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Durable complete response in BCLC stage C hepatocellular carcinoma with portal vein tumour invasion following sorafenib and stereotactic ablative radiotherapy: a case report

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Abstract

We report the case of a 62-year-old male patient with Barcelona Clinic Liver Cancer (BCLC) stage C hepatocellular carcinoma (HCC), diagnosed clinically in 2020 on the basis of characteristic imaging and markedly elevated alpha-fetoprotein (AFP) of 278,835 IU/mL. He had a history of hepatitis C infection, successfully treated in 2014, and background liver cirrhosis was suspected radiologically. Initial imaging demonstrated a massive polylobulated hepatic tumour measuring approximately 13 cm, centred on segment IV and extending into segments VIII, V and I, with portal vein tumour invasion involving the main portal vein and right and left portal venous branches.

The patient commenced sorafenib in December 2020 and subsequently received stereotactic ablative radiotherapy (SABR) to residual viable disease in May 2021, delivered as 50 Gy in 5 fractions. He demonstrated a marked biochemical and radiological response, with AFP falling from 278,835 IU/mL to 2 IU/mL by December 2021. Sorafenib was discontinued in January 2022, and the patient continued on surveillance. He was discussed for liver transplantation and surgical options following response, but was not considered a transplant candidate and remained under imaging surveillance.

Serial follow-up has shown sustained remission. Magnetic resonance imaging (MRI) liver at Month 62 demonstrated an unchanged treated segment IVa lesion measuring 19 mm without enhancement, consistent with Liver Imaging Reporting and Data System Treatment Response (LR-TR) non-viable disease. There was no evidence of recurrent or new hepatic disease, no ascites, and patent portal and hepatic veins. AFP remained suppressed at 1 IU/mL, and the patient maintained Eastern Cooperative Oncology Group (ECOG) performance status 0. This case illustrates an exceptional durable complete response in advanced HCC with portal vein tumour invasion following combined systemic therapy and SABR.

Categories: Radiation Oncology, Oncology, Gastroenterology
Keywords: barcelona clinic liver cancer (bclc) staging, hepatocellular carcinoma (hcc), sabr, sorafenib, tyrosine kinase receptor inhibitors

Introduction

Hepatocellular carcinoma (HCC) with macrovascular invasion is associated with advanced-stage disease and limited curative options. Portal vein tumour invasion is an adverse prognostic feature and classifies disease as Barcelona Clinic Liver Cancer (BCLC) stage C [1]. Systemic therapy is generally the standard approach in this setting, although selected patients may benefit from local consolidative treatment when a meaningful response is achieved [1-4].

Sorafenib was historically the standard first-line systemic treatment for advanced HCC in patients with preserved liver function, based on phase III evidence demonstrating an overall survival benefit compared with placebo [2,3]. Stereotactic ablative radiotherapy (SABR) has also been used as a local treatment option in selected patients with HCC, including cases with vascular invasion where local disease control may be clinically meaningful [4].

We describe a patient with initially extensive BCLC stage C HCC, including main portal vein tumour invasion and markedly elevated alpha-fetoprotein (AFP), who achieved a durable complete response following sorafenib and SABR.

Case Presentation

Case Presentation

A male patient born in February 1963 presented in 2020 with recurrent episodes of sudden severe right upper quadrant abdominal pain, radiating to the right shoulder tip. The pain reached maximal severity within minutes and was associated with constitutional decline, including weakness, fatigue and reduced performance status, initially Eastern Cooperative Oncology Group (ECOG) performance status 1. He also reported intermittent alternating diarrhoea and constipation. One of the severe pain episodes occurred while he was in the United States, where he underwent initial imaging before returning to the United Kingdom.

His past medical history included previously treated hepatitis C infection. He had initially received alpha-interferon and ribavirin in 2007 without successful viral clearance, but was subsequently cured in 2014 using ribavirin and a protease inhibitor. At that time, he was considered non-cirrhotic, although some fibrosis was present. Additional past medical history included low-grade non-muscle invasive transitional cell carcinoma of the bladder, treated with transurethral resections and intravesical mitomycin-C, as well as previous renal calculi. He remained under annual cystoscopic surveillance. He also had hypertension, treated with lacidipine 2 mg once daily.

