interest because the tumor occurred in a patient with prolonged cocaine inhalation. Such an association has not been previously described. The exact causal relationship between cocaine inhalation and the tumor is not known.

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Solitary fibrous tumor (SFT) is a spindle cell tumor of adults that typically occurs in the pleura.[1–3] Solitary fibrous tumors have been reported in various other locations, including those with no relation to serosal surfaces. Solitary fibrous tumors involving the nasal cavity are extremely rare, and only about a dozen cases have been reported,[4–6] none of which have been described in a chronic cocaine abuser.

CLINICAL HISTORY

A 48-year-old woman presented with a history of bilateral nasal obstruction and long-standing cocaine "snorting." Physical examination revealed a large mass filling the entire right nasal cavity and extending into the posterior aspect of the left nasal cavity. The computed tomography scan revealed opacification of airways. During surgery, a large polypoid mass was identified, involving the entire right nasal cavity with extension to the right maxillary sinus, posterior nasal airway, and left nasal cavity. The attachment of the mass appeared to be at the posterior tip of the middle turbinate. The mass was excised completely.

MATERIALS AND METHODS

This tumor was processed as a surgical pathology specimen. Histologic sections were prepared from formalin-fixed paraffin-embedded tissue. The tissue sections were stained with hematoxylin-eosin. To classify this neoplasm, immunoperoxidase staining using the avidin-biotin complex technique was performed. The antibodies used were AE1/AE3 (monoclonal mouse isotype immunoglobulin [Ig] G kappa, 1:200 dilution, Zymed Laboratories, South San Francisco, Calif), CD34 (monoclonal QBend/10 mouse isotype IgG1, dilution 1:40, BioGenex, San Ramon, Calif), vimentin (monoclonal mouse isotype IgG1, dilution 1:1000, BioGenex), S100 (polyclonal rabbit anti-cow, dilution 1:800, Dako Corporation, Carpinteria, Calif), desmin (monoclonal mouse isotype IgG1 kappa, dilution 1:50, Zymed), HMB-45 (monoclonal mouse isotype IgG1, predilute antibody, Zymed), and c-Kit (polyclonal rabbit anti-human, dilution 1:100, Dako).

PATHOLOGIC EXAMINATION

Gross examination revealed a polypoid mass 3.7 × 3.0 × 1.2 cm. The external surface of the mass was yellow-tan and smooth. The cut surface was pink-tan, slightly firm, and homogeneous. Microscopically (Figures 1 through 5), this tumor was unencapsulated and composed of cytologically bland spindle cells in a background of ropey and nodular collagen, with no identifiable pattern. Hypercellular areas alternated with hypocellular areas. Numerous small vessels and dilated vascular channels were present. Mitotic figures were rare. Immunohistochemically, the neoplastic cells were positive for CD34 and vimentin and negative for S100 protein, keratin, desmin, HMB-45, and c-Kit. This immunohistochemical pattern confirmed the diagnosis of SFT.

COMMENT

Solitary Fibrous Tumor

Solitary fibrous tumor was originally described as a localized fibrous mesothelioma.[2,7] The histogenesis of SFT has been controversial, but immunohistochemical and structural analyses have strongly suggested a mesenchymal origin rather than an epithelial or mesothelial origin.[7] Most SFTs have a benign behavior, although about 15% to

