Reviewer's specific comments:

1) Page 3, Lines 73-74: "In several cases, immunotherapy or targeted therapy is very effective in patients with angiosarcoma." This sentence is out of context, lacking citations.

   Thank you very much for your comment. We added literatures in our paper.

2) Lines 77 – 131 should be provided with a table of the patient's demographics.

   Thank you very much for your work and comment. As for your comment to provide the patient's demographics, we have reviewed our paper again. We thought that the basic information was mentioned in **Case presentation** in detail. And in other published case reports, few paper provided these kinds of table. In our opinion, although table of the patient's demographics may make the information clearer, it may be needless for a case report. For the sake of brevity, we do not intend to provide such a table in this paper. We hope the reviewers could kindly understand our approach.

3) Fig 2 should be provided with scale bars.

   Thank you very much for your work and comment. We made some amendments to our paper.

4) Fig 3: "Figure 3: Digital subtraction angiography (DSA) revealed the condition of the spleen before and after artery embolization. Figure 2A: Imaging before splenic artery embolization. The black arrow indicates bleeding points. Figure 2B: Imaging after splenic artery embolization." Why did they label Fig 2A and 2B in Fig 3? In detail should be shown in the Figure legend.

   Thank you very much for your work and comment. We made some amendments to our paper.

5) Fig 4: "Figure 4: Plain and enhanced CT revealed multiple round shadows of low density in the spleen and liver. Figure 3A: Plain CT scan revealed the presence of circular hypo-density regions with variable densities in the left and right liver parenchyma. The spleen was enlarged, and multiple abnormal cystic solid dense shadows were observed in and around the spleen. Figure 3B: Results of the enhanced CT scan indicate slight enhancement of the solid components and no enhancement of the hypo-density regions." Why did they label Fig 3A and 3B in

Fig 4? In detail should be shown in the Figure legend with arrowheads for the region of interest, plus scale bars. How did they show "multiple round shadows?"

Thank you very much for your work and comment. We made some amendments to our paper.

6) Fig 5: "necrosis" – did they have any cellular and molecular assays to confirm?

   Thank you very much for your work and comment. Under the microscope, it can be seen that the tumor nucleus is fragmented, cytoplasm is concentrated, nuclear membrane is broken, and the pathological type of the tumor cannot be distinguished. Macroscopic view shows that the tissue lacks blood supply and appears as yellow-white tissue. The yellow-white area between the red hemorrhage and normal liver tissue shown in this specimen is the necrotic area. In general, we confirm necrosis by clinical experience and clinical practice. Actually, cellular and molecular assays are necessary, we will try to conduct cellular and molecular assays in future patients in our future work.

7) Fig 6: "Figure 6: HE staining and IHC of the specimen. Figure 5A: HE staining showed the morphology of the tumor cells. The tumor cells were arranged in sheets, fissures, or papillae with red cytoplasm. The nuclei were fusiform, oval, or irregular. Simultaneously, mitosis was easily seen. Figure 5B: IHC revealed that the patient was positive for CD31 and Ki-67, which was the characteristic of tumor cells. Figure 5C: The results of IHC revealed that the patient was positive for S-100 but negative for CD34. " Why did they label Fig 5A, 5B, 5C, in Fig 6? The detail of the description should be shown in the Figure legend with scale bars in the images. How did they tell which is which for positive for CD31 and Ki-67? So did it for S-100 but negative for CD34? Could they identify those with arrowheads in the images?

   Thank you very much for your work and comment. We made some amendments to our paper.

8) Fig 7: "Figure 7: The level of PDL1 protein was detected using IHC by the Dako PD-L1 IHC 22c3 PharmDx kit. Figure 6A: HE staining of the specimen. Figure 6B: Negative control for the test. Figure 6C: Positive control for the test. Figure 6D: IHC revealed that this patient was positive for PD-L1 in the cytomembrane of tumor cells (TPS = 20%, CPS = 22)." Why did they label Fig 6A, 6B, 6C, 6D in Fig

7? The detail of the description should be written in the Figure legend with scale bars and arrowheads in the images.