At Month 0, imported imaging from the United States was reviewed at the hepatopancreatobiliary multidisciplinary team meeting. Imaging demonstrated a large hepatic mass measuring approximately 12-13 cm, involving segments VIII, IV and V, with direct invasion of the main portal vein. No extrahepatic abdominal disease was identified, although computed tomography (CT) chest staging had not been performed. Alpha-fetoprotein (AFP) was markedly elevated at 278,835 IU/mL. The diagnosis was Barcelona Clinic Liver Cancer (BCLC) stage C hepatocellular carcinoma (HCC), made clinically on the basis of characteristic imaging, portal vein tumour invasion and very high AFP.

Baseline magnetic resonance imaging (MRI) confirmed a massive polylobulated tumour centred predominantly on segment IV, extending into segments VIII, V and I, with an exophytic anterior component and central necrosis. There was angioinvasion of the portal vein and its branches supplying both the right and left liver. The dominant lesion measured 13 cm in maximal transverse diameter. Smaller daughter lesions were present, including a 16 mm lesion in segment VII. Tumoural infiltration was also seen at the liver hilum. The retrohepatic inferior vena cava and hepatic veins were compressed or laminated but not infiltrated. No free intraperitoneal fluid was seen on that examination. The baseline MRI findings are shown in Figure *1*.

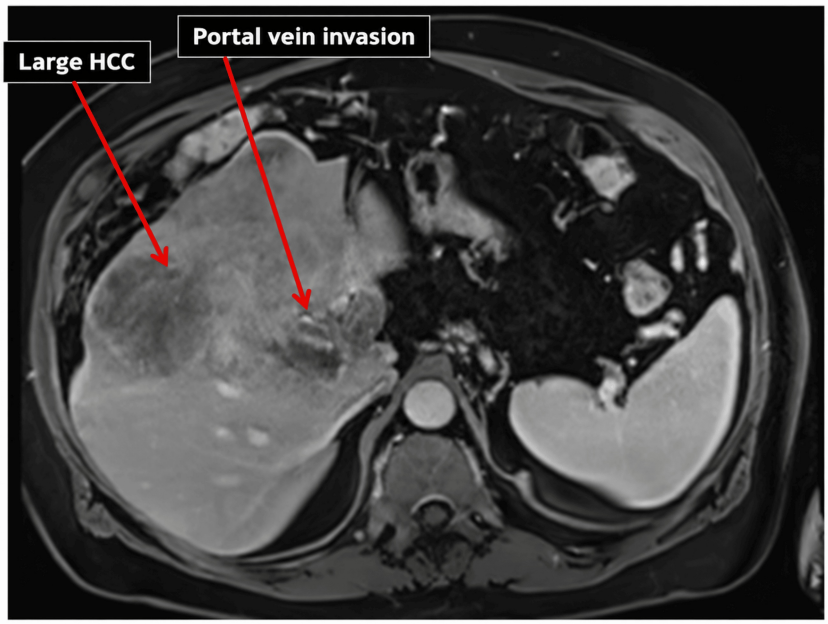


FIGURE 1: Baseline MRI liver image

Baseline MRI liver image. Axial T1-weighted fat-suppressed post-contrast delayed phase MRI image at diagnosis demonstrates a large tumour burden predominantly within segments VIII and IVa, with radiological features consistent with HCC including washout and enhancing capsule. Labels highlight the large HCC and suspected portal vein invasion. Abbreviations: HCC, hepatocellular carcinoma; MRI, magnetic resonance imaging.

Treatment

The patient commenced sorafenib in Month 0. He demonstrated an early biochemical response, with AFP falling from 278,835 IU/mL at baseline to 44,505 IU/mL by Month 2, 7,732 IU/mL by Month 3, and 775 IU/mL by Month 4.

MRI at Month 3 showed a marked reduction in tumour volume, with the tumour measuring approximately 73 × 46 mm. Further imaging demonstrated ongoing treatment response. At Month 5, due to residual viable disease, he received stereotactic ablative radiotherapy (SABR), delivered as 50 Gray (Gy) in 5 fractions on alternate days, using an active breathing coordinator motion-management strategy on a linear accelerator.