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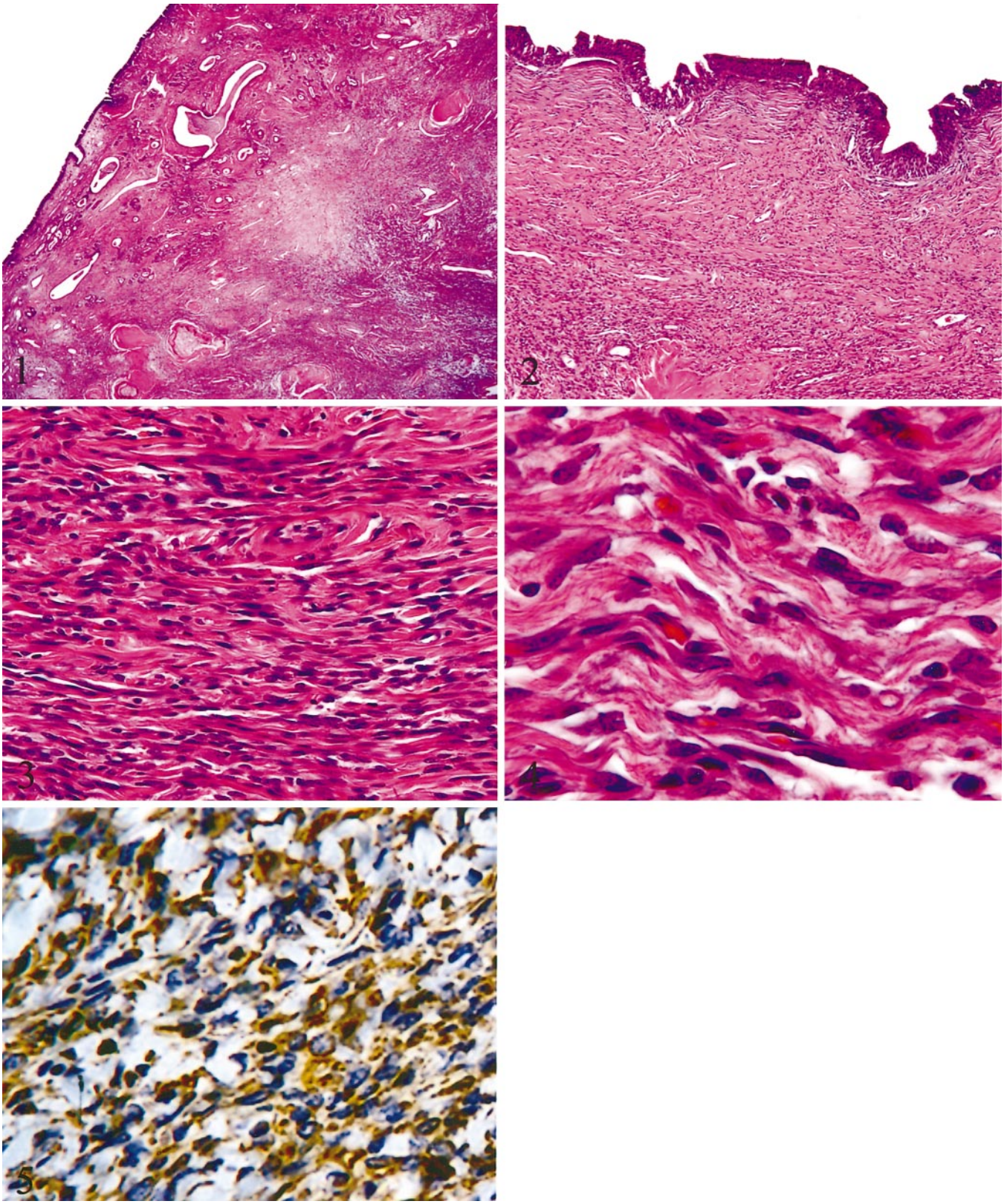

Figure 1. *Solitary fibrous tumor with overlying nasal mucosa (hematoxylin-eosin, original magnification ×20).*

Figure 2. *Solitary fibrous tumor; cellular area composed of bland spindle cells (hematoxylin-eosin, original magnification ×40).*

Figure 3. *Solitary fibrous tumor; bland spindle cells admixed with disrupted collagen (hematoxylin-eosin, original magnification ×160).*

Figure 4. *Solitary fibrous tumor; spindle cells and collagen (hematoxylin-eosin, original magnification ×400).*

Figure 5. *CD34-positive staining of solitary fibrous tumor cells (immunoperoxidase CD34, original magnification ×400).*

20% of cases can be aggressive. The suggested pathologic criteria for the characterization of malignancy include very large size, high cellularity, more than 4 mitotic figures per 10 high-power fields, cellular pleomorphism, hemorrhage, and necrosis.

Resectability has been considered the single most important indicator of clinical outcome.[3] Spindle cell tumors of the nasal cavity can mimic other tumors at this site both in clinical symptoms and pathologic findings. Microscopically, SFTs are composed of bland spindle cells without a pattern, hypo- and hypercellular areas, abundant collagen, and prominent vascularity. Benign tumors usually have minimal or no mitotic activity and no necrosis, hemorrhage, or pleomorphism. The tumor in this woman contained bland-appearing spindle cells, no hemorrhage or necrosis, and very rare mitotic figures. Therefore, based on clinical, radiologic, and pathologic examination, this tumor would be expected to behave indolently.

