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Review Article

Nodular Fasciitis Associated with Atypical Clinicopathological Features: A Review of the *USP6* Fusion Gene Partner

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ABSTRACT

USP6 gene rearrangement has been identified in the most of Nodular Fasciitis (NF) cases. Though the detection of several *USP6* fusion partners has been identified, most of nodular fasciitis cases have exhibited the presence of *MYH9-USP6* gene fusion. Recently, a literature review of rare intradermal nodular fasciitis focusing on the genetic analysis has been provided, suggesting that the detection of *USP6* gene rearrangement may be the very useful and important genetic analysis for accurate diagnosis in even intradermal nodular fasciitis. The result provided *MYH9*, *EIF5A* and *TPM4* as a fusion partner with *USP6* in rare intradermal nodular fasciitis. In this review, the author summarized the rare nodular fasciitis accompanied by atypical clinicopathological findings including recurrence and/or metastasis, malignant morphology, atypical mitosis and different fusion partners with *USP6*. The result provided that *USP6* rearrangement is a potential tool for accurate diagnosis in atypical nodular fasciitis. Several fusion partners including *PPP6R3*, *MYH9*, *EIF5A*, *CALD1*, *COL6A2*, *PAFAH1B1* and *SREBF1* with *USP6* have been identified by Fluorescence In Situ Hybridization (FISH) and/or Next Generation Sequencing (NGS), suggesting that these fusion genes may have different biological spectrum reflecting unusual clinicopathological features. Further study is needed to clarify the biological spectrum of *USP6* fusion partner to avoid aggressive and invasive treatment for nodular fasciitis associated with atypical clinicopathological features. Meanwhile, a few cases of nodular fasciitis with atypical manifestations and typical *MYH9-USP6* fusion have been revealed, therefore it is significant to identify *USP6* rearrangement for diagnostic conformation of nodular fasciitis by molecular analysis.

Keywords: Nodular fasciitis; *USP6*-associated neoplasia; Biological spectrum; *USP6* fusion gene partner; Atypical clinicopathological features

INTRODUCTION

USP6 gene rearrangement has been identified in the most of NF. Although the detection of numerous *USP6* fusion partners has been identified, nodular fasciitis cases have typically exhibited the presence of *MYH9-USP6* fusion gene. The novel and rare fusion patterns with *USP6* including *KIF1A*, *TPM4*, *SPARC*, *EIF5A*, *MIR22HG* and *COL1A2* have been also reported [1]. Recently, rare intradermal nodular fasciitis focusing on the molecular analysis has been reviewed [2]. In this article, the author summarized the nodular fasciitis associated with atypical clinicopathological feature including recurrence and/or metastasis, malignant morphology, atypical mitosis and several *USP6* fusion gene partners having different biological spectrum.

Transient neoplastic nature in nodular fasciitis

NF is a benign soft tissue tumor of fibroblastic/myofibroblastic differentiation that was first described in 1955 by Konwaler et al. and a rare intradermal nodular fasciitis has been firstly reported in 1990 [3,4]. Erickson-Johnson et al. suggested that USP6 transcriptional upregulation may be the driving force behind the high proliferative activity and growth and the consistent involutional nature of NF [5]. Previous study emphasized that presentation of clinical, ultrasonographic and pathological features

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features of NF are attributed to the cytogenetic nature having high proliferative growth and involutional nature [6,7]. Regarding clinical manifestations, NF presents most typically in the upper extremities, the trunk and the head and neck in the ages between 20 years and 40 years. The subcutaneous, fascial, intramuscular and rarely intradermal type have been reported with a peculiar clinical manifestation characterized by rapid, self-limited growth and spontaneous regression after a few weeks [8,9]. Pathologically. NF typically featured uniform spindled cells arranged in irregularity intersecting short fascicles and occasional storiform patterns. NF contains of myofibroblasts with brisk mitotic activity without atypical mitosis [10]. Immunohistochemical findings showed that spindle cells of NF were positive for Smooth Muscle Actin (SMA) [1].