Following SABR, AFP continued to fall substantially, reaching 50.5 IU/mL at Month 5, 2.7 IU/mL at Month 6, and approximately 2 IU/mL by Month 12. Imaging also showed continued response. Computed tomography (CT) at Month 6 demonstrated reduction in tumour size to approximately 58 mm, with post-SABR changes. MRI at Month 8 showed no arterial phase hyperenhancement and a residual lesion measuring approximately 51 mm, with stable portal vein thrombus. CT at Month 9 was limited by an inadequate arterial phase and showed a low-attenuation abnormality in segment IV; this finding had already been assessed on the Month 8 MRI and was not considered to represent definite viable disease.

By Month 12, MRI showed no convincing arterial phase hyperenhancement or washout and a residual lesion measuring approximately 48 mm. CT chest imaging showed no lung metastases. The case was discussed by the hepatopancreatobiliary and transplant teams. Although there had been a major response to sorafenib and SABR, he was not considered a liver transplant candidate because of the original tumour burden and vascular invasion. Surgical resection with cholecystectomy and segment V liver resection was considered technically possible if active tumour recurred, but in the absence of radiological evidence of viable disease, surveillance was recommended.

Sorafenib was discontinued at Month 13 after achieving an essentially complete response.

Follow-up and outcome

From Month 13 onward, the patient remained off systemic therapy and continued on surveillance. His liver function remained preserved, with subsequent Child-Pugh class A status. He maintained excellent functional status, with ECOG performance status 0.

AFP remained persistently low, generally between 1 and 2 IU/mL, from late 2021 onward. CT thorax at Month 34 demonstrated no evidence of metastatic disease. MRI liver at Month 34 showed stable appearances, and continued surveillance was advised.

At Month 53, CT and MRI demonstrated a Liver Imaging Reporting and Data System (LI-RADS) 3 lesion in segment VIII, and repeat imaging was planned. MRI liver with contrast at Month 56 demonstrated a stable 5 mm right lobe subcapsular cyst and no evidence of recurrent or new disease.

Most recently, MRI liver with Gadovist contrast at Month 62 demonstrated cirrhosis without features of portal hypertension. There was no ascites, and the portal and hepatic veins were patent. The treated lesion in segment IVa was unchanged in size, measuring 19 mm, and showed no enhancement, consistent with LI-RADS treatment response (LR-TR) non-viable disease. No further focus of arterial phase hyperenhancement was identified, and no new hepatic lesion was demonstrated. Simple hepatic cysts were present elsewhere. There was no periportal lymphadenopathy, and appearances elsewhere were unchanged. AFP at Month 63 was 1 IU/mL. The post-treatment MRI appearance is shown in Figure *2*, and the serial AFP trend is shown in Figure *3*.