Thank you very much for your work and comment. We made some amendments to our paper.

9) Lines 133-134: "Splenectomy and liver tumor resection were performed not only to cure the disease but also to cure the disease and for the histopathological diagnosis." This statement contradicts their reasoning of Treatment with Sorafenib plus Camrelizumab – if surgery could cure, why did they need it?

Thank you very much for your work and comment. We made some amendments to our paper.

10) Lines 140-141: "The histopathological biopsy and next-generation sequencing (NGS) were then carried out" – Where did they have NGS data sets? What values of those biomarkers?

Thank you very much for your work and comment. We have provided gene list for this patient as illustrated in following table. And we also made some amendments to our paper to explain the mutation site in this patient.

免疫治疗相关基因													
ALK	*ATM*	*ATR*	*BRCA1*	*BRCA2*	*BRIP1*	*CCND1*	*CD274*	*CHEK1*	*CHEK2*	*DNMT3A*	*EGFR*	*FANCA*	*FGF19*
FGF3	*FGF4*	*JAK1*	*JAK2*	*KRAS*	*MDM2*	*MDM4*	*MLH1*	*MSH2*	*MSH6*	*PALB2*	*PBRM1*	*PDCD1LG2*	*PMS2*
POLD1	*POLE*	*PTEN*	*RAD50*	*STK11*	*TP53*								
靶向治疗和预后和耐药相关基因													
ABL1	*ABRAXAS1*	*AKT1*	*AKT2*	*AKT3*	*APC*	*AR*	*ARAF*	*ARID1A*	*ATM*	*ATR*	*AURKA*	*BARD1*	*BCL2L11*
BRAF	*BRCA1*	*BRCA2*	*BRIP1*	*BTK*	*CCND1*	*CCND3*	*CD274*	*CDK12*	*CDK4*	*CDK6*	*CDKN1B*	*CDKN2A*	*CDKN2B*
CHD1	*CHEK1*	*CHEK2*	*CRBN*	*CSF1R*	*CTNNB1*	*DDR2*	*DNMT3A*	*EGFR*	*EPCAM*	*EPHA2*	*EPHA3*	*ERBB2*	*ERBB3*
ERBB4	*ERCC3*	*ERRFI1*	*ESR1*	*EZH2*	*FANCE*	*FANCL*	*FAT1*	*FBXW7*	*FGF3*	*FGF4*	*FGFR1*	*FGFR2*	*FGFR3*
FGFR4	*FLCN*	*FLT1*	*FLT3*	*FLT4*	*FOXA1*	*FRS2*	*GEN1*	*GLI1*	*GLI2*	*GLI3*	*GNAS*	*HDAC2*	*HGF*
HOXB13	*HRAS*	*IDH1*	*IDH2*	*IGF1R*	*IGF2*	*IL7R*	*INPP4B*	*JAK1*	*JAK2*	*KDR*	*KIT*	*KRAS*	*LRP1B*
MAP2K1	*MAP2K2*	*MCL1*	*MDM2*	*MDM4*	*MET*	*MLH1*	*MLH3*	*MRE11*	*MSH2*	*MSH6*	*MTOR*	*MYC*	*MYCN*
NBN	*NF1*	*NF2*	*NFKBIA*	*NKX2-1*	*NRAS*	*NRG1*	*NTRK1*	*NTRK2*	*NTRK3*	*PALB2*	*PBRM1*	*PCDH9*	*PDCD1LG2*
PDGFRA	*PDGFRB*	*PIK3CA*	*PIK3R1*	*PIK3R2*	*PLCG2*	*PLXNA1*	*PML*	*PMS2*	*POLD1*	*POLE*	*PPP2R2A*	*PTCH1*	*PTEN*
RAC1	*RAD50*	*RAD51*	*RAD51B*	*RAD51D*	*RAD54L*	*RAF1*	*RARA*	*RB1*	*RET*	*RICTOR*	*RNF43*	*ROS1*	*RPTOR*
RXRA	*SETD2*	*SMARCA4*	*SMARCB1*	*SMO*	*SRC*	*STAG2*	*STK11*	*SYK*	*TP53*	*TSC1*	*TSC2*	*VEGFA*	*VHL*
ZBTB16	*ZNRF3*												