LITERATURE REVIEW

A novel fusion partners with *USP6* in nodular fasciitis

Genetic analysis of *USP6* gene rearrangements has been identified in the most of NF cases. The detection of the *USP6* gene rearrangement has also been recognized as a diagnostic procedure in cases with morphological uncertainty [1, 11]. While *MYH9* gene has been identified as the most common *USP6* partner. Wang et al. described that *MYH9-USP6* gene fusion has been observed in most of NF cases [12]. The detection of other *USP6* fusion partners includes *TPM4*, *EIF5A*, *PPP6R3*, *CTNNB*, *SPARC*, *THBS2*, *COL6A2*, *TNC*, *SEC31A*, *COL1A1*, *COL1A2*, *COL3A1*, *CALU*, *NACA*, *SLFN11*, *LDHA*, *SERP1NH1*, *PDLIM7*, *MYL12A*, *PAFAH1B1* and *MIR22HG* as previously reported [1,13,14]. The previous study showed the new and rare fusion patterns with *USP6* in NF including one novel *KIF1A* and five rare types (*TPM4*, *SPARC*, *EIF5A*, *MIR22HG*, *COL1A2*). A novel partner, *KIF1A* gene has been identified in patient with superficial lesion of the arm [1]. Meanwhile, palmar nodular fasciitis with atypical morphology and a novel *SREBF1-USP6* fusion gene has been demonstrated [15]. The diagnosis of low-grade fibroblastic/myofibroblatic tumors at acral locations is important due to the differential diagnosis from malignant tumors. Recently, the current knowledge and trends of the intradermal nodular fasciitis focusing on the molecular analysis have been outlined, exhibiting *USP6* rearrangement in all cases and two cases of *MYH9*, one case of *EIF5A* and one case of *TPM4* as a fusion partner with *USP6*. The detection of *USP6* gene rearrangement using FISH may be the very useful and important genetic analysis for accurate diagnosis in even intradermal NF as previously described [2]. The identification of numerous fusion partners with *USP6* in NF has been common, furthermore the detection of *USP6* fusion partner may contribute to understand the biological nature resulting in accurate diagnosis and the prevention for the unnecessary aggressive therapy [1]. The feature of USP6 fusion partner may reflect biological nature, therefore the identification supports the daily pathological practice, particularly in cases with malignant potential findings.

USP6-Associated Neoplasms (UAN)

USP6 gene rearrangements have been demonstrated in Aneurysmal Bone Cyst (ABC), NF, Myositis Ossificans (MO), Fibro-Osseous Pseudotumor of digits (FP) and Fibroma of Tendon Sheath (FTS) such as cellular type [10]. Due to the sharing a common pathogenesis with so-called *USP6* rearrangement, these diseases are referred to as *USP6*-Associated Neoplasms (UAN) representing clinicopathological similarities. Recent study has also suggested that these diseases share similar clinicopathological features [14]. The transcriptional upregulation of *USP6* is made through a promotor-swapping mechanism leading to the *USP6* overexpression. The *USP6* overexpression promotes tumorigenesis through several pathways including Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells (NF-kB), Wingless-Related Integration Site (Wnt/β-catenin), Janus kinase-signal transducer and activator of transcription (JAK-STAT3) and c-jun and transforms mesenchymal cells [14,16]. Multimodality imaging findings along with molecular analysis of *USP6*-associated neoplasms have been reported [17]. Regarding FTS, it is clinically important to differentiate between FTS and Giant Cell Tumor of the Tendon Sheath (GCTTS). Previous report provided that GCTTS showed hypoechoic nodule with vascularity on US study and represented low signal intensity on T1 and T2 weighted MR images due to the hemosiderin [18]. While molecular analysis is helpful for accurate diagnosis in FTS. *USP6* rearrangements were previously revealed in cellular cases of FTS and found also in hypocellular and hypercellular FTSs [18-20]. FISH and targeted next-generation Ribo Nucleic Acid (RNA) sequencing can be a useful tool to detect a *USP6* rearrangement. However, FISH is limited due to decalcified materials, hypocellular lesions or old age. Meanwhile NGS has limitations for decalcification and poor DNA quality of the lesion and/or the gene rearrangement with a new unknown fusion partner [10].

Table 1: Clinicopathological features in atypical nodular fasciitis.

Study	Age/gender	Location/depth	Atypical clinicopathological features
Guo et al. [26]	42/f	Thigh/subcutaneous	Recurrence and metastases
Teramura et al. [27]	27/m	Upper arm/muscle	Malignant transformation
Sawamura et al. [28]	53/f	Palm/NA	Aggressive behavior

Lenz et al. [32]	41/f	Forearm/predominantly dermis	Atypical mitosis
Papke et al. [29]	7/m	Upper extremity/NA	Malignant morphology
Tomassen et al. [30]	10/m	Chest wall/muscle	Malignant morphology
Qiu et al. [31]	22-Month/m	Scapular/subcutaneous	Atypical mitosis
Wang et al. [33]	27/m	Upper arm/muscle	Malignant morphology
Mejbel et al. [15]	60/m	Palm/NA	Atypical morphology

Note: NA-Not Applicable.