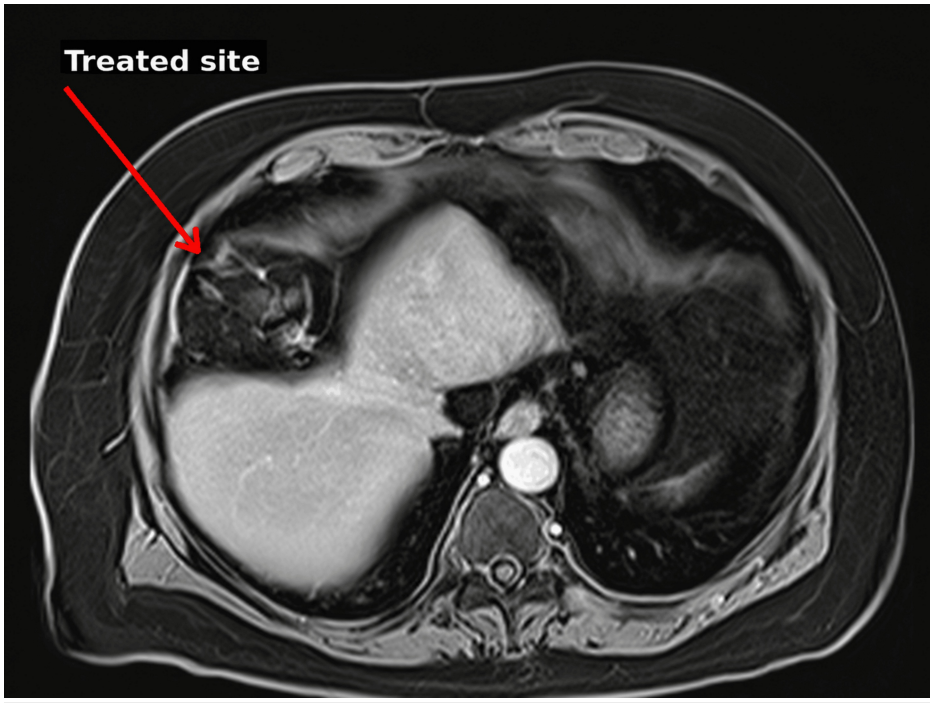


FIGURE 2: Follow-up MRI liver image

Follow-up MRI liver image. Axial T1-weighted fat-suppressed post-contrast delayed phase MRI image at Month 62 demonstrates complete resolution of previously demonstrated viable tumour with capsular retraction at the treated site and no evidence of residual enhancing HCC. Abbreviations: HCC, hepatocellular carcinoma; MRI, magnetic resonance imaging.

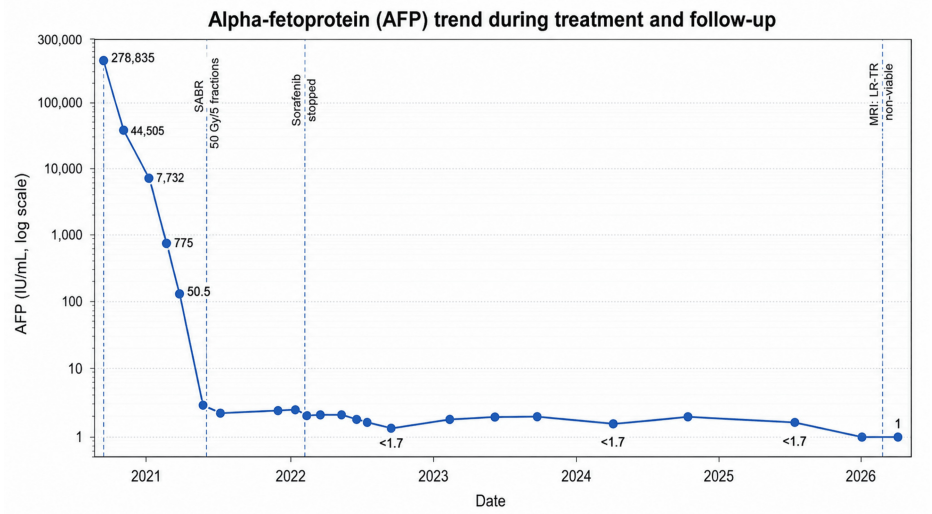


FIGURE 3: Serial alpha-fetoprotein trend from diagnosis through follow-up

Serial alpha-fetoprotein trend from diagnosis through follow-up. AFP was markedly elevated at diagnosis, reaching 278,835 IU/mL, and declined rapidly following sorafenib and stereotactic ablative radiotherapy. AFP has remained suppressed from late 2021 onward, with the latest value of 1 IU/mL in March 2026.

At latest follow-up, the patient remains clinically well, off systemic therapy, with no radiological evidence of recurrent hepatocellular carcinoma.

Laboratory Findings

Available laboratory findings are summarised in Table *1*. Only documented values are included; unavailable

parameters have not been tabulated. Reference ranges are provided for context and may vary between laboratories.