化疗相关基因													
ARID1A	ATM	ATR	BRCA1	BRCA2	CHEK1	CHEK2	CYP2C19	CYP2D6	DPYD	MLH1	RAD50	TPMT	UGT1A1
ZNF217													

DDR通路相关基因													
ABRAXAS1	ALKBH2	ALKBH3	APEX1	APEX2	APLF	APTX	ATM	ATR	ATRIP	BARD1	BLM	BRCA1	BRCA2
BRIP1	CCNH	CDK7	CENPS	CETN2	CHAF1A	CHEK1	CHEK2	CLK2	DCLRE1A	DCLRE1B	DCLRE1C	DDB1	DDB2
DMC1	DUT	EME1	EME2	ENDOV	ERCC1	ERCC2	ERCC3	ERCC4	ERCC5	ERCC6	ERCC8	EXO1	FAAP100
FAAP20	FAAP24	FAN1	FANCA	FANCB	FANCC	FANCD2	FANCE	FANCF	FANCG	FANCI	FANCL	FANCM	FEN1
GEN1	GTF2H1	GTF2H3	GTF2H4	GTF2H5	H2AFX	HELQ	HES1	HFM1	HLTF	HMGB1	HUS1	LIG1	LIG3
LIG4	MAD2L2	MBD4	MDC1	MGMT	MLH1	MLH3	MMS19	MNAT1	MPG	MPLKIP	MRE11	MSH2	MSH3
MSH4	MSH5	MSH6	MUS81	MUTYH	NABP2	NBN	NEIL1	NEIL2	NEIL3	NHEJ1	NTHL1	NUDT1	OGG1
PALB2	PARP1	PARP2	PARP3	PCNA	PER1	PMS1	PMS2	PNKP	POLB	POLD1	POLE	POLG	POLH
POLI	POLK	POLL	POLM	POLN	POLQ	PRKDC	PRPF19	RAD1	RAD18	RAD23A	RAD23B	RAD50	RAD51
RAD51B	RAD51C	RAD51D	RAD52	RAD54B	RAD54L	RAD9A	RBBP8	RDM1	RECQL	RECQL4	RECQL5	REV1	REV3L
RIF1	RMI1	RMI2	RNF168	RNF4	RNF8	RPA1	RPA2	RPA3	RPA4	RRM2B	SEM1	SETMAR	SHPRH
SLX1A	SLX4	SMUG1	SPO11	SPRTN	TDG	TDP1	TDP2	TOP3A	TOP3B	TOPBP1	TP53	TP53BP1	TREX1
TREX2	UBE2A	UBE2B	UBE2N	UBE2T	UBE2V2	UNG	USP1	UVSSA	WRN	XAB2	XPA	XPC	XRCC1
XRCC2	XRCC3	XRCC4	XRCC5	XRCC6									

白细胞抗原HLA		
HLA-A	HLA-B	HLA-C

融合基因													
ALK	BCR	BRAF	EGFR	ERBB2	ERBB4	ETV1	ETV4	ETV5	EWSR1	FGFR1	FGFR2	FGFR3	FGFR4
FLT3	FUS	HEY1	JAK2	KIT	KMT2A	MET	MYB	NAB2	NOTCH2	NR4A3	NRG1	NTRK1	NTRK2
NTRK3	NUTM1	PAX3	PAX7	PDGFB	PDGFRA	PDGFRB	RAF1	RARA	RET	ROS1	SS18	TFE3	TMPRSS2
YWHAE													