Differential diagnosis of SFTs at this site includes sinonasal hemangiopericytoma, fibrous histiocytoma, fibromatosis, angiofibroma, and fibrosarcoma. Usually, the morphology and immunohistochemical staining pattern help distinguish SFTs from other tumors.[2,8] The tumor cells in SFTs stain positively for vimentin and CD34 and negatively for S100, kerain, epithelial membrane antigen, and smooth muscle markers. Reactivity to CD34 (although not absolutely specific) combined with the specific histomorphology is very helpful in differentiating SFTs from other lesions.[8]

Sinonasal hemangiopericytoma[9,10] typically presents as intranasal tumors with symptoms of epistaxis and obstruction and frequently occurs in middle-aged patients (mean age, 53 to 58 years). Hemangiopericytomas are composed of sheets of bland, round spindle cells with ramifying blood vessels of various sizes. Although focally this pattern can be seen in SFTs, SFTs predominantly show a haphazard or patternless arrangement of cells.

Although angiofibroma[11] most commonly occurs in teenage boys, exceptional cases have been reported in elderly patients and women (although most of the latter cases are misdiagnoses). In our case, vascular channels embedded in a collagen matrix suggested an angiofibroma. Differentiating features for these entities are the parallel arrangement of collagen fibers in angiofibroma compared with the haphazard or patternless arrangement of collagen in SFT.

Benign and malignant fibrous histiocytomas occur rarely in the nasal cavity. Features useful in distinguishing these tumors are the presence of multinucleated histiocytes such as giant cells in fibrous histiocytoma and the greater degree of atypia and mitotic activity present in most malignant nasal fibrous histiocytomas.

Although very rare, fibrosarcoma and fibromatosis also have been reported in the nasal cavity.[12] Solitary fibrous tumors should be differentiated from low-grade fibrosarcoma. Histologically, fibrosarcoma has a herringbone pattern of neoplastic fibroblasts. Fibromatosis is generally more uniformly collagenous than SFTs, and its cells tend to be larger, with more abundant and distinct cytoplasm. Fibromatosis also has broad, sweeping fascicles.

Cocaine Abuse and Complications

Cocaine is a naturally occurring alkaloid that is extracted from the leaves of the *Erythroxylon coca* plant. Classified as a psychostimulant, cocaine exhibits both local anesthetic and neurotransmitter effects.[13] Peripherally, cocaine increases the activity of norepinephrine, which is a neurotransmitter. This profound enhancement of sympathetic tone is responsible for the vasoconstrictive, tachycardiac, and dysrhythmic actions of the drug. Cocaine is well absorbed from mucous membranes. It can be taken intravenously, can be smoked or inhaled in its ''crack'' or ''free base'' form, or can be inhaled by ''snorting.'' Cocaine has an acidic pH of 4. Its purity and sterility and the type of adulterants added to it all directly affect its potential for local and systemic complications. Cocaine that is snorted is vasoconstricting and locally irritating to the respiratory epithelium of the nasal airway. Repeated snorting sets up a cascade of ischemia, inflammation, micronecrosis, infarction, and healing or macronecrosis leading to perforation. Nasal septum perforation of both the cartilagenous and bony tissue has been well documented. Cocaine can also cause other complications in the nasal region, including optic neuropathy, osteolytic sinusitis,[14] nasal septal necrosis, purulent rhinorrhea, focal pain, and strabismus.[14] Reactive vascular lesions of the nasal septum simulating angiosarcoma have also been reported in chronic cocaine abusers.[15]

Neoplasms in the nasal sinus in association with chronic cocaine abuse have not been described. In our case, there was a definite long-term clinical history of cocaine abuse. Although a definite clinical relationship between cocaine and SFT cannot be established, some interesting speculations can be made. Chronic irritation with repeated vasoconstriction/vasodilatation may have created a stromal milieu for the development of a neoplasm. In addition, particular solvents or adulterants in the snorted cocaine may act as chronic stimulants to the primitive fibroblastic cells in the nose, which at some point acquire a neoplastic growth potential resulting in neoplastic growth. Similar cases and further investigation may give more insight and meaning to these speculations.

CONCLUSION

Solitary fibrous tumor is extremely rare in nasal and paranasal sinuses, and its etiology is not known. Cocaine or other irritants may in some cases act as a trigger for growth of these generally benign neoplasms at this site. This case illustrates that neoplasms can also be included in the list of complications of cocaine abuse. Although there was an association of SFT with cocaine inhalation in this particular case, it may be merely coincidental. Further case studies are needed to support the association hypothesis.

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