HOME

FDA Approves a New Targeted Breast Cancer Pill That May Help Lower the Risk of Progression or Death

By bestfolkmedicine · 09/09/2026 · 8 min read



For many people living with advanced breast cancer, one of the most frustrating realities is that a treatment can appear to be working while the tumor is quietly developing resistance beneath the surface.

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Now, a newly FDA-approved treatment introduces a different strategy: **look for molecular signs of resistance in the blood before scans show that the cancer is growing—and change treatment earlier.**

On September 4, 2026, the U.S. Food and Drug Administration granted accelerated approval to **camizestrant, sold under the brand name Etcamah**, in combination with a CDK4/6 inhibitor for certain adults with hormone receptor-positive, HER2-negative advanced or metastatic breast cancer.

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The development is particularly interesting because the decision to switch treatment can be guided by a **blood-based circulating tumor DNA test**, rather than waiting for visible disease progression on imaging.

And in a major Phase III clinical trial, this approach reduced the risk of disease progression or death by **56%** compared with continuing standard treatment.

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That number deserves attention—but also some careful explanation.

What Exactly Did the FDA Approve?

Etcamah is an oral medicine called an **estrogen receptor antagonist**.

It is approved for adults with:

- Hormone receptor-positive breast cancer
- HER2-negative breast cancer
- Locally advanced or metastatic disease
- An acquired **ESR1 mutation**
- No visible disease progression yet
- Current treatment with an aromatase inhibitor plus a CDK4/6 inhibitor

The treatment is **not used alone**.

Patients switch from their aromatase inhibitor—such as anastrozole or letrozole—to camizestrant while continuing the same CDK4/6 inhibitor, which may be:

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- Abemaciclib
- Palbociclib
- Ribociclib

The recommended camizestrant dose is 75 mg orally once daily.

Why the ESR1 Mutation Matters

Most hormone receptor-positive breast cancers rely, at least partly, on estrogen signaling.

That is why endocrine therapies that reduce estrogen production or interfere with estrogen receptors are so important.

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But cancer cells can evolve.

One important mechanism of resistance involves mutations in a gene called **ESR1**, which contains instructions for the estrogen receptor.

An ESR1 mutation can allow the cancer to continue using estrogen receptor signaling even when estrogen levels have been suppressed by aromatase inhibitors.

These mutations often emerge during treatment.

Instead of waiting until the tumor becomes larger or appears on a scan, the new strategy looks for traces of the mutation in **circulating tumor DNA, or ctDNA**, found in the bloodstream.

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A Blood Test May Reveal Resistance Before a Scan Does

Tumors continuously release tiny fragments of DNA into the bloodstream.

Modern liquid-biopsy technology can analyze those fragments and look for genetic changes associated with treatment resistance.

The FDA simultaneously authorized **Guardant360 CDx** as a companion diagnostic test for identifying eligible patients with ESR1 mutations.

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That creates a potentially significant change in cancer care:

Instead of waiting for cancer to visibly progress, doctors may be able to detect molecular resistance and intervene earlier.

This is the first FDA approval of a cancer treatment guided by detection of a resistance mutation in circulating tumor DNA before imaging confirms disease progression.

The Study Behind the Approval

The FDA decision was largely based on the Phase III **SERENA-6 trial**.

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Researchers initially screened more than 3,200 patients for emerging ESR1 mutations. Ultimately, 315 eligible patients whose tumors developed an ESR1 mutation while receiving first-line endocrine therapy were randomly assigned to one of two strategies.

One group:

Switched from an aromatase inhibitor to camizestrant while continuing a CDK4/6 inhibitor.

The other:

Continued their aromatase inhibitor plus CDK4/6 inhibitor.

Importantly, these patients had not yet developed obvious disease progression on imaging.

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Where the “56% Lower Risk” Comes From

The study’s primary outcome was progression-free survival—the amount of time patients lived without their cancer progressing or dying.

Median progression-free survival was:

16 months with camizestrant + CDK4/6 therapy

compared with:

9.2 months with continued aromatase inhibitor + CDK4/6 therapy.

The hazard ratio was 0.44, corresponding to a **56% relative reduction in the risk of progression or death during the study period.**

That is an important result.

But there is an equally important distinction:

It does NOT mean camizestrant reduced the risk of dying from breast cancer by 56%.

The trial endpoint combined **disease progression or death**.

Overall survival data were still immature when the FDA evaluated the treatment.

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This distinction matters whenever impressive percentages appear in health headlines.

Researchers Also Saw Longer-Term Disease Control

Follow-up analyses have continued to look at what happens after patients eventually need another treatment.

In an updated analysis, median time to a second disease progression or death was:

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- **25.7 months** in the camizestrant group
- **19.1 months** in the control group

Researchers also reported substantial decreases in detectable circulating tumor DNA among many patients receiving camizestrant. These findings are encouraging but do not replace the need for longer-term survival data.

Why the FDA Used Accelerated Approval

Etcamah received **accelerated approval**, which is different from saying every question about the treatment has been answered.