其他基因													
ABCB11	ABI1	ACKR3	ACSL3	ACVR1	ACVR1B	ACVR2A	AEN	AFF3	AFF4	AMER1	ANK1	APOBEC3B	AREG
ARHGAP5	ARID1B	ARID2	ARNT	ASXL1	ATP1A1	ATP2B3	ATRX	AXIN1	AXIN2	AXL	B2M	BAP1	BAZ1A
BCL10	BCL11A	BCL11B	BCL2	BCL2L1	BCL6	BCOR	BCORL1	BIRC3	BIRC5	BMP5	BMPR1A	BRD4	BTG1
BUB1B	CACNA1D	CALR	CAMTA1	CANT1	CARD11	CARS	CASP8	CBFA2T3	CBFB	CBL	CBLB	CCDC6	CCNB1IP1
CCND2	CCNE1	CCNO	CD74	CD79A	CD79B	CDC73	CDH1	CDH10	CDH11	CDK2	CDK8	CDKN1A	CDKN1C
CDKN2C	CDX2	CEBPA	CENPX	CHD2	CHD4	CHIC2	CIC	CIITA	CLIP1	CLTCL1	CNBP	CNOT3	COL7A1
CREB3L1	CREB3L2	CREBBP	CRKL	CRLF2	CRNKL1	CRTC1	CRTC3	CSF3R	CTCF	CTNND2	CTR9	CUL1	CUL3
CUL4A	CUL5	CUX1	CXCR4	CYLD	CYP17A1	CYSLTR2	DAXX	DDIT3	DDX10	DDX3X	DDX5	DDX6	DICER1
DIS3	DIS3L2	DKC1	DNM2	DNMT1	DNTT	DOCK8	DROSHA	EBF1	EED	EIF3E	EIF4A2	ELANE	ELF3
ELF4	ELK4	ELL	ELOA	EMSY	EP300	EPAS1	EPHA7	EPHB1	EPS15	ERC1	EREG	ERF	ETNK1
ETV6	EXT1	EXT2	EZR	FAH	FAM135B	FAM47C	FAS	FAT4	FES	FH	FHIT	FOXL2	FOXP1
FRK	FUBP1	G6PD	GALNT12	GAS7	GATA1	GATA2	GATA3	GBA	GFI1	GJB2	GNA11	GNA13	GNAQ
GPC3	GRB2	GREM1	GRIN2A	GSK3B	GSTT1	H3F3A	HDAC1	HFE	HIF1A	HIP1	HIST1H3B	HMBS	HMGA2
HNF1A	HNRNPA2B1	HOOK3	HOXA11	HUS1B	IKBKE	IKZF1	IL6ST	IRS2	ITGAV	ITK	JAK3	JMJD1C	JUN
KCNJ5	KDM5A	KDM5C	KDM6A	KEAP1	KLF4	KMT2C	KMT2D	KNL1	LASP1	LATS1	LATS2	LCK	LEF1
LIFR	LMNA	LMO1	LZTR1	MAP2K4	MAP3K1	MAPK1	MAX	MECOM	MED12	MEF2B	MEN1	MGA	MITF
MLLT3	MLST8	MPL	MTAP	MYCL	MYD88	MYOD1	NCOA3	NCOR1	NCOR2	NDRG1	NFE2L2	NFIB	NHP2
NME1	NONO	NOP10	NOTCH1	NOTCH3	NOTCH4	NPM1	NRG3	NSD2	NSD3	NT5C2	NUP93	PAK1	PARP4
PAX5	PAX8	PDPK1	PER2	PER3	PHF6	PHOX2B	PICALM	PIK3CB	PIK3CD	PIK3R3	PIM1	PLXNB1	POLD3
POLD4	POLE2	POLE3	POLE4	POT1	POU2AF1	POU5F1	PPARG	PPM1D	PPP2R1A	PPP4R1	PPP4R2	PPP4R3A	PPP4R3B
PPP4R4	PPP6C	PRCC	PRDM1	PRDM16	PRDM9	PREX2	PRF1	PRKACA	PRKAR1A	PRKCH	PRSS1	PSIP1	PTK2
PTK6	PTPN11	PTPN13	PTPRD	PTPRT	QKI	RAD21	RAD54L2	RAD9B	RANBP2	RAP1GDS1	RASA1	RBM10	RBX1
RFC1	RFC2	RFC3	RFC4	RFC5	RFWD3	RGS7	RHBDF2	RHEB	RHOA	RHOH	RIT1	RNF213	RPS6KA3
RPS6KB1	RUNX1	RUNX1T1	SBDS	SDC4	SDHA	SDHAF2	SDHB	SDHC	SDHD	SERPINA1	SERPINB3	SETBP1	SF3B1
SFPQ	SGK1	SH2B3	SH2D1A	SHOC2	SLC25A13	SLC29A1	SLC34A2	SLC45A3	SLIT2	SMAD2	SMAD3	SMAD4	SMARCA1
SMARCA2	SOCS1	SOS1	SOX2	SOX9	SPEN	SPOP	SPRED1	SPTA1	SRGAP3	SRSF2	SRY	STAT3	SUFU
SUZ12	TBL1XR1	TBX3	TCF3	TCF7L2	TCL1A	TEAD2	TELO2	TERT	TET1	TET2	TGFBR1	TGFBR2	THBS2
TIMELESS	TMEM127	TMEM189	TNFAIP3	TOP2A	TP63	TRAF7	TRIM37	TSHR	TSPAN31	TYK2	U2AF1	UROD	USP6
USP8	WAS	WDR48	WIF1	WT1	XPO1	YAP1	ZFHX3	ZNF479	ZNF703	ZNF750			