Relative time point	Clinical context	Test	Result	Unit	Reference range
Month 0	**Diagnosis / pre-treatment**	**AFP**	**278,835**	**IU/mL**	**<7**
Month 2	On sorafenib	AFP	44,505	IU/mL	<7
Month 3	On sorafenib	AFP	7,732	IU/mL	<7
Month 4	On sorafenib	AFP	775	IU/mL	<7
Month 5	Around SABR	AFP	50.5	IU/mL	<7
Month 6	Post-SABR	AFP	2.7	IU/mL	<7
Month 8	Post-SABR surveillance	AFP	1.9	IU/mL	<7
Month 12	Deep response	AFP	2	IU/mL	<7
Month 13	Sorafenib stopped	AFP	2	IU/mL	<7
Month 15	Surveillance	AFP	1.7	IU/mL	<7
Month 16	Surveillance	AFP	1.8	IU/mL	<7
Month 17	Surveillance	AFP	1.8	IU/mL	<7
Month 18	Surveillance	AFP	1.5	IU/mL	<7
Month 19	Surveillance	AFP	1.4	IU/mL	<7
Month 21	Surveillance	AFP	1.2	IU/mL	<7
Month 27	Surveillance	AFP	<1.7	IU/mL	<7
Month 31	Surveillance	AFP	2	IU/mL	<7
Month 34	Surveillance	AFP	2	IU/mL	<7
Month 40	Surveillance	AFP	<1.7	IU/mL	<7
Month 46	Surveillance	AFP	2	IU/mL	<7
Month 54	Surveillance	AFP	<1.7	IU/mL	<7
Month 59	Surveillance	AFP	1	IU/mL	<7
Month 63	Latest follow-up	AFP	1	IU/mL	<7
Month 12	Deep response	Albumin	43	g/L	35-50
Month 12	Deep response	Bilirubin	14	micromol/L	0-21
Month 12	Deep response	Alkaline phosphatase	67	IU/L	30-130
Month 12	Deep response	Alanine aminotransferase	123	IU/L	0-50
Month 12	Deep response	Creatinine	74	micromol/L	60-120
Month 12	Deep response	Haemoglobin	172	g/L	130-180
Month 12	Deep response	Platelets	162	x10^9/L	150-400
Month 34	Surveillance	Platelets	174	x10^9/L	150-400

TABLE 1: Available laboratory findings during treatment and follow-up.

Abbreviations: AFP, alpha-fetoprotein; SABR, stereotactic ablative radiotherapy. Values reported as less than a threshold are presented as documented by the reporting laboratory. Reference ranges are approximate adult reference ranges and should be interpreted according to local laboratory standards.

Timeline

The clinical course is summarised using relative time points from diagnosis in Table 2.

Relative time point from diagnosis	Clinical event
Month 0	**Diagnosis of BCLC stage C HCC with large hepatic tumour, daughter lesions, and portal vein tumour invasion; AFP 278,835 IU/mL; sorafenib commenced.**
Month 2	AFP decreased to 44,505 IU/mL.
Month 3	AFP decreased to 7,732 IU/mL; MRI showed reduction in tumour volume to approximately 73 x 46 mm.
Month 4	AFP decreased to 775 IU/mL.
Month 5	SABR delivered to residual viable disease as 50 Gy in 5 fractions on alternate days.
Month 6	AFP decreased to 2.7 IU/mL; CT showed reduction in tumour size to approximately 58 mm with post-SABR changes.
Month 8	MRI showed no arterial phase hyperenhancement, residual lesion approximately 51 mm, and stable portal vein thrombus.
Month 12	MRI showed no convincing arterial phase hyperenhancement or washout; CT chest showed no lung metastases.
Month 13	Sorafenib discontinued after essentially complete response.
Month 34	CT thorax showed no metastatic disease; MRI liver showed stable appearances.
Month 53	LI-RADS 3 segment VIII observation identified; repeat imaging arranged.
Month 56	MRI showed a stable 5 mm right lobe subcapsular cyst and no recurrent or new disease.
Month 62	MRI showed stable 19 mm treated segment IVa lesion without enhancement, LR-TR non-viable.
Month 63	AFP remained suppressed at 1 IU/mL; patient clinically well with ECOG performance status 0.

TABLE 2: Timeline of clinical course using relative time points.

Abbreviations: AFP, alpha-fetoprotein; BCLC, Barcelona Clinic Liver Cancer; CT, computed tomography; ECOG, Eastern Cooperative Oncology Group; HCC, hepatocellular carcinoma; LI-RADS, Liver Imaging Reporting and Data System; LR-TR, Liver Imaging Reporting and Data System Treatment Response; MRI, magnetic resonance imaging; SABR, stereotactic ablative radiotherapy.