The FDA specifically noted that it remains uncertain whether changing therapy when an ESR1 mutation first appears—rather than waiting for confirmed disease progression—will ultimately translate into a clinically meaningful long-term survival benefit.

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Because of that uncertainty, continued approval may depend on confirmatory studies that verify the clinical benefit.

That context is important.

This is a promising new treatment strategy—not proof that advanced breast cancer has become easy to treat.

Are There Side Effects?

Yes.

Cancer treatments capable of altering hormone signaling and cell growth can have significant effects elsewhere in the body.

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The FDA labeling includes warnings concerning:

- **Slow heart rate, or bradycardia**
- QT interval changes that may increase the risk of abnormal heart rhythms
- Potential harm to an unborn baby

When camizestrant is combined with certain drugs that affect cardiac rhythm, monitoring may be particularly important.

Patients should never adjust cancer medication without their oncology team.

Natural Supplements Require Extra Caution During Cancer Treatment

This is especially important for readers interested in herbal medicine.

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Herbs can be pharmacologically active, and that means they can sometimes interfere with prescription medications.

For example, official product information specifically identifies **St. John's wort** as a strong CYP3A4/5 inducer that can reduce camizestrant exposure and should be avoided.

Other medications may also interact.

Anyone taking Etcamah—or any targeted cancer therapy—should give their oncologist or oncology pharmacist a complete list of:

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- Herbal supplements
- Vitamins
- Teas and tinctures
- Mushroom products
- CBD or cannabis products
- Over-the-counter medicines
- Prescription drugs

"Natural" does not automatically mean interaction-free.

What Holistic Support Can Safely Complement Treatment?

Holistic cancer care should mean **supporting the whole person while evidence-based oncology treats the cancer**, not replacing proven treatment.

Depending on a patient's condition and medical team, supportive habits may include:

Eat Enough Protein and Nutrient-Dense Foods

Cancer and treatment can affect appetite, muscle mass, and energy.

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Helpful foods may include:

- Eggs
- Fish
- Yogurt
- Beans and lentils
- Nuts and seeds
- Vegetables
- Berries
- Whole grains
- Olive oil

Patients struggling with nausea, weight loss, mouth sores, or digestive problems may benefit from an oncology dietitian rather than trying a restrictive “anti-cancer diet.”

Keep Moving When Possible

Even gentle movement—walking, stretching, resistance exercises, or supervised rehabilitation—may help preserve strength and improve quality of life.

Exercise should always match the person’s energy level, bone health, blood counts, treatment side effects, and metastatic sites.

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Protect Sleep

Cancer treatment can disrupt sleep through anxiety, pain, hormonal changes, medications, or hot flashes.

A simple evening routine can help:

dim lighting, regular sleep hours, gentle breathing, less screen exposure, and a comfortable bedroom environment.

Use Mindfulness as Support, Not Treatment

Meditation, breathing exercises, journaling, prayer, gentle yoga, and counseling cannot eliminate cancer.

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They can, however, help some people cope with uncertainty, anxiety, and the emotional burden of long-term treatment.

That matters too.

Who Is This Treatment For?

Etcamah is not a general breast cancer pill.

It is designed for a **specific molecular subgroup** of patients with advanced HR-positive, HER2-negative breast cancer whose tumors develop an ESR1 mutation during first-line treatment.

That is why biomarker testing is central to this approach.

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Two people can both be diagnosed with breast cancer and require completely different treatments because their tumors behave differently at the molecular level.

Modern oncology is increasingly moving away from treating cancer solely according to where it started and toward understanding **what is driving an individual tumor.**

A Glimpse of Where Cancer Treatment Is Heading

Perhaps the most fascinating part of this approval is not simply that another breast cancer medicine became available.

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It is **when treatment can now be changed.**

Traditionally, clinicians often wait for symptoms or imaging to reveal that treatment is failing.

With ctDNA monitoring, doctors may increasingly be able to see molecular resistance developing months earlier.

In other words, cancer care is beginning to move from:

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“The tumor has progressed—what do we do next?”

toward:

“The tumor is beginning to evolve—can we intervene before progression happens?”

That shift could eventually influence treatment far beyond breast cancer.

The Bottom Line

The FDA approval of camizestrant represents an important step toward more personalized and earlier intervention in advanced breast cancer.

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For selected patients with HR-positive, HER2-negative disease who develop an ESR1 mutation, switching endocrine therapy before visible progression extended progression-free survival substantially in the SERENA-6 trial.

But the treatment is not a cure, the reported 56% figure does not mean a 56% reduction in mortality, and longer-term survival data are still being collected.

For patients and families facing advanced breast cancer, however, having another evidence-based option—and the ability to identify treatment resistance earlier—offers something genuinely valuable: **more information, more choices, and potentially more time with the disease under control.**

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Save this article if you want to follow developments in personalized cancer treatment, and consider sharing it with someone who may find this new approach encouraging.

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