As for the method of NGS: Tissue processing and genomic DNA extraction Formalin-fixed paraffin-embedded (FFPE) tissue sections were evaluated for tumor cell content using hematoxylin and eosin (H&E) staining. Only samples with a tumor content of ≥20% were eligible for subsequent analyses. FFPE tissue sections were placed in a 1.5 microcentrifuge tube and deparaffinized with mineral oil. Samples were incubated with lysis buffer and proteinase K at 56 ° C overnight until the tissue was completely digested. The lysate was subsequently incubated at 80 °C for 4 hours to reverse formaldehyde crosslinks. Genomic DNA was isolated from tissue samples using the ReliaPrep™ FFPE gDNA Miniprep System (Promega) and quantified using the Qubit™ dsDNA HS Assay Kit (Thermo Fisher Scientific) following the manufacturer's instructions. DNA

extracts (30-200 ng) were sheared to 250 bp fragments using an S220 focused-ultrasonicator (Covaris). Libraries were prepared using the KAPA Hyper Prep Kit (KAPA Biosystems) following the manufacturer's protocol. The concentration and size distribution of each library were determined using a Qubit 3.0 fluorometer (Thermo Fisher Scientific) and a LabChip GX Touch HT Analyzer (PerkinElmer) respectively. For targeted capture, indexed libraries were subjected to probe-based hybridization with a customized NGS panel targeting exons of 733 cancer-related genes and introns of 733 frequently rearranged genes, where the probe baits were individually synthesized 5′ biotinylated 120 bp DNA oligonucleotides (IDT). Repetitive elements were filtered out from intronic baits according to the annotation by UCSC Genome RepeatMasker [1]. The xGen® Hybridization and Wash Kit (IDT) was employed for hybridization enrichment. Briefly, 500 ng indexed DNA libraries were pooled to obtain a total amount of 2 μg of DNA. The pooled DNA sample was then mixed with human cot DNA and xGen Universal Blockers-TS Mix and dried down in a SpeedVac system. The Hybridization Master Mix was added to the samples and incubated in a thermal cycler at 95℃ for 10 min, before being mixed and incubated with 4 μl of probes at 65℃ overnight. The target regions were captured following the manufacturer's instructions. The concentration and fragment size distribution of the final library were determined using a Qubit 3.0 fluorometer (Thermo Fisher Scientific) and a LabChip GX Touch HT Analyzer (PerkinElmer) respectively.

11) Lines 145: "positive for CD31, S-100, and Ki-67 (positive rate of 60%)," Where was their calculation?

Thank you very much for your work and comment. Ki67 is a nuclear protein that appears during the proliferation stage of the cell cycle and is stained brown after binding with antibodies. The stained cells account for 60% of all tumor cells under the microscope. Ki67 is a routine and common test in hospitals, and we do not show IHC images here. If necessary, we can provide the patient's pathology report.