Nodular fasciitis with atypical clinicopathological features and different *USP6* fusion partner

The author previously outlined NF as *USP6*-associated neoplasia along with therapeutic strategy [21]. In addition to the presentations of clinical, ulrtrasonographic and pathological appearances of NF, the evaluation of percentage of *USP6* break-apart FISH signals reflecting lifetime and mitotic counts in NF may be a potential procedure for accurate diagnosis [7,21,22]. It is known that *USP6* promotes tumorigenesis through multiple pathways including Wnt, Jak-1, c-Jun and STAT3 [14,23-25]. Previous report has emphasized that the detection of USP6 gene rearrangement may be the very useful and important molecular finding for even intradermal nodular fasciitis [2]. A fusion partner with *USP6* showed that *MYH9*, *EIF5A* and *TPM4* genes have been identified in rare intradermal nodular fasciitis [2]. In this review, the author summarized the rare NF with atypical clinicopathological findings and molecular features at (Tables 1 and 2). Until now, only nine cases have been reported [15, 26-33]. *PPP6R3-USP6* fusion has been shown in two cases of NF accompanied with malignant manifestation [26, 27]. Guo et al. showed a case of NF at the subcutaneous region of the thigh, 6.2 cm in size with multiple recurrences and metastatic manifestation showing classic histologic findings, the amplification and unbalanced rearrangement in FISH and novel *PPP6R3-USP6* gene fusion by molecular analysis [26]. Another case represented the mass at the muscle of upper arm, 13.2 cm in size with aggressive and no regression growth with local invasion exhibiting typical histologic feature, amplification and unbalanced rearrangement in FISH pattern and *PPP6R3-USP6* gene fusion by targeted RNA sequencing [27]. Two cases accompanied with malignant morphology in NF have been demonstrated [29-30]. Papke et al. reported a morphologically malignant NF with striking nuclear pleomorphism, exhibiting a *USP6* translocation in FISH and the novel *CALD1-USP6* fusion by next-generation-sequencing [29]. Another case represented NF with malignant morphology showing pronounced pleomorphism, atypical mitoses features and a myoid immuno-phenotype and *USP6* rearrangement with a collagen type VI, alpha 2 *(COL6A2)-USP6* fusion was confirmed by FISH, Archer Fusion Plex (Sarcoma Panel) and RNA sequencing [30]. Cases of the atypical mitoses in pathologic features have been documented [31,32]. Lenz et al. showed one atypical mitoses in pathology harboring a novel *EIF5A-USP6* fusion gene by NGS [32]. Though clinicopathological findings are benign, the unbalanced rearrangement and unusual break point of USP6 with a novel *PAFAH1B1-USP6* fusion have been shown, leading to the diagnosis of NF with uncertain malignant potential [31]. This case revealed the occurrence of *USP6* breakpoint in intron 8. According to the previous report, *PAFAH1B1-USP6* fusion has been demonstrated in two cases of ABC only [31]. Meanwhile, two cases of palmer NF have been reported suggesting that this entity exhibited aggressive presentation as previously described [15,28]. An aggressive behavior of NF protruding from the palm with ulnar nerve palsy has been previously reported demonstrating that the diagnosis of NF is supported by the identification of the *MYH9-USP6* fusion gene [28]. Recently, palmar nodular fasciitis with a novel *SREBF1-USP6* fusion gene has been recognized, suggesting that it is important to differentiate between this entity and low-grade fibroblastic/myofibroblastic tumors such as sarcoma [15]. In particular, as palmer NF tends to show aggressive manifestation, therefore the confirmation of *USP6* rearrangement may be a potential procedure for accurate diagnosis of NF. While, in addition to the previous study of NF with atypical presentation and common *MYH9-USP6* fusion, recent study also provided a large intramuscular NF with pathologically local infiltrative margin diagnosed by *USP6* rearrangement in FISH [28,33]. It is important to differentiate between intramuscular NF and Low-Grade Myofibroblastic Sarcoma (LGMFS) for pathologically moderate cellular atypia and diffusely infiltrative margin [33]. Additionally, the recurrence of the skin NF with common *MYH9-USP6* fusion has been shown [1]. As a few cases of NF with common *MYH9-USP6* gene fusion accompanied by atypical manifestation have been reported, furthermore, the detection of *USP6* rearrangement is a potential tool for making accurate diagnosis of NF [1,28,33]. Because the identification of the *USP6* gene rearrangement has been demonstrated as a significant diagnostic tool in cases accompanied by morphological uncertainty [1,11]. In this review, the results showed that eight cases revealed *USP6* rearrangement, exhibiting *PPP6R3*, *MYH9*, *EIF5A*, *CALD1*, *COL6A2*, *PAFAH1B1* and *SREBF1* as a fusion partner with *USP6* by FISH and/or NGS. The detection of *USP6* rearrangement is a potential tool for accurate diagnosis in NF associated with atypical clinicopathological presentations including recurrence and/or metastases, malignant morphology, atypical mitosis and different USP6 fusion partner. Several USP6 fusion partners have been identified, suggesting that these fusion genes may have different biological spectrum reflecting atypical clinico-pathological behavior. Though NF associated with typical

MYH9-USP6 fusion gene and atypical clinicopathological features have been shown, thereby, it is significant to identify *USP6* rearrangement for diagnostic confirmation of NF.