Discussion

This case describes an exceptional durable complete response in a patient with initially advanced Barcelona Clinic Liver Cancer (BCLC) stage C hepatocellular carcinoma (HCC), characterised by a very large intrahepatic tumour, daughter lesions, and main portal vein tumour invasion. The initial alpha-fetoprotein (AFP) of 278,835 IU/mL reflected a high tumour burden and biologically active disease. Macrovascular invasion is a major adverse disease feature and is incorporated into advanced-stage classification within the BCLC framework [1].

The clinical course is notable for several reasons. First, the patient achieved a profound biochemical response to sorafenib, with AFP falling from nearly 280,000 IU/mL to within the normal range. Second, consolidative stereotactic ablative radiotherapy (SABR) to residual viable disease was associated with continued radiological and biochemical response. Third, despite the initial presence of macrovascular invasion, he has remained free of viable disease for more than four years after stopping systemic therapy.

For many years, sorafenib was the benchmark systemic treatment for advanced HCC. In the SHARP trial, sorafenib improved median overall survival compared with placebo from 7.9 months to 10.7 months [2]. The Asia-Pacific phase III trial confirmed benefit in a population with more advanced disease characteristics, with median overall survival of 6.5 months with sorafenib compared with 4.2 months with placebo [3].

The depth and duration of response in this case are therefore unusual. Complete and durable responses to sorafenib are rarely reported, particularly in patients with portal vein tumour invasion. The addition of SABR may have contributed to local control of residual disease. Radiotherapy-based approaches have been

evaluated in HCC with portal vein thrombosis, and systematic review evidence suggests that selected patients may derive local control benefit [4]. In the present case, SABR was delivered using respiratory motion management, which is important in liver radiotherapy because of tumour movement during breathing and the proximity of normal liver and adjacent organs at risk.

Real-world evidence has also supported the feasibility of sorafenib in routine practice, while emphasising the importance of preserved liver function and careful clinical selection [5,6]. These factors are relevant to the present case, as the patient maintained preserved liver function and good performance status, allowing him to receive systemic therapy, consolidative SABR, and long-term surveillance.

This case also highlights the value of integrating biochemical and radiological response assessment. The patient had a rapid and sustained AFP decline, which paralleled the imaging response. Later MRI appearances were consistent with Liver Imaging Reporting and Data System Treatment Response (LR-TR) non-viable disease, a standardised imaging category used to describe treated observations without features of viable tumour [7]. Although AFP cannot replace cross-sectional imaging, it was a useful marker of response and long-term disease control in this patient.

The case was repeatedly reviewed in multidisciplinary settings, including hepatopancreatobiliary and transplant discussions. Although the response was sufficient to prompt consideration of radical options, transplantation was not pursued because of the original tumour burden and vascular invasion. Surgical resection was considered only if active disease recurred; however, continued surveillance was recommended in the absence of viable tumour.

This report has limitations. It describes a single patient and therefore cannot establish treatment efficacy or generalisability. The diagnosis was made clinically using characteristic imaging and markedly elevated AFP rather than biopsy, which is consistent with accepted diagnostic pathways for HCC in appropriate clinical and radiological contexts but remains a limitation. The case nevertheless illustrates that selected patients with advanced HCC and preserved performance status may achieve durable disease control when systemic therapy is integrated with local treatment to residual active disease.

Conclusions

A male patient with initially extensive BCLC stage C hepatocellular carcinoma, main portal vein tumour invasion and AFP of 278,835 IU/mL achieved a durable complete response following sorafenib and SABR. He remains off systemic therapy with AFP of 1 IU/mL and MRI evidence of LR-TR non-viable disease in February 2026. This case highlights the potential for long-term disease control in selected patients with advanced HCC treated using combined systemic and local ablative approaches.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Bahaaeldin Baraka, Rahul Munyal, Abdus Samee, Arvind Arora

Acquisition, analysis, or interpretation of data: Bahaaeldin Baraka, Luis Aznar-García, Arvind Arora

Drafting of the manuscript: Bahaaeldin Baraka, Rahul Munyal, Luis Aznar-García, Abdus Samee, Arvind Arora

Critical review of the manuscript for important intellectual content: Bahaaeldin Baraka, Abdus Samee, Arvind Arora

Supervision: Bahaaeldin Baraka, Arvind Arora

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. issued approval Not applicable. This case report was reviewed by the local audit lead and was deemed not to require formal research ethics committee approval. Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical information. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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