12) Lines 140-146: Fig 6's resolution could not support these statements.

Thank you very much for your work and comment. Ki67 and S-100 are routine and common tests in hospitals, and we do not show IHC images here. If necessary, we can provide the patient's pathology report. "The histopathology revealed that the tumor cells were arranged in sheets, fissures, or papillae, with the cytoplasm in fusiform, oval, or irregular nuclei, and mitosis was easily seen."

Is from pathology report. If necessary, we can provide the patient's pathology report.

13) Table 1：Review of case reports published in the last 10 years (2011 to 2021), which indicated some patients of 27 cases survived for much longer than their case report: How did they conclude "targeted therapies and immunotherapy" were advantageous?

Thank you very much for your work and comment. As we mentioned in our **DISCUSSION**, despite the long survival of some patients, the prognosis for patients with liver metastases or splenic rupture after splenectomy was generally poor. OS of these patients may less than 6 mouths according to the retrospective analysis from Abbott RM et al (1). Most of PSA patients with liver metastases performed adjuvant chemotherapy after surgery and it seems that it may improve patients' survival according to some cases of **Table 1** in our paper. In this case, the patient suffered from liver metastases and splenic rupture, we believed that her prognosis may be poor if we didn't take any measure after surgery. After treated by targeted therapies and immunotherapy after surgery, the patient of this case has survived for one and a half year which is longer than some liver metastases cases with adjuvant chemotherapy in **Table 1**(2,3). However, we also realized that some cases showed excellent prognosis in some cases with adjuvant chemotherapy in Table 1(4,5), and the patient in this case is still alive, so it is difficult to compare the OS of this patient and patients with better OS in Table 1. So, as we mentioned in our **Conclusion**, adjuvant targeted therapy and immunotherapy **may** improve the prognosis in patients with PSA.

14) Lines 150-152: "The NGS revealed somatic mutations in the PDGFRA, KIT, KDR (VEGFR2), and TP53, while IHC showed the expression of PD-L1 (Figure 7)." This statement is misleading, as no data for PDGFRA, KIT, KDR (VEGFR2), and TP53. Did they PCR to confirm?

Thank you very much for your work and comment. We made some amendments to our paper to explain the mutation site in this patient. Due to the limited space of case report, we did not provide the data of NGS in the article, but we can provide you with the genetic test report of the patient. As for the PCR confirmation, since the 3DMed is qualified to conduct clinical laboratory developed test (LDT), we did not conduct PCR verification. In our opinion, the sensitivity of NGS (Sequencing Depth =35000X, LoD=0.1%-0.01%) was higher than that of REAL-TIME PCR. As for ddPCR, it is a good method to verify result

of NGS. However, our hospital does not have relevant qualifications. If conditions permit, we will conduct PCR verification in future patients.

genetic test report(only for reviewer): 

genetic test report

15) Lines 179-181: "Despite the prolonged survival of some patients, the prognosis for patients with liver metastases or splenic rupture after splenectomy was generally poor (Table 1)." Fig 2: "Red arrows indicate masses in the liver, while the white one indicates masses in the spleen" – simultaneous appearance? How did they verify which was the first and which was due to metastases?

Thank you very much for your work and comment. In our case, the patient was dignosed splenic hemangiosarcoma with liver metastasized, because We could see from imaging that splenic tumors were larger and numerous, while liver tumors were relatively small and uniform in size, which was consistent with the characteristics of metastatic liver cancer. In addition, our patient had no history of hepatitis, preoperative AFP was normal, CT examination showed no obvious characteristics of primary liver cancer, and blood from spleen returned to liver is the way of tumor to metastasise, which further supported the diagnosis of spleen tumor metastasized to the liver.

16) In Discussion, Lines 244 – 246: "Therefore, we performed NGS and IHC for PD-L1, and fortunately, the patient was sensitive to sorafenib and PD-L1 inhibitor and received periodic treatment." What was the data?