Regarding the therapeutic strategy, the inhibition of the *USP6*-related genes including Jak-1, Frizzled genes and c-Jun may be potential treatment for recurrent and inoperable ABC [21,23-25].

Though the process of NF is typically rapid growth, self-limited, and spontaneous regression. It is putative that the inhibition of *USP6*-related genes might be the potential therapeutic strategies for the extremely rare malignant behavior of recurrence and metastasis in nodular fasciitis shown by *USP6-PPP6R3* gene fusion as previously mentioned [21].

Table 2: *USP6* fusion gene partner in atypical nodular fasciitis.

Study	Age/gender	FISH	NGS	USP6 fusion partner
Guo et al. [26]	42/f	*USP6* rearrangement	NA	*PPP6R3*
Teramura et al. [27]	27/m	*USP6* rearrangement	Done	*PPP6R3*
Sawamura et al. [28]	53/f	*USP6* rearrangement	NA	*MYH9*
Lenz et al. [32]	41/f	*USP6* rearrangement	Done	*EIF5A*
Papke et al. [29]	7/m	*USP6* rearrangement	Done	*CALD1*
Tomassen et al. [30]	10/m	*USP6* rearrangement	Done	*COL6A2*
Qiu et al. [31]	22-Month/m	*USP6* rearrangement	Done	*PAFAH1B1*
Wang et al. [33]	27/m	*USP6* rearrangement	NA	*MYH9*
Mejbel et al. [15]	60/m	NA	Done	*SREBF1*

Note: NA-Not Applicable; NGS: Next-Generation Sequencing; FISH: Fluorescence *In Situ* Hybridization.

DISCUSSION

Though the detection of several *USP6* fusion partners has been identified, most of NF cases have exhibited the presence of *MYH9-USP6* gene fusion. The novel and rare fusion patterns with *USP6* including new *KIF1A* and rare *TPM4*, *SPARC*, *EIF5A*, *MIR22HG* and *COL1A2* have been reported [1]. Recently, a literature review of rare intradermal nodular fasciitis with emphasis on the molecular analysis has been provided, suggesting that the detection of *USP6* gene rearrangement may be the very useful and important genetic analysis for accurate diagnosis in even intradermal NF [2]. The result showed *MYH9*, *EIF5A* and *TPM4* as a fusion partner with *USP6* in rare intradermal NF [2]. In this article, the author outlined the atypical NF with clinicopathological manifestations including recurrence and/or metastasis, malignant morphology, atypical mitosis and several *USP6* fusion gene partners having different biological spectrum. *USP6* rearrangement is a potential tool for accurate diagnosis in even unusual NF. Several fusion partners with USP6 including *PPP6R3*, *MYH9*, *EIF5A*, *CALD1*, *COL6A2*, *PAFAH1B1* and *SREBF1* have been identified by FISH and/or NGS, suggesting these fusion genes may have different biological spectrum reflecting atypical clinicopathological behavior. Because NF with unusual fusion partners with *USP6* tends to show malignant clinicopathological manifestations, further study is needed to clarify the biological spectrum of fusion partner of USP6 to avoid the overtreatment for atypical NF. Meanwhile, it is important to differentiate between palmer NF and low-grade fibroblastic/myofibroblastic tumors such as sarcoma in the palmar lesions [15]. As palmer NF tends to show locally aggressive and indistinguishable from sarcoma, therefore the confirmation of *USP6* rearrangement may be a potential procedure for accurate diagnosis of NF [28]. Though NF is generally benign and self-limited soft tissue tumor, a few cases with atypical manifestations and the presence of *MYH9-USP6* fusion gene have been reported, therefore it is very important to confirm USP6 rearrangement for accurate diagnosis of NF by FISH and NGS. Further studies are also needed to verify for the biology in NF associated with atypical clinicopathological presentation and typical *MYH9-USP6* fusion [29-33].

CONCLUSION

A few cases of nodular fasciitis associated with unusual behavior and typical *MYH9-USP6* fusion have been documented, therefore it is important to identify USP6 rearrangement for diagnostic conformation of nodular fasciitis by molecular analysis. The detection of USP6 rearrangement is a potential tool for accurate diagnosis in atypical nodular fasciitis associated with clinicopatho logical presentation including recurrence and metastases, malignant morphology, atypical mitosis and different USP6 fusion partner. Several *USP6* fusion partners including PPP6R3, MYH9, *EIF5A*, *CALD1* , *COL6A2*, *PAFAH1B1* and *SREBF1* have been identified by FISH and/or NGS in atypical nodular fasciitis, suggesting that these fusion genes may have different biological spectrum reflecting unusual clinicopathological behavior. Further study is needed to verify the biological spectrum of USP6 fusion partner for atypical nodular fasciitis.

CONFLICT OF INTEREST

Author declares that no conflicts of interest.

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