Thank you very much for your work and comment. This statement is misleading, so we made some amendments to our paper. Although there is no guideline for PSA, we found that Sorafenib could target these mutated genes in the patient. Thus we thought that the patient may benefit from Sorafenib (6). The PDL1 was positive (Dako 22C3, TPS=20%， CPS=22) base on the report, we believed that this patient may also benefit from ICI base on clinical practice in other tumors (7). This was an attempt of off label use.

17) Lines 246 – 251: "However, After 15 months of follow-up, there is no progress or recurrence of the disease, and the prognosis is good compared to other patients without adjuvant therapy. However, there was no quantitative assessment in this

patient." What did they mean "compared to other patients without adjuvant therapy?"

Thank you very much for your work and comment. This statement is misleading, so we made some amendments to our paper. Actually, after 15 months of follow-up, there is no progress or recurrence of the disease, and the prognosis is good compared to other PSA patients of liver metastases without adjuvant therapy in cases published in the last 10 years (Table1).

18) the authors should update the literature on subclonal evolution in targeted therapies and immunotherapy. e.g., Hunting down the dominating subclone of cancer stem cells as a potential new therapeutic target in multiple myeloma: An artificial intelligence perspective. World J Stem Cells. 2020 Aug 26;12(8):706-720. doi: 10.4252/wjsc.v12.i8.706. Review. PubMed PMID: 32952853; PubMed Central PMCID: PMC7477658.

Thank you very much for your work and comment. We added and updated literatures in our paper.

(1) Abbott RM, Levy AD, Aguilera NS, Gorospe L, Thompson WM. From the archives of the AFIP: primary vascular neoplasms of the spleen: radiologic-pathologic correlation. Radiographics. 2004 Jul-Aug;24(4):1137-63. doi: 10.1148/rg.244045006. PMID: 15256634.
(2) Batouli A, Fairbrother SW, Silverman JF, Muniz Mde L, Taylor KB, Welnick MA, Mancini SA, Hartman MS. Primary Splenic Angiosarcoma: Clinical and Imaging Manifestations of This Rare Aggressive Neoplasm. Current problems in diagnostic radiology 2016; 45(4): 284–287
(3) Cho EA, Choi WY, Kim SH, Hong JY, Jung SH, Kim MJ, Hwang JE, Bae WK, Shim HJ, Lee KH, Cho SH, Chung IJ. Rapidly progressing primary splenic angiosarcoma with fatal hemorrhagic event. Journal of chemotherapy (Florence, Italy) 2014; 26(4): 248–252
(4) Xu L, Zhang Y, Zhao H, Chen Q, Ma W, Li L. Well-differentiated angiosarcoma of spleen: a teaching case mimicking hemagioma and cytogenetic analysis with array comparative genomic hybridization. World journal of surgical oncology 2015; 13: 300
(5) Hadidy A, Alsharif A, Sheikh-Ali R, Abukhalaf M, Awidi A, Abukaraki A, Nimri C, Omari A. Odontogenic myxofibroma synchronous with primary angiosarcoma of the spleen. The British journal of radiology 2010; 83(985): e10–13

(6) Pacey S, Ratain MJ, Flaherty KT, Kaye SB, Cupit L, Rowinsky EK, Xia C, O'Dwyer PJ, Judson IR. Efficacy and safety of sorafenib in a subset of patients with advanced soft tissue sarcoma from a Phase II randomized discontinuation trial. Invest New Drugs. 2011 Jun;29(3):481-8. doi: 10.1007/s10637-009-9367-9. Epub 2009 Dec 18. PMID: 20016927.
(7) Tamura K, Hasegawa K, Katsumata N, Matsumoto K, Mukai H, Takahashi S, Nomura H, Minami H. Efficacy and safety of nivolumab in Japanese patients with uterine cervical cancer, uterine corpus cancer, or soft tissue sarcoma: Multicenter, open-label phase 2 trial. Cancer Sci. 2019 Sep;110(9):2894-2904. doi: 10.1111/cas.14148. Epub 2019 Sep 3. PMID: 31348579; PMCID: PMC6